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A

TREATISE ON GOUT.

43

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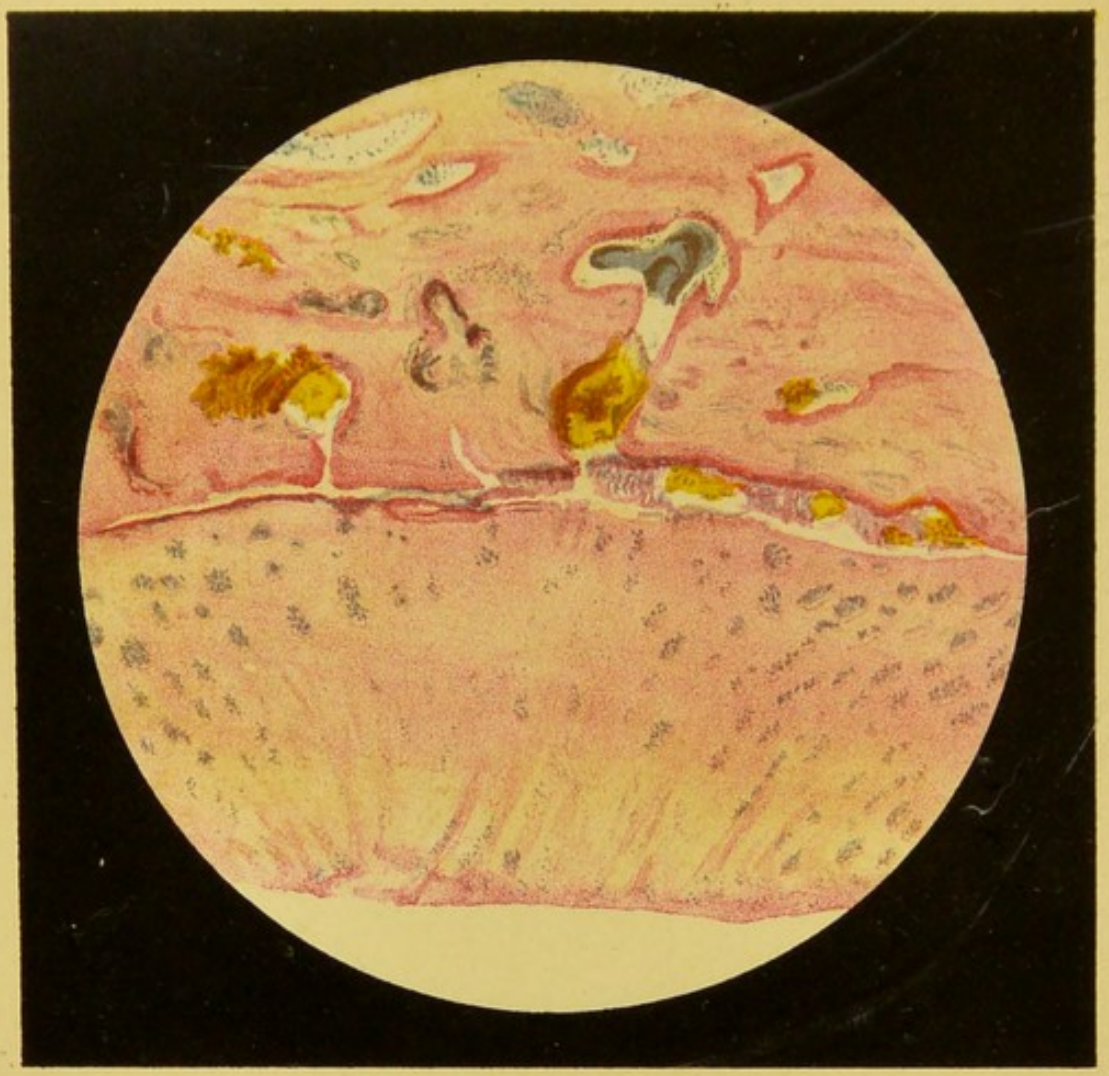


Fig. 1.



Fig. 2.

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PLATE I.

FIG. 1.—Section through the proximal phalanx of a gouty great toe-joint.

Fibrillation and degeneration of cartilage, with absence of uratic deposit. At the junction of the cartilage and the bone is seen a deposit of urate of sodium. Uratic deposit is also observed in the bone, chiefly occupying the Haversian canals.

FIG. 2.—Illustrates uratic tophi, much resembling patches of xanthoma, in the eyelids of a gouty man. In this patient there were extensive deposits in the integuments of all parts of the body.

To face Plate I.]



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A

TREATISE ON GOUT.

Presented to the Library by the Author.

BY

SIR DYCE DUCKWORTH, M.D. EDIN.

M.D. (HON. CAUSÂ) ROYAL UNIV. IRELAND.

FELLOW, AND TREASURER, OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON;
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"Ego bonâ saltem fide tradam quæ hactenus rescivi omnia; difficultates salebrasque sive rationem morbi ipsius, sive curationis methodum, spectantes, Tempori, duci veritatis, evincendas complanandasque relinquens."—THOMAS SYDENHAM (*Tractatus de Podagrâ*, 1685).

"PELL MELL, *December* 10, 1687.

"I have bin very careful to write nothing but what was the product of careful observation. Soe when the scandall of my person shall be layd aside in my grave, it will appear that I neither suffered myselfe to be decieved by indulging in idle speculations, nor have decieved others by obtruding anything to them but downright matter of fact."—*MS. Letter of Sydenham to Dr. Gould* (first published in the 2nd edit. of *Horæ Subsecivæ*. John Brown, Edinburgh, 1859).

"A knowledge of the real nature of gout and of its kindred malady rheumatism is, in my opinion, at the very foundation of all sound pathology."—TODD.

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"The real physician is the one who cures: the observation which does not teach the art of healing, is not that of a physician; it is that of a naturalist."—BROUSSAIS.

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PREFACE.

From time to time during the last twelve years, I have been engaged in the study of Gout, and have published some contributions to the subject in certain volumes of the St. Bartholomew's Hospital Reports. Five or six years ago, I determined to write a treatise on Gout, and had made some progress with the work. Various causes, however, led me to falter in this resolution, and I laid aside my manuscripts. Yet this subject has always had interest for me, and I have never ceased to study and make notes of it as opportunities offered. Two years ago, I was urged, by those whose opinion I value, to complete the work I had begun, and, with some misgivings, I resumed it. The result I now venture to lay before the Profession.

In doing so, I cannot but feel that some apology is due for intruding myself into the company (already too large) of authors on this well-worn subject—especially since the attempt to write a work that should be worthy of all that is now demanded from an author who ventures to publish a treatise on a special disease, is confessedly difficult.

In the case of a malady like Gout, the task is, in my opinion, beyond the powers of any one Physician, if he seeks to write a *complete* work, and to bring to each part of it fresh contributions and new light. It would require no less than that he should be, at once, a good Anatomist, Physiologist, Pathologist, and Chemist, as well as a trained and accomplished clinical observer.—One may well ask, therefore, who is sufficient for all this? My own stand-point throughout the present work is that of a Physician

who, as a Hospital Teacher, has to study and treat all forms of disease, and deduce from them such lessons as may be illustrated by them. My point of view is, therefore, very different from that of a so-called specialist, whose thoughts and practice must necessarily be narrowed and warped by devotion to any one subject.

While the experience of a disease like Gout attained in twenty years of service in London in a great general Hospital, like St. Bartholomew's, is necessarily very large, it is one of the privileges attaching to the office of a Physician in such an Institution, that it absolutely prevents the holder of it from becoming a specialist.

Inasmuch as London practice affords probably the largest field of observation in the world for the study of Gout and gouty ailments, it is only right that such opportunities should be utilized for the benefit of the Profession everywhere. Hospitals in London also present fuller opportunities for the study of the morbid anatomy of Gout than are elsewhere available, and in this volume will be found some results of this particular inquiry, for many of which I have to thank my friend and colleague, Dr. Norman Moore, who has paid much attention to the matter.

The classical and epoch-making work of Sir Alfred Garrod on Gout still holds, and will long continue to hold, the foremost place in the English language on the whole subject of Gout, and I must here express my indebtedness to that work, and, no less, to many suggestions kindly afforded me by my friend, its distinguished author, while writing this volume.

I may state that while a large part of my experience has come from many years' observation of Gout amongst the patients, both at the Royal General Dispensary and St. Bartholomew's Hospital, yet a more complete knowledge of the disease, as a whole, is due to an experience of it gained in another line of practice, and amongst such classes as do not frequent hospitals. With many of the phases of Gout and gouty disease, no sort and no amount of hospital practice avail to render the Physician familiar.

I am of opinion that many of our modern text-books occa-

sionally fall short of completeness and lucidity, because so much of their experience is drawn from the hospital side alone. Few can doubt that, if the notes of private case-books were subjected to the same discipline as obtains in those drawn up for hospital-purposes, some new chapters in clinical medicine and prognostics would have to be written. Some of the greatest clinical masters have in this way added greatly to the value of their writings, in proof of which I would only adduce the names of Graves, Bright, Watson, Latham, and Todd.

I have endeavoured to point out the relations of Gout to other morbid states, and its modifying influence on many of these. As will be found, I am old-fashioned enough still to believe in the existence of distinct diathetic habits of body, and venture to think that such conceptions are not only true, but also very helpful in guiding towards a better treatment of patients suffering from the disorders attaching to such habits. This teaching is not in vogue at the present moment, and is believed by some rather to hinder than advance the progress of our art. I am altogether of a different opinion.

Many of the views expressed in this work are such as have long held sway in the French School of Medicine. I have to confess myself much imbued by these, and would here express my indebtedness to the acumen and discrimination which have been brought to bear in France by a long succession of eminent teachers on the whole subject of arthritic disorders. I do not find myself so often in accord with the teaching of German authorities in respect of Gout and gouty diseases, but I gladly claim for Virchow and Ebstein that they have each thrown light on parts of the subject which were previously wrapped in obscurity. I should fail in my duty if I did not acknowledge how much I have learned from my distinguished colleague, Sir James Paget, whose contributions to this, as to all subjects on which he has written and taught, are amongst the most lucid and accurate in our possession. To the teaching of my former master, Professor Laycock of Edinburgh, and to the writings of Mr. Jonathan Hutchinson, MM. Charcot, Lecorché, Rendu, Dr.

Latham, of Cambridge, Dr. Ord, and many others, I am also under obligation.

The chapters on Treatment have been expanded to greater proportion than is common in treatises of this kind. I offer no apology for this, inasmuch as I conceive the duty of the Physician to consist as much in averting disease and treating his patient, as in discovering the nature of his maladies. Few can deny that studies in pathogeny, morbid anatomy, and diagnosis have of late years rather overridden those in practical therapeutics. Progress is demanded in all, and not in one only; but it may be affirmed that the tendency in modern times is rather in the direction of a helpless expectancy than in a strenuous effort to apply, for the patient's comfort, the clinical art in treatment, an art which was, with some exceptions, in many ways better practised half a century ago. I am disposed, indeed, to think that Medicine as an Art is now in some danger of being lost amidst futile efforts to exalt it into an exact Science. I maintain that a great Physician is, and must be, a great Artist.

I must, further, acknowledge various kinds of help afforded me by Mr. D'Arcy Power in our Hospital Museum, and state that I have had the advantage of the skill of Mr. Mark, and of my present senior house-physician, Dr. Wynne, in illustrating this volume, their original drawings having been admirably engraved by Mr. Danielsson.

In the preface to his famous *Tractatus de Podagrâ*, addressed by Sydenham two hundred and six years ago "to the most learned Dr. Short," he remarked:—"It is my nature to think where others read; to ask less whether the world agrees with me than whether I agree with the truth; and to hold cheap the rumour and applause of the multitude. . . . Why should I be anxious about the judgment of others?" Such words are rarely to be found in any modern preface, but they well-illustrate the moral elevation of that most eminent man, and convey a lesson which much needs to be learned by authors in our own time. I could wish to repeat every word of it in respect of this present effort;

but, with full consciousness of the many imperfections in this volume, I will yet dare to affirm that I have sought to be guided in writing it by those high traditions which have come down to our Profession, and which the English School of Medicine will ever have cause to venerate as emanating from the illustrious Sydenham.

The delay which has occurred in issuing this work has, at all events, enabled me to profit by the Horatian maxim :—"*Nonum prematur in annum membranis intus positis ;*" for I have, happily, surpassed that period.

Lastly, I will express my thanks to my publishers for much consideration and many courtesies.

LONDON, *St. Bartholomew's Day*, 1889.

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ERRATA.

1. Page 63, line 2 of description of Fig. 2, *for* "reflected" *read* "transmitted."
2. Page 133, line 9 from top, *for* "Nicol" *read* "Nichol."

A TREATISE ON GOUT.

CHAPTER I.

DEFINITION OF GOUT.

NOTHING is more difficult than the task of forming an exact definition of a morbid state. It is notoriously easier to criticize than to propound one. The besetting danger, or fallacy, that must always underlie attempts in this direction consists in the tendency to take typical or exquisite examples, and to formulate from them theories which are insufficiently comprehensive.

"Definitions, if they are to be more than convenient helps to arrangements, belong only to sciences more exact than pathology can be. It is better at present to think of diseases as in groups with borders that are not clearly marked; or as of nations with ill-defined frontiers, and with inhabitants intermingling, and even intermarrying. We may find typical examples, as of peoples, . . . and may call them by distinct names, . . . but we must use them very cautiously in the real study of pathology."[1]

In attempting to define what, in the existing state of our knowledge, constitutes gout, I shall take heed to the caution thus expressed. In my study of this disease, I have for long been trying to discover how, and on what lines, the changes proper to it work themselves out, and thus to be able to say in any given case, this is, and this is not, a manifestation or product of gout. It is of the last importance to seek to be thus exact in the case of a disorder such as this, because other pathological conditions certainly run on lines almost parallel with it; and what is of more consequence, as will be shown, is that some of these conditions are occasionally mixed up with those proper to gout, thus producing hybrid states very difficult to unravel. Throughout this

[1] Sir J. Paget, Morton Lecture, Roy. Coll. of Surgeons, 1887.

treatise I propose only to deal with such perversions as are due to unequivocal gout, and to disentangle from them all changes and manifestations which are not thus fairly to be explained. I shall also beware of what Mr. Hutchinson calls "a vice of clinical study," viz., the selection of a few well-marked symptoms in probably exaggerated cases, the giving to these of a special name, and then proceeding to describe and classify the disease so named, as if it were essentially distinct, and needed only acumen in diagnosis for its recognition.

Gout is a constitutional or diathetic malady,[1] manifesting itself in very varied aspects. In its acute forms it usually, but not invariably, presents the characters of localized inflammation, accompanied by peculiarly intense pain; the inflammation in its course, and the attendant pain, being of a specific nature. In its chronic forms there may be no manifest inflammatory features, and even no pain. The male sex, chiefly in the third decade, is most frequently the subject of the disorder in the acute form, and the articular system not seldom bears the brunt of its incidence. In the earlier manifestations the inflammatory trouble seizes especially upon the first joint of the great-toe, spreading subsequently to other articulations, and a suppurative stage but very rarely occurs. The digestive system is largely involved, and in the fully developed forms of the malady hardly any of the viscera or textures are unaffected. The nervous system is likewise specially implicated, whether primarily or not is as yet a vexed question.

The disorder is either inherited or newly acquired. In most of its manifestations it is plainly associated with perturbed relations of uric acid in the economy, and the inflammatory attacks are accompanied by deposits of urate of sodium, for the most part in articular cartilages and fibrous structures. A measure of pyrexia commonly forms part of the acuter gouty processes, but profound, though slow, nutritional changes may proceed quietly in the chronic forms of the malady without any febrile movement.

Gout has been placed by some of the older nosologists amongst the order of Fevers, and has been described as "a tertian fever terminating in fourteen days." This definition was formulated before the days of clinical thermometry, and was manifestly applicable only to acute attacks. Acute gout has been classed with

[1] Hunter thought it probable that gout is not always an act of the constitution, but that parts may be so susceptible, or rather disposed for this action, that they may immediately run into it when deranged. Scudamore believed, and I agree with him, that this tendency proved gout to be an act of the constitution.

rheumatic fever as an "excretory fever." [1] Parkes [2] wrote: "I define gout, after Garrod, as a febrile affection, with inflammation about joints, leading to a deposit of urate of soda."

The essential elements in any case of gout relate, therefore, to peculiarity of diathesis, to diminished alkalescence of blood, owing to impregnation with uratic salts, and to the deposition of the latter, especially in the textures of joints. Sometimes the local manifestations prevail more than the constitutional. Again, the latter may be alone prominent, without marked articular element in the case, as evinced in instances of incomplete or irregular gout. In some cases we meet with alternations of local and constitutional disturbance.

The main conception of gout should have regard to its constitutional nature, and to the essential unity of the disorder, whether manifested in acute and regular, or in chronic and irregular fashion. In one sense the malady is always chronic, since we must regard any one having once given evidence of unequivocal gout as goutily disposed for his lifetime.

Respecting what is often called "poor gout," or "poor man's gout," it must be stated that many of such cases can be plainly referred to chronic rheumatic arthritis. Others are as plainly examples of true gout as can be demonstrated. They occur in persons of feeble constitution with faulty circulation and digestive incapacity, and who are prone to early textural decay. They are often quite temperate, but not always so. Women are sometimes the subjects of poor gout. It may, and often does, appear before the third or fourth decade; this is always a feature indicating gravity in any case, and is due to strong hereditariness. More than fifty years ago, Dr. Billing wrote: [3] "Temperate persons have gout, because they have, whether hereditarily or not, a feeble nervous system and weak digestion. Abstemiousness will not cure such gout, which is called 'poor gout,' that which has come on in weak constitutions without excess." Some cases of this class are simply examples of incomplete gout, and, by excess, they might eventuate in more sthenic and frank forms of the disease.

I think I have made it plain that no brief or trite definition of this malady will suffice to explain its varied characters. A fitting conception of it now demands, with increasing knowledge, a comprehensive survey of a very large field of pathological processes.

[1] Laycock, Medical Observation and Research, 2nd edit., p. 124. Edinburgh, 1864.
[2] The Composition of the Urine, 1860, p. 292.
[3] Principles of Medicine, p. 183.

Those who study carefully the multiform phases of any one malady lay themselves open to the charge of seeing signs and symptoms of it in almost any case of disease. The evils of specialism are indeed only too manifest at the present time. Taunts such as I have alluded to are freely cast at those who see "gout" everywhere, and are, perhaps, often not unfairly cast. The competence and honesty of the observer can alone shield him from such charges. It is chiefly in respect of the imperfect developments and manifestations of gout that difficulty arises, and this perplexity is only enhanced by the challenge to bring every feature in every case to the ultimate test of the presence of uratic deposit. This is, in the nature of things, impossible, and the prudent observer must, perforce, fall back upon as complete clinical study as can be had. Where this is cautiously and honestly attempted, I would venture to affirm with Gairdner, that "the gouty diathesis is often very perfectly developed in individuals who never see its local manifestations, and that the strumous is not more frequent than the gouty habit."

Note.—"Gout," remarked Trousseau, "is an admirable name, because in whatever sense it may have been originally employed by those by whom it was invented, it is not now given to anything else than that to which it is applied. . . . The name is all the better in that it has but little nosological meaning."[1]

The term appears to be loosely employed in Germany, where the disease is not prevalent. Thus, Dr. Pye-Smith affirms that "'Gicht' is popularly credited with all the pains which are called 'rheumatics' in England. Sometimes 'Gicht' is nothing but bad corns, and is rarely true gout."

It is probable that the earliest English (Saxon) name for gout was *fotadle*, or *foot-addle*. The word *addle* appears to have been a synonym for ailment; thus, "Shingles was hight circle addle." *Vide* Leechdoms, Wortcunning, and Starcraft of Early England, collected and edited by Rev. Oswald Cockayne, M.A., Cantab. London, 1864. Herbarium of Apuleius, vol. i. pp. 81, 85. (I am indebted to my colleague, Dr. Gee, for this reference.)

Dr. J. Mason Good, in his Physiological System of Nosology, 1817, p. 194, remarks that "Gout is one of the maladies which seem to have been common in England in its earliest ages of barbarism. It is frequently noticed by the Anglo-Saxon historians, and the name assigned to it is fot-adl."

"Cyragra," so termed in the *Sinonoma Bartholomei*, by John Marfelde, monk of the Order of St. Austin, St. Bartholomew's Monastery, London, edited by J. L. G. Mowat, M.A., Fell. Pem. Coll. Oxon., Oxford, 1882. = "Gutta vel dolor in manibus sicut podagra in pedibus."—*Anecdota Oxoniensia* (MS. 14th century).

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[1] Clin. Med., vol. iv. p. 359.

CHAPTER II.

PATHOLOGICAL DOCTRINES CONCERNING GOUT.

I DO not propose to discuss the manifold opinions that have been entertained through centuries regarding the nature of gout. Most of the good writers on the subject have given historical summaries, and few in recent times have presented more readable accounts of these than Sir Alfred Garrod[1] and Professor Ebstein of Göttingen.[2]

The term "gout" at once suggests the idea of a humoral pathology, and this has been, certainly for two centuries past, the most largely accepted view in medical history. The earliest name by which the malady was known was "podagra," a term still in use, and of value so far as it sufficiently expresses the most obvious feature of a typical case without implying any theory of causation. Cullen was the first to dispute the long-held view of the humoral pathology of gout, and in 1784 promulgated in its place a theory that the disorder was one primarily of the nervous system.[3]

He stated that he adopted this view from Stahl.[4] Henle in 1847 published his opinion that the origin of gouty inflammation was probably to be found in the central nervous system. At the present time the humoral and neurotic theories are still in conflict, but greater acceptance is perhaps found for the former.

Cullen's doctrine excited much interest at the time it was set forth. His opinion carried weight everywhere, and his theory did not entirely exclude the views of the humoralists, for he allowed that a peculiar matter appeared in some gouty patients after the disorder had subsisted for a long time, and he regarded

[1] Gout and Rheumatic Gout, 3rd edit. London, 1876.

[2] *Die Natur und Behandlung der Gicht.* Wiesbaden, 1882.

[3] First Lines of the Practice of Physic, vol. ii. part 1, chap. xiv. Edited by John Thomson, M.D. Edinburgh, 1827.

[4] *Theoria Medica Vera*, &c. G. E. Stahl (Halle, 1737). *De Doloribus Spasticis Arthritico-Podagricis*, § xxxviii. p. 1040.

it as the effect, but not the cause, of the disease.[1] Senator,[2] referring to the views of the solidists as represented by Cullen, remarks that they have never been able to hold their ground against the various humoralistic theories.

Great impetus was given to the humoral doctrine by the discovery of the peccant matter, which had so long been suspected.[3] For more than half a century there was a growing suspicion that lithic (uric) acid was the malign agent in inducing gout; and although Mr. Murray Forbes in 1793,[4] Wollaston,[5] Parkinson,[6] Pearson,[7] and Sir Henry Holland[8] in this country, and Andral,[9] Rayer,[10] Cruveilhier,[11] and Petit in France, all regarded gout as intimately connected with the presence of uric acid, it was not till Garrod[12] unequivocally demonstrated the fact in 1848, that this discovery made plain one portion at least of the pathology of this affection, and thereby constituted one of the most brilliant advances made in modern medicine.

Thus far the ground is clear, and it is necessary at this point to review the various theories which have been propounded to explain the relations between uric acid and manifestations of gouty disease.

Before proceeding to enumerate the several opinions held respecting this relationship, it is fitting to record that about ten years before Garrod's demonstration that uric acid was the peccant matter of gout, Sir Henry Holland surmised that "there was a presumable relation between lithic acid and its compounds and the matter of gout;" "that the accumulation of this matter of the disease may be presumed to be in the blood, and its retrocession or change of place, when occurring, to be effected through

[1] His theory was opposed by Dr. Tode in an inaugural thesis at Copenhagen in 1784, and by Dr. Luther in another at Halle in 1786. Sir Charles Scudamore and Garrod also criticized Cullen's definition and theory of the disease in their Treatises on Gout, 1819 and 1859. Parkinson alludes to Cullen's theory, and hesitated to advance his adherence to the old humoral theory in consequence. *Vide* Preface to his Observations on Nature and Cure of Gout. London, 1805.

[2] Ziemssen's Cyclopædia, art. "Gout," Eng. transl., p. 101.

[3] Scheele discovered lithic acid in urinary calculi and urine in 1775. Sydenham originated this term ("materia peccans") in his famous and classical Treatise, 1685.

[4] A Treatise upon Gravel and upon Gout, &c.

[5] On Gout and Urinary Concretions. Philosoph. Trans., ii. 386, 1797.

[6] *Op. cit.*

[7] Phil. Trans., 1798.

[8] Medical Notes and Reflections, p. 252, 1839.

[9] *Précis d'Anatomie pathologique*, 1829, vol. i. p. 553, and vol. ii. p. 387.

[10] *Traité des Maladies des Reins*, 1839, vol. i. p. 243.

[11] *Atlas d'Anatomie pathologique*, 4e livraison, planche iii.

[12] Med.-Chir. Transactions, 1848.

the same medium;" and "that an attack of gout consists in, or tends to produce, the removal of this matter from the circulation, either by deposits in the parts affected, by the excretions, or in some other less obvious way through the train of actions forming the paroxysm of the disorder."[1]

Garrod's theory of the relation of uric acid to gout is founded on the view that the kidneys fail, either temporarily or permanently, to excrete this acid, and that the premonitory symptoms, and those of the paroxysm, arise from retention of excess of it in the blood and the effort to expel it from the system. He conceived that this renal incapacity, or a tendency to it, might be transmitted hereditarily. He allows, however, that these views are not by themselves sufficient to explain all the phenomena of gout.

In proof of his views, he has demonstrated that prior to, and at the time of, a seizure, urate of sodium is present in abnormal amount in the blood. He is careful to state that this condition may exist sometimes without any overt gouty manifestation, as, for example, in cases of lead-poisoning.[2]

He avers further, that gouty inflammation is always accompanied with deposit of urate of sodium in the inflamed part; that the deposit is interstitial and infiltrated, and also permanent. He regards the deposition of urate of sodium as the *cause*, and not the *effect*, of the gouty inflammation. He believes that the inflammation in a gouty attack tends to the destruction of the urate of sodium in the blood of the inflamed part, and consequently in the system generally; that the kidneys are probably implicated functionally in the early, and certainly structurally in the chronic, stages of gout, and that the urine is altered in composition; that the causes predisposing to gout are either such as produce increased formation of uric acid in the system, or lead to its retention in the blood, and that the causes of a gouty fit are those which induce a less alkaline condition of the blood or augment the formation of uric acid, or are such as temporarily check renal elimination; that in no disease but true gout is there deposition of urate of sodium in the tissues.

Garrod seeks to prove his propositions by clinical and pathological observations. Most of these views are now universally accepted, but some of them are still the subject of debate, notably that in which he attributes defective elimination of uric acid to

[1] *Op. cit.*, chap. ix., 3rd edit.

[2] It will be shown subsequently that in many parts of Great Britain and Ireland, and on the continents of Europe and America, lead-poisoning is not found to be associated with gout in the manner in which it undoubtedly is in London.

temporary failure on the part of the kidneys, and that in which he regards uratic deposition as the cause of the paroxysm, and not the effect of it.

Dr. W. Gairdner[1] in his well-known work combated the views of Garrod, and regarded "the disappearance of urea and uric acid in the urine, and their accumulation in the blood," as but a frequent symptom and consequence of gout, itself again being the cause of other important phenomena, such as headaches, somnolence, dyspepsia, &c. He was disposed to attribute the arrest of renal function to some great emotion or violence affecting any great function of the body, and he pointed out that such an arrest was even more remarkable in hysteria than in gout. Dr. Gairdner's views obviously required the intervention of nervous influence, though he did not thus express this opinion in so many words.

Charcot remarks that Garrod's facts do not as yet make a physiological theory of gout possible. He accepts Garrod's views in the main, but believes that the local changes depend for the most part on the direct consequences of the general change, and that gout is in all cases a chronic and constitutional disease.

Cruveilhier regarded the deposition of urate of sodium as the cause of gout, and subsequent attacks as coincident with fresh secretions or deposits of it.

Dr. Barclay[2] regarded the uric acid theory as "far too mechanical." He allowed that in the case of the joints we find the inflammation and the deposit harmonizing together, but asked, "Does it necessarily follow that if, during the existence of gout, inflammation of any tissue does not present the same deposit, it must be excluded from our idea of the disease?" And again, "Must we of necessity find urate of soda in the stomach and the bronchi before we can admit gouty gastritis or gouty bronchitis?" He thought that because such deposits were not found in these situations,[3] we were warranted in denying that "true gouty inflammation is always associated with, or caused by, the deposit;" and he thought "this conclusion acquired additional force from the consideration that though the deposit and the inflammation were associated together in the joints, the urate of soda was seen in other parts without any evidence of its exciting inflammation there." Barclay believed the first change to be in the molecular structure of the blood itself, this being set up by the repeated

[1] Gout, its History, its Causes, and its Cure. London, 1849, v. p. 99; 3rd edit., p. 88, 1854.

[2] On Gout and Rheumatism in Relation to Disease of the Heart. London, 1866.

[3] "Bence Jones found a deposit of crystallized urate of soda in the walls of the bronchial tubes." *Vide* Garrod, *op. cit.*, p. 204, 3rd edit.

introduction of gout-producing elements into the circulation. The blood-globules having received a certain impress, were succeeded by others which had a general resemblance to them, and thus a morbid tendency came to be transmitted. He believed "the retention of uric acid to be a symptom, a consequence of the attack of gout, and not its cause. The good living and the stimulants do not simply cause an excess of uric acid to be formed, but they end by causing some more permanent change, and probably one affecting the blood-globules, which reacts on the kidney, putting a stop to the excretion of uric acid, and causing its retention in the serum, where, passing in the round of the circulation, it is very apt to become deposited as urate of soda." The effects of colchicum in checking a gouty paroxysm he believed to indicate "that there is a disease to which the name 'gout' is applied, distinct from the excess of uric acid in the blood-serum which attends its progress." The fact that alkaline remedies, which, if the purely chemical theory were true, should readily neutralize and lead to elimination of the peccant matter, do not materially influence the progress of gouty inflammation, he thought pointed in the same direction.

Dr. Parkes was of opinion that the elimination, and not the formation, of uric acid was impeded in gout, and that there was probably increased production of it in the system. He doubted the inadequacy of the kidney, as alleged, to excrete it, and surmised that there was some peculiar and unnatural combination in the blood or organs which held back this and some other substances, notably phosphoric acid. "If this be the case, the deficient elimination is, as it were, only a consequence of more important antecedent aberrations in metamorphosis, of which impeded excretion is a natural sequence. What these are, however, is quite unknown; but an unnatural formation of uric acid, either from food or tissues, may possibly be part of them."[1]
Later researches add force to these prescient views.

Professor Laycock considered Garrod's theories inadequate for the explanation of the whole phenomena of gout. In his lectures at Edinburgh, twenty-five years ago, he taught that:—

(*a.*) Gout is not necessarily articular, nor even associated with articular inflammation.

(*b.*) Gout is characterized not by urates in the blood, but by the genesis of uric acid *in the tissues*, and its action thereon, and is especially characterized by *peculiar changes in the innervation of the individual.*

[1] On Urine, p. 298. E. A. Parkes, M.D. London, 1860.

Dr. Edward Liveing[1] has expressed his doubt as to the dependence of the phenomena of gout upon the associated presence of uric acid in the blood, because excess of this matter is found in other pathological states which have no connection with gout, and he believes that many of the features of the malady betoken a nervous origin.

Sir William Roberts[2] accepts Garrod's teaching. He thinks that the defective power in the kidneys to eliminate uric acid probably arises from diminished alkalescence of the blood.

A very different view has been set forth by Dr. Ord[3] in an original and thoughtful essay. He regards gout as arising from a tendency to a special form of degeneration or want of tissue organization in some of the fibroid tissues, either inherited or acquired, wherein an excessive formation of urate of soda occurs, and whence this salt is discharged into the blood, and also deposited promiscuously in such parts as are least freely supplied with vascular and lymphatic structures—to wit, cartilage. The paroxysms of gout he would attribute to special local existing causes, as injuries, exposure to cold, and the like. Dr. Ord thus believes that the uratic deposits are not to be regarded as significant of their elimination from the blood, that the local processes are not dependent on these deposits, and that the latter are not the result of the inflammation. He takes cognizance of nervous influence so far as to admit that "all authors, in one way or another, admit the direct influence of the nervous system," and he believes that local gouty "degeneration and inflammation tend to infect the rest of the system through the blood, and to set up similar actions elsewhere through reflex nervous influence."

This theory, then, is a return in part to the views of the old solidists, set forth according to modern ideas; but it is partly neuro-humoral, and, in any case, combative of Garrod's theory. It opens up very suggestively the large question whether or not there be, as part of the intimate nature of gout, a specific tendency to degeneration and abnormal transformation of certain tissues leading to uratic deposit; and a definite reply to this is not yet forthcoming.

Suggestive views on the relation of uric acid to gout have been set forth by Dr. Ralfe,[4] who believes the first step in the

[1] On Megrim, Sick Headache, and some Allied Disorders, p. 404. London, 1873.

[2] On Urinary and Renal Diseases, p. 66, 3rd edit., 1882.

[3] St. Thomas' Hosp. Reports, New Series, vol. iii. p. 227, 1872; and Med. Times and Gazette, vol. i. p. 233, 1874.

[4] Clinical Chemistry, p. 295, 1883.

production of the disease to be diminished alkalinity of the blood by reason of the accumulation in it of acid and acid salts.

He believes that uric acid is formed in health and disease in but minute quantities, and that deposition of it is due rather to its insolubility than to excessive production of it. In this view, the uratic deposits are a consequence, and not the cause, of the disorders commonly attributed to them.

Garrod found the reaction of the blood in chronic gout to be nearly neutral. Retention in the system is due to a fault in the tissues, leading to incomplete elimination. The amount thrown out by the kidneys is believed to be that formed by those organs, and at once discharged instead of being destroyed.

The diminution of uric acid in the urine is found chiefly in chronic gout where the kidneys are already damaged, and hence Dr. Ralfe is disposed to doubt Garrod's theory that the failure in renal elimination is the prime cause of the retention of uric acid in the system, and he believes that "the first step in the process lies in the failure of the tissues to reduce the acid, as occurs in health. In the large glands, or where the current of the circulation is free, the uric acid is carried into the blood, and gradually reduced to urea; in tissues outside the current of the circulation, the insoluble uric acid is not so readily carried off, and so on the slightest disturbance is deposited, as is the case in cartilages of the joints, the ear, &c." Dr. Ralfe, then, accepts Dr. Ord's views as to textural degeneration, either hereditary or acquired, in which the tissues and blood become loaded with effete products, and he next invokes the agency of the nervous system, supposing that "such predisposing conditions lead at last to disturbance of some special nerve-centre," which constitutes the determining cause of the gouty attack, "the result of which is the accumulation of uric acid in the blood, and the deposition of urate of soda in the tissues." These views are partly solidistic and partly neuro-humoral.

I may now mention the views of the late Dr. Murchison,[1] who regarded gout merely as a result or variety of lithæmia. This condition of the blood is recognized on all hands as due to imperfect digestion and functional derangement of the liver. "Articular gout is, so to speak, a local accident, which, though sometimes determined by an injury, yet may occur at any time in persons in whom the normal process, by which albuminous matter becomes disintegrated in the liver into urea, is persistently deranged. In other words, gout, like diabetes, is the result of a

[1] Lecture on Diseases of the Liver, 2nd edit., p. 568, 1877.

functional derangement of the liver." Murchison accepted Garrod's views as to previous accumulation of uric acid in the blood, and failure of renal action in elimination of it at the later stages of gout, though he pointed out, as Garrod did, that the kidneys are generally healthy at the first onset. He believed that the innate defective power of the liver, whereby its functions are readily deranged, is capable of hereditary transmission, and, so, often passed on to the offspring. Hence, as Bristowe points out, gout, according to Murchison, would bear something of the relation to the liver that uræmic dropsy does to the kidney.

Professor Latham, of Cambridge, has discussed the relations of uric acid to gout, and the recondite question of the formation of uric acid in animals.[1]

He holds a very similar opinion to that expressed by Murchison as to the hepatic origin of gout, and states: "Just as in diabetes the essential fault lies in the inability of the system, either in the liver, or it may be elsewhere, to effect the metabolism of glucose, which then passes into the circulation and is discharged by the kidneys, so in gout or gravel the imperfect metabolism of glycocine" (a derivative of glycocholic acid) "is the primary and essential defect. Unchanged, it passes from the alimentary canal, or elsewhere, into the liver; there, under the action of the gland, it is conjugated with urea, resulting from the metabolism of the other amido-bodies, leucine, &c., and is converted into hydantoin, or a kindred body, then passes on to the kidneys, to be combined there with another molecule of urea forming ammonium urate, a portion of which overflows into the circulation, and is converted into sodium urate." Dr. Latham believes, further, that there is some change in the nervous system which determines the attacks, their incidence on the joints, and the hereditary nature of gouty disease.

Mr. Jonathan Hutchinson,[2] as the result of large experience and much thought, has promulgated some noteworthy and important views in respect of the relation of uric acid to gout. In subsequent chapters I shall have occasion to refer to many of the doctrines on the whole subject of gouty disease which have been laid down by this eminent observer.

He accepts as undoubted evidence of true gout all cases of arthritis and inflammation of fascia and allied structures which

[1] On the Formation of Uric Acid in Animals: its Relation to Gout and Gravel. Cambridge, 1884. *Vide* also Croonian Lectures, 1886. Royal College of Physicians. London, 1887.

[2] On the Relations which Exist between Gout and Rheumatism. Trans. Internat. Med. Congress, London, 1881, vol. ii. p. 92.

occur in association with accumulation of urate of soda, this fact being proved after death, or by examination of the blood or urine during life. But he believes that we are not to wait for proof of the presence of this salt in the blood, far less of its deposit in the tissues, before we are entitled to use this term. Whatever can be proved to be connected with tendency in this direction must rank as gout, though there may be only dietetic idiosyncrasy, but no tophi, lithæmia, or arthritis. "Much that occurs in connection with inherited gout in young people, and many forms of 'quiet gout' in those who have both inherited and acquired it, is probably without any proved tendency to the accumulation of lithates. The gout-process is partly due to defective assimilation, and partly to deficient excretion, and it is probably only when the kidneys are decidedly affected that any great tendency to the formation of tophi is witnessed. It is possible, indeed probable, that in some of the inherited forms neither digestion nor excretion is much impaired, and that the inheritance is of peculiarity of tissue." Mr. Hutchinson believes in a basic arthritic diathesis, and that upon this may be built up, under the influence of special causes, a tendency to gout, rheumatism, or any one of their various modifications and combinations. Hence, he regards gout as, in many cases, but a superaddition to rheumatism.

Ebstein's[1] views differ, in some respects, from most of those I have mentioned. He found, after a study of many of the affected tissues in gout, that one change is common to all of them, independently of the uratic crystallizations, and that is, a necrosis of the parts wherein such depositions take place. He regards such necrosis in gout as equally characteristic as is uratic deposit. Both changes must coexist in any texture in order to constitute truly gouty patch; and he has detected such patches in the kidneys, in hyaline and fibro-cartilage, tendons, and connective tissue. He calls attention to an early stage of this necrosing process, in which as yet no deposition has occurred, and, therefore, maintains that nutritive tissue-disturbance is the primary factor, and uratic crystallization the secondary one in the gouty process, this last not occurring before complete death of the damaged texture. He has never seen crystallized urates in healthy tissue. With Garrod, he agrees that uric acid is excreted at first in liquid form as sodium urate into certain textures, becoming rapidly inspissated, owing to its insolubility, and tending to crystallize out and solidify. He maintains that this compound is a directly poisonous irritant wherever deposited, the injurious effects vary-

[1] *Op. cit.*

ing according to the quantity and concentration of the uratic deposit, and also according to the vulnerability of the special tissue involved, firm textures resisting this process better than those of looser character. He regards this incrustation as analogous to calcification, in which lime-salts are deposited in tissues whose nutrition is greatly or completely destroyed. As will be shown later on, Cornil and Ranvier maintain that uratic deposit occurs primarily in cells, penetrating subsequently into the neighbouring ground-substance, notwithstanding the resistance offered, whereas in calcification the infiltration begins primarily in the ground-substance. Ebstein admits that lime-salts may be subsequently deposited in gouty tissues, just as in the case of other necrosed textures.

Having now given a summary of the principal doctrines which have been laid down by the best authorities respecting the relations of uric acid to the specific manifestations of gout, I am in a position to discuss these, and to offer, as concisely as possible, the views which best commend themselves to me as illustrating the pathogeny of the disorder. This I propose to do in the next chapter.

CHAPTER III.

THE PATHOGENY OF GOUT.

"Pathology is a part of biology, and not derived chiefly from the study of anatomy and chemistry."—PAGET.

"No very limited theory, and no one particular hypothesis, can be found applicable to explain the whole nature of gout."—SCUDAMORE.

IT may be confidently asserted that, according to present knowledge, no conception of this malady is possible which should exclude from its purview the part played in it by uric acid. This is, without doubt, the peccant matter which works much of the varied and far-reaching mischief. It will, however, be shown that the peculiarly perverted relations of uric acid, even in true gout, do not constitute the whole of the disorder. In spite of teachings to the contrary, I would affirm at the outset that this part of the pathogeny of gout is certain, so that it may be plainly stated—"No uric acid, no gout." That I may not be misunderstood hereafter, I would express my adherence to the view that the most unequivocal evidence of true gouty disease is that derived from the presence of uratic salts in the tissues; and, I suppose, no one differs from this view thus stated. This deposition, however, is a manifestation of the extremest and most gross change that can be wrought in any case. I am confident that much gouty disease and many textural changes can also be induced without this specific deposit in overt form, and I shall seek to give proof of this. While accepting the view that uric acid is the peculiar irritant, I venture to maintain that it may work its varied evils without in every instance or in every tissue giving token of its presence. Many of the difficulties in diagnosis, especially in differential diagnosis between gouty and other forms of arthritis, are explained by admission of this view. To clear the ground at once, I would add that I distinctly claim another field of pathological process for the disorder known as

chronic rheumatic or rheumatoid arthritis, often miscalled (as I think) "rheumatic gout."

All that it is proposed to treat of in this volume relates to true and unequivocal gout, as distinguished from all other forms of disease which affect and disable joints. Articular gout is only a variety of gout, the best-marked forms of which constitute the extreme outcome of the malady. Unlike other forms of arthritis, in which disorder is mainly or altogether confined to the structures of joints, gouty arthritis is only a part of a widespread disease affecting variously many other parts of the body.

It is this character which has led to the application of the term *protean* to gouty manifestations,—a term not, perhaps, inappropriate, but one which has no doubt sheltered much hasty and erroneous diagnosis.[1] As will be shown later, many troubles may occur in a gouty person which are not truly gouty; and certainly many such occur in non-gouty persons which are wrongly attributed to gout, albeit, in a truly gouty individual, any disturbance of the balance of health is apt to be modified specially by the diathetic habit of body. Hence, I cannot accept Ebstein's dictum that "we must seek the causes of gout in the place where uric acid is formed."

Before proceeding to discuss the specific relations of uric acid to gout, as commonly recognized, it will be well to endeavour to gain as large and comprehensive a view of the entire chain of morbid events as possible. In the first place, we have to face the fact, that although the disorder is very widely spread, it is not universal. As is the case in respect of rheumatism, not every one is or can become rheumatic; so, I believe, not every one is or can become gouty.

Rheumatic proclivity is, however, greater and more widely spread than gouty proclivity. Nothing is better established in the nature of gout than its hereditary transmission. Where in any community there is most gout, there has probably been much gout in the ancestry. It is also believed that the disease may be induced or newly acquired, or, at any rate, so far as careful inquiry allows past family history to be invoked, the disease occurs in persons who can trace no overt inheritance of the tendency. Without doubt, one is on difficult ground here, for none so well know the fallacies surrounding inquiries into family history as those who have been at the pains honestly to try and learn it. In the case of gout, however, we may be the more

[1] "Almost every symptom, from an eruption on the skin to threatening apoplexy, may be gout."—*Herbert Mayo, F.R.S., Philosophy of Living*, p. 25, 1837.

confident, since any marked expression of it, if ascertained by a skilled inquirer, is less apt to be misunderstood than is the case with many other diseases, and the field of observation is certainly large enough, at all events in this country.

Other difficulties arise, too, in working out this part of the great problem of gout. Even true gout does not always "breed true," and transformations and comminglings of different morbid states are certainly handed down in the process of transmission to offspring. All this must be acknowledged and allowed for. The whole matter is, in truth, one of exceeding difficulty, but for that reason, among others, it is worth the attempt to unravel it reasonably and with an open mind. The view set forth by several distinguished observers, amongst whom I will mention Laycock, Charcot, and Hutchinson, respecting a basic diathetic habit of body called *arthritic*, has well commended itself to my mind.

Not to enter upon the large question of the several diathetic or constitutional habits which may be observed in the human race, which would be foreign to the scope of this treatise, I would express my belief in the existence of such distinct diatheses, regarding these views as being extremely helpful to the practising physician in the present state of our knowledge, as affording means for securing further light and certainty in studying the nature of diseases, and also much aid in the recognition of the best means for averting the evil tendencies of such states.

This study does not present attractions to all clinical observers. Some despise it, and many, not having been trained in this mode of thought and observation, discard its teachings, and prefer to come face to face with each case of disease as it presents itself, and to deal with it simply on what are called general principles. For myself, I may state that I count myself happy to have been trained for some time under Laycock to pay the fullest regard to such indications as have been laid down by the best observers in this study, and I can affirm that I daily draw help and gain insight by the practice of his principles. With due care, and subject to the correction of other methods, great assistance is secured from the physiognomical method of diagnosis. I am far from asserting that this line of investigation is absolutely indispensable, because precision in it is not to be gained without great pains, and excellence in it is perhaps not attainable by many; but I would strongly urge its practice, and, where possible, the regular demonstration of it to students in addition to the usual clinical methods.

I believe that there is a basic arthritic stock, or diathetic habit

of body, from which arise, as branches, two main and distinct classes of disorder, commonly recognised as gout and rheumatism. This was Pidoux's theory, it is accepted by Charcot and Hutchinson, and I think it a good one to work with in prosecuting research into the nature of the two disorders referred to.[1] This nosological position entails indirect relation between all forms of rheumatism and gout. Arthritically-disposed individuals are peculiarly vulnerable, and thus sensitive to changes of temperature, soil, and climate. They manifest this for the most part by trophic changes in the joints and other structurally allied tissues.

Heredity is a strongly-marked feature of the arthritic diathesis, and hence gouty or rheumatic affections may supervene in the descendants of either gouty or rheumatic persons. The nervous system is plainly involved in this diathesis.

It is on lines such as these that I venture to propound the view which best commends itself to my mind in respect of the pathogeny of gout. It is, I hold, in individuals who either inherit or acquire such peculiarities of tissue-potentiality that we must look for the development, in one direction, of gout, and in the other of what we recognize as rheumatism. The specific characters of gout are only, I believe, induced in those individuals who are thus diathetically predisposed. I do not believe, as has been alleged, that this arthritic habit of body is universal.

In 1880 I published an essay[2] in which I put forward a plea for the neurotic theory of gout. The following propositions expressed the views I held at that time:—

First, I urged that the diseased conditions which are recognized as of unequivocally gouty nature are primarily dependent upon a functional disorder of a definite tract of the nervous system, and that thus gout is a primary neurosis.

Secondly, That there is much in the nature of the malady itself, and much evidence forthcoming by way of analogy, to warrant the conjecture that the portion of the nervous system specially involved is situate in some part of the medulla oblongata, where, possibly, may be placed a trophic centre for the joints.

Thirdly, That the gouty neurosis may, like others, be acquired, intensified, and transmitted; also, that it may be modified variously, and commingled with other neuroses; that it may suffer metamorphic transformations, or be altogether repressed.

[1] My friend M. Lancéreaux attributes this view to Féréol.

[2] Brain, April 1880. Translated into French by Dr. Sordes, with introduction by Prof. Ball. Paris, 1884.

Fourthly, That this diathetic neurosis imposes its type upon the affected individual in definite nutritional modes, affecting the assimilating and excreting powers, exhibiting marked peculiarities in nervous impressibility, and determining, in more or less degree, a physiognomy of the gouty.

Fifthly, That a large part of the phenomena known as gouty are due to perverted relations of uric acid and sodium salts in the economy, resulting from the morbid peculiarities mentioned under the last head. Thus, there is excess of urate of soda in the blood before and during gouty explosive manifestation, and there is determination (by nervous influence, in all probability) either of this salt to the affected part (*Garrod*[1]), or there is a too free formation of it at these inflammatory points, whence it is deposited locally, and also set free into the circulation (*Ord*).

The renal excretory power for uric acid appears to be temporarily inhibited as part of the process of gouty paroxysm. This measure of renal inadequacy would appear to prevail in varying degree as a part of the specific neurosal disorder. In chronic gout, when structural disease has occurred, either tubal, with deposition of urate of soda, or interstitial, with shrinking of the organs, the renal inadequacy may admit of more mechanical explanation.

Sixthly, That in primary, or inherited, gout, the toxæmia is dependent on the gouty neurosis; is the outcome, in whatever degree, of it, and is therefore a secondary manifestation.

Seventhly, That in what I term secondary or acquired gout, the toxæmia is directly induced by such habits as overload the digestive and excretory organs, and constantly prevent complete secondary disposal of nutritional elements of food; that if, together with such toxæmia, distinctly depressing and exhausting agencies, affecting the nervous system, come into operation, the special neurotic manifestations of the gouty diathesis will occur, and be impressed more or less deeply upon the individual and his offspring.

Eighthly, That this theory of gout, better than any other, correlates all the known factors concerned in the production of the varied symptoms of the malady; and while it displaces its humoral pathology from the pre-eminence it has so long occupied, it takes full cognizance of it, and seeks to place it in a clearer relation to the phenomena of the disease.

Ninthly, That if it be desirable to refer various maladies to their distinct place in pathology, without reference merely to their chemistry, histology, or neurology, the affection known as gout

[1] *Op. cit.*

may perhaps most correctly be relegated, along with some others, to a class of diseases which may be termed neuro-humoral.

On reviewing these several propositions, I am, at this distance of time, disposed to be less dogmatic respecting some of them. I stand firmly by the position that gout owns a nervous as well as a humoral pathogeny. I am not now so strongly disposed to insist on that part of the theory which tended to localize definitely the actual centre of disturbance in a limited portion of the cerebro-spinal axis. Nor am I prepared to shift my ground, and claim any other definite tract of the nervous mass as the affected and unstable centre. I must freely admit my inability to do so, and would express myself more cautiously in deference to the opinion of the eminent Parisian professor who did me the honour to criticize my theory. He reproached me for seeking to localize "at too limited a point the primordial lesions of a malady essentially general, one which is, and always will be, typical of one of the best-marked of all diathetic conditions." While, however, ceasing to insist on this part of my theory, I still look kindly upon it, because many of the features of arthritic diseases afford support to it, and none can doubt that the medulla oblongata consists of series of centres, some amongst them possessing intimate relations to the most distant and varied organs and structures of the body.

I think it not only helpful, but absolutely essential, to add to the purely humoral views which have chiefly prevailed respecting the nature of gout a conception of the presiding nervous element. In accepting this, we greatly enlarge our point of view, and explain some of the most difficult points which have hitherto perplexed careful inquirers. For my part, I cannot dissever the two ideas, and, hence, I affirm that gout is a neuro-humoral disease.[1]

It is, without doubt, the case that, hitherto, no theory has been set forth which appears to embrace all the multiform phenomena of gouty disease. The greatest advance of modern research has been to establish the certainty of some special relation to it, in the greater number of instances, of uric acid, and so far there is clear warrant for retaining a measure of humoral pathology in our conception of the malady.

I propose to discuss, *first*, the arguments which lend support to the view that the nervous system is largely involved in gouty pathogeny; and in doing so, I shall have to refer to many points which must later on engage our attention more in detail.

[1] In respect of rheumatism, I am prepared to affirm the same, thus explaining the manifestations of that branch of the basic arthritic diathesis.

Secondly, I shall treat of the pathogenetic relation which uric acid or its salts bear to gout.

The best approach to the line of argument I purpose to take up in the first place will manifestly be to review the special characters of neuroses in general, and then to examine, coincidently, how far the well-ascertained features of gout conform to such characters.

Before proceeding to this analysis, I would first assert that gout is something beyond the resultant effects of aberrant relations of uric acid; that it consists in something more than a perversion of animal chemistry; that it is not to be explained as a mere outcome of gastric or hepatic distemper; and that it is not the appanage only of the middle-aged or elderly high-liver and intemperate drinker, because, as is well known, it affects also, sometimes in early life, the high-thinker and the laborious bread-winner. Without doubt, while accepting all (and that is much) that it is good for, one is impelled to look beyond what may be termed the chemical pathogeny of gout. The researches into the nature and functions of the nervous system, as carried out during the past quarter of this century, come to our aid at this stage of our inquiry, and, amongst these, we have learned two main and important points respecting the neuroses in general. The first is, that they may be primary or central, and the other is, that they may be secondary or induced. In other words, it may be averred that a neurosis is implanted, or a tendency to it established, and this shall be handed down, hereditarily passed on, and, thus, a diathetic tendency be formed; or, owing to some toxæmic condition or blood-degeneration, a secondary or induced neurosis may be established.

It is on this basis that I shall endeavour to establish that part of the pathogenic theory of gout which relates to nervous influence.

Representing special conditions, or rather special morbid modes of evolution, of nerve-force, neuroses are implanted in the individual as a part of his intimate nature. They belong to the individual, and are characteristic of him in the same degree that are his features and other physiognomical traits. An implanted neurosis is, as it were, the representative of a morbid physiognomy for the cerebro-spinal axis. A neurosis, then, is a peculiar disposition or tendency on the part of the nervous system, or some definite tract of it, towards morbid evolution or manifestation of nerve-functions. It does not necessitate the existence of any coarse disease, directly obvious to the eye, but it is a more

or less abiding condition, ready to come into action upon suitable provocation.

It is specially characteristic of neuroses that, being thus primarily impressed upon an individual, they tend to be transmitted by heredity. It has been alleged that the female sex is more neurotically disposed than the male; but facts do not support this opinion thus broadly put forth. Certain neuroses appear to prevail with greater frequency in males, and others in females; and not only so, but in the case of those that are common to both sexes, the manifestation occurs at different epochs of life.

Thus, some outbreaks of neurotic disorders are seen to occur at the several septennial climacteric periods, at the times of dentition, puberty, and often at the grand climacteric. In this manner an element of distinct periodicity attaches to neuroses in general.

Further, a most marked feature in all neurotic affections is that of paroxysmal tendency. Thus, there is the abiding element, with proclivity to recurring outbreak.

Again, it is certainly known that a law of alternation or substitution prevails in neuroses, and thus we meet with certain neurotic affections in the parent or ancestors, and with others in the collateral relations or descendants. We thus have to deal with distinct types of nervous impression. These abiding conditions are more or less prone to be excited into activity according to various circumstances.

It is not difficult to understand the course pursued by a neurotic taint, once laid down or impressed; but it is not so easy to conceive the original implanting of such a morbid functional tendency. The mischief, however, is constantly originating in individuals, and as constantly undergoing further development, modification, or even repression.

Excessive activity of the nervous system, or of any part of it, as Laycock has shown, becomes a highly-disposing cause of the neuroses. Habitual or prolonged excess develops hereditary tendency. *Undue mental labour, gluttony, alcoholic intemperance, debauchery, and other indulged evil propensities in the parent, come to be developed into definite neurotic taint and tendency in the offspring.* Particular examples of this are not far to seek, and amongst them comes out the disorder so widely and variously manifested as gout. According to this view, for which I plead, gout appears as a diathetic neurosis, and a link in the long chain of its phenomena, so long missing, is now forthcoming.

I have already stated that there is clear warrant for retaining,

as *part of the pathology* of gout, a humoral hypothesis, and it may perhaps be applied and relegated to its proper place, as follows. Granting that gout in any individual is the outcome of a central neurotic taint ("primordial vice of nutrition"), we have the ordinary manifestations of it more or less severe. This we may term primary or central gout. The tendency may be transmitted or modified, or, conceivably, may be allowed to die out.

In another individual, gout may "grow up" where previously there was no neurotic taint or tendency. A patient is commonly said to earn his gout by high-living and over-indulgence of appetites. In this instance a morbid blood-state is induced, and excess of uric acid is generated.

But is this all? Is this enough to explain all the phenomena which we recognize clinically in gouty disease? I believe not. We are compelled at this point to widen our view, and are driven, perforce, to invoke the operations of the nervous system. Having arrived thus far at nothing beyond a special toxæmia, we must drop humoral pathology, and seek for the effects of the blood-dyscrasia upon the nerve-centres. And we have full warrant for this course in contemplating the analogy of other toxæmic states, together with their effects upon the nervous system. The nutrition of this system is plainly affected by morbid blood-conditions, and thus expression is given to such poisoning in the form of convulsion and other nervous symptoms.

I venture, then, to suggest that a secondary affection of some nerve-centre occurs as a consequence of the altered blood state *ab intra*, and that thus the order and particular process of the gouty attack is evolved. This we may term secondary or acquired gout. A diathetic neurosis is thus impressed upon the individual,[1] and we witness the results of a vicious circle of events in the economy.

It is certainly a matter of much interest to study side by side with gouty processes the several joint-affections or arthropathies which have come to be regarded of late as of distinctly spinal or otherwise nervous origin. It seems impossible to separate gouty arthritis from this connection. And if it be conceded that this particular form, which is but one of many others, is truly and directly dependent upon nerve-influence, the greatest part of the difficulty in establishing a neurotic theory of gout is forthwith

[1] The frequency and severity of gout in England is explicable on the view of the impressed neurosis. The habits leading to gout—high-living, intemperance in strong drinks (malt liquors and wines), along with much mental energy—have certainly prevailed more, and amongst larger classes, in England than in either Scotland or Ireland.

removed. I suppose no greater obstacle has stood in the way of the acceptance more generally of a nervous theory of this malady than the impossibility hitherto of connecting arthritic disposition with any form of neurosis. So many of the other, and less obvious, manifestations of gout are plainly dependent on nervous influence, that the whole phenomena now appear to fall more naturally into their places.[1]

It is, however, only right to mention here that thoughtful physicians have long ere now conceived the special action of nerve-influence on joints, and of arthritic affection on nerve-centre.[2] The relation sometimes existing between rheumatic fever and chorea is an example in point, as Dr. Liveing has shown.

Much light has been thrown of late upon spinal arthropathies by the researches of Charcot, Ball, Weir Mitchell, and of Dr. Ord. The latter has contended for a more scientific revision of our present views upon the pathology of chronic rheumatic arthritis. His views are not only eminently ingenious, but they accord remarkably with well-observed clinical facts, not hitherto correlated.

As Sir James Paget has remarked, the changes in the nerve-centres, which determine the locality of the gouty process, are a part of the pathology of gout which is not yet clinical. They are, therefore, no more than speculative at present, but we gain much from the prosecution of an inquiry in this direction.

With respect to the particular locality affected in the arthropathy of locomotor ataxia, there is some discrepancy of opinion. Charcot has declared for implication of the anterior cornua of the spinal chord. Dr. Buzzard, however, has not confirmed this opinion, and, guided by the noteworthy frequent association of gastric crises with joint-affections in this malady, as previously observed by Dr. Ball,[3] has suggested a sclerosing lesion, involving the roots of the vagus in the medulla oblongata, in close relation to some trophic centre that may be localized there, presiding over the osseous and articular systems. And he further indicates the bond that may thus exist between implication of joints and such metastasis as may occur to the heart in rheumatic fever: also the occurrence of hyperpyrexia, which is sometimes present in such cases. We have yet to seek for

[1] "The preference which tophus shows for the joints is a remarkable fact, which we cannot explain, and which presents a great subject of reflection to physicians."—*Trousseau.*

[2] Liveing, *op. cit.*, p. 247.

[3] Med. Times and Gazette, vol. ii. (1868); vol. ii. (1869), p. 498.

this hypothetical nutrient centre for joints, but in the meantime we are fairly warranted in widening our view, and in directing attention to the high significance of predicating such a trophic centre.

"Discovery by true analogies is always progressive, . . . one analogy leads on to another investigation and arrangement of phenomena, and another analogy."[1]

It remains now to be shown, more in detail, how the phenomena of gout conform to the recognized manifestations of the neuroses in general.

It can be shown, I believe, that the plea for the neurotic element in true gout is not difficult to establish.

First, there is to be considered the marked tendency of gout to be hereditarily transmitted. This is notorious. The disorder may pass from either parent, and may be mingled with other taints and tendencies passed on from the progenitors.[2] The outbreak may occur in slight or in graver degrees, and may be deferred, overtly, till even the thirteenth climacteric period. Thus, the first plain attacks of gout may not appear till the patient is sixty or over ninety years of age. In all such cases, however, I am convinced that many minor tokens of the disorder have been overlooked in previous years, all of which are sufficiently obvious to the trained clinical eye. As a rule, the manifestations are prone to occur at definite ages in each sex, most commonly in the fourth decade in men, and in the fifth in women. My own experience appears to show that gout is frequent in men early in the third decade.

Certain peculiarities attending gouty transmission are deserving of study. Mr. Hutchinson has called attention to one of these in a suggestive lecture.[3] He expresses his belief that what is transmitted is not the active gouty dyscrasia itself, but rather a sus ceptibility to the influence of certain exciting causes, together with some peculiarly disordered condition of the assimilating and excretory viscera, which renders them unable to deal with particular articles of food. Now, this special susceptibility to definite exciting factors is neither more nor less than a nervous peculiarity,

[1] Laycock, *op. cit.*, p. 190.

[2] "If the countenance, the outside, is hereditary, why not the inside?"—*Letter to Dr. Cadogan, occasioned by his Dissertation on the Gout, &c.*, London, 1771. (Cadogan tried to show that gout was not hereditary.)

"Hereditariness of gout is like the hereditariness of a Roman nose; it is part of a family likeness; the particular chemical type, the particular mode and rate of the chemical change of tissue, passes from father to son as the shape of features passes."—*Sir John Simon, Path. Soc. Trans.*, vol. xxvii. p. 419.

[3] Medical Times and Gazette, vol. i. p. 543, 1876.

of which the chief character is its liability to break away in certain morbid directions—its *instability*, in short. This is, I submit, the gouty neurosis. Mr. Hutchinson further believes that gout is wont to show itself with greater frequency and in more marked form in the younger than in the older members of a gouty family, the diathesis strengthening in the parent with advancing years. I can confirm this observation.[1] Resemblance to the gouty parent has been specially recognized in those of the offspring most distinctly affected.[2] In other members of the family the tokens of gout may be present, but less marked. These facts are, of course, in accordance with ordinary laws of hereditary transmission. Dr. Wickham Legg has called attention to the fact that gout, like hæmophilia, pseudo-hypertrophic paralysis of Duchenne, and some other affections, is not unfrequently found to be transmitted by the female line, although especially manifested in males, the mothers themselves being unaffected by readily recognized gout.

A noteworthy feature in gouty ailments is their sudden supervention. As in epilepsy, not uncommonly, the patient often feels remarkably well, and realises his sense of *bien être* before the outbreak suddenly takes place. This euphoria, or delusive corporeal satisfaction, is itself a nervous derangement. Explosiveness is a distinct feature in several of the neuroses, and attaches to such ailments as angina pectoris, asthma, epilepsy, and various neuralgiæ.

The time of the occurrence of the attack is also strongly marked. The majority of the outbreaks take place in the early morning. This is true both of grave and classical cases, and also of many of the minor forms of gouty trouble. The same thing is met with in asthma, neuralgia, and in epilepsy. The pyrexia proper to acute gout is paroxysmal, with remissions, and the pain of gout is likewise paroxysmal. One is here reminded of the influence of marsh-poison upon the nervous centres.

This paroxysmal, no less than the periodic, element in gout, stamps a nervous character upon the malady, and binds it in alliance with other neuroses.[3]

An important connection of the same kind is seen in the unquestionable commingling of gout with other well-recognized neuroses. Thus, hemicrania is sometimes distinctly a manifestation of gout in both sexes, and may be the form of neurosis

[1] Cases illustrating this are given by Sir Spencer Wells, Bart., *op. cit.*, p. 18

[2] *Op. cit.*, Sir Henry Holland, Bart., M.D., F.R.S., 3rd edit., 1855, p. 29.

[3] *Vide* Scudamore, *op. cit.*, p. 152.

impressed upon an individual whose parent was gouty, or may itself alternate with gouty arthritic attacks in the same person.[1]

It is not far to seek for an allied condition of trophical lesion in herpes zoster, itself the outcome of disordered innervation.

The doctrine of metastasis must next be considered in relation to gout. The pure humoralist seeks to explain this clinical fact upon his theory, but such is manifestly insufficient to account for the phenomena. It must be conceded that some nervous law regulates the occurrence of shifting inflammation. It has been supposed to be due to reflex influence. Some distinct predisposition to take on the morbid action exists in the part selected, apparently, by caprice. The same class of tissue is apt to suffer. Thus, the gouty or rheumatic process flies from joint to joint, or, as in gouty phlebitis, from vein to vein, sometimes symmetrically, but not always. The serous and fibro-serous structures suffer especially, but also mucous surfaces. Laycock has shown how these several tissues are related embryologically, and are thus prone to suffer in common when diathetically impressed.[2]

Localized trophical changes follow locally acting causes of depressed nervous power. Thus, impairment of certain centres may lead to the specific nutritional changes witnessed in metastases, and, thus, the apparent capriciousness is explained in this process.

Amongst the nervous symptoms of gout must next be considered the occurrence of certain sensory perversions, such as tingling and numbness of the fingers and toes, sensations of heat in the palms, thighs, and soles (paræsthesia), and tickling in the throat. As pointed out by Sir James Paget, "gout affects the sensory much more than the motor elements of the nervous system;" and he remarks, too, that the pain of acute gout is seemingly out of all proportion to the amount of inflammatory process in the affected part. So, too, all other disorders, modified by gout, seem to be especially painful; for example, cancer, as pointed out also by Paget.

Grinding of the teeth is met with in the gouty. Graves first observed this.[3] Dr. Donkin has recorded cases associated with somnambulism,[4] and I have intimate knowledge of two others in which the same phenomena are manifested—the gnashing of teeth and somnambulism in a sister, and the talking in sleep in

[1] Stahl, *op. cit.*, § xxxvi.; Trousseau, Clin. Méd.; Liveing, *op. cit.*, &c.; Sir H. Holland, *op. cit.*, Relation of Asthma to Gout, p. 36.

[2] *Op. cit.*, p. 196.

[3] Clin. Méd., p. 351, edit. 1864.

[4] Brit. Med. Journal, Feb. 21, 1880, p. 279.

the brother. The maternal grandfather and the mother are distinctly gouty. Cramps in the muscles of the legs and priapism are amongst nocturnal manifestations in the gouty. Of insomnia, due to gout, there is much to say. It was originally noted by Cullen, and it conforms remarkably with other periodic neurosal phenomena.[1]

Gouty neuralgia is largely recognized, and is known to be both severe and prone to recur. It is frequently occipital, and is met with in the heel, tongue, breast, arms, and more often in the great sciatic nerve. One proof, amongst others, of the truly gouty nature of these is gained from the fact that they yield most readily to anti-gouty medication, and another lies in the frequency with which they are provoked by conditions which elicit other gouty processes.

Amongst the strongest evidences of gout depending upon nervous influences are the unquestionable facts bearing upon the induction of its attacks.

The influence of many of the existing causes of gouty paroxysms illustrates well the explosive character of the malady. As Sydenham expresses it, before the onset of an attack, "totum corpus est podagra." The precipitation of the seizure sometimes ensues almost immediately upon the provoking cause. In a large number of instances, the latter is of a nature *to depress nervous power*. Thus, unwonted muscular energy, prolonged exercise, stirring emotions, fright, undue excitement, venereal excess, rage, worry and vexation, are all excitors of gouty paroxysm. So, too, sudden shock to the body, as from injuries and surgical operations, will evoke gout. Dietetic errors are well recognized as factors; thus, a full meal, and excess or mixing of strong liquors, will act in upsetting the equilibrium of a quiescent gouty habit. It will be conceded that many of the causes just enumerated are equally potent to elicit manifestations of other neuroses, such as epilepsy, asthma, hemicrania, and angina pectoris. The provoking agency, however, need not always be primarily exhausting. In proof of this, the outbreaks of gout following hydropathic treatment, internally or externally, may be instanced; and, indeed, the causal element need only be such as shall induce some change in the acquired vital habits.

Thus it is that the subjects of the neuroses hold much of their comfort in life by following a very equable routine. They are prone to give way under any extraordinary pressure.

[1] *Vide* author's paper, St. Barth. Hosp. Reports, *jam cit.*, p. 105; and Brain, July 1881.

These considerations explain, in part, why men are more liable to gout than women. They carry on the world's rough work; are engaged in more exciting occupations, and have commonly the greater burden of anxiety to bear.

The more sedentary the occupation, the more profound the mind-working, and the more intense the strain of life, the greater the tendency to nervous depression, and to the peculiar form of its expression in gout. If to such habits be added high-living, as often occurs in the cases of eminent statesmen, lawyers, and speculators, no link is wanting in the chain of causation, and all the elements for gout are present.

Climatic influence is important amongst these agencies. The dull and "shifty" weather and the cold east winds of northern latitudes are certainly bad for gout. The nervous depression ensuing upon months of sunless skies—negation of light powerfully lowering nervous tone—is too little regarded as an element of devitalization in England. The cutaneous eliminant power is checked, and, so, aberrant chemical relations are apt to be determined in any parts specially prone to gouty invasion.

The same mal-determination ensues upon the suppression of various discharges, whether from the uterus, from hæmorrhoids, or other sources.

The mental condition of the goutily-disposed is a subject worthy of attention in relation to the pathology of the ailment.

Hypochondriasis has long been associated with gouty taint. It commonly precedes an outbreak, and disappears subsequently. A tendency to sighing has also been observed, and is a plain indication of nervous exhaustion. Hysteria has also been observed to precede gouty attacks in women, and to disappear with the onset of articular symptoms.[1]

Irritability of temper is a proverbial condition in the gouty, and furious outbursts of this kind appear to be, at times, a metamorphic substitution for a more overt and regular attack. It is important to know that many of the minor, but none the less well-marked, phases of gouty paroxysm are in no degree arthropathic. Much error in diagnosis has arisen from taking no heed of any but articular symptoms when searching for gouty tokens in a given case. These less classical attacks very commonly precede the onset of typical ones at a later period in life. The necessity for prompt recognition of these less well-expressed symptoms is obvious, if good treatment is to be applied.

[1] On the Relations between Gout and Hysteria, *vide* Treatise on the Nervous Diseases of Women, p. 163. By T. Laycock. 1840.

Epilepsy has been known to disappear on the supervention of gout.

Sensations of giddiness and dimness of vision, not uncommon in the gouty, are noteworthy in relation to nervous symptomatology.[1] So, too, the disturbances of the cardiac rhythm, and the co-existent (neurotic) vascular throbbings which are sometimes met with. The cardiac irregularity has been noted to cease with the induction of a regular attack. Dysphagia was noted in connection with gout by no less careful an observer than the late Dr. Brinton.

A consideration of the effects of lead-impregnation in relation to gout, and of the certain liability of the gouty to be more readily than others influenced by lead, leads to the belief that the nervous system is specially implicated in these relations. The fact is, that the blood is imperfectly freed from uric acid in cases of lead-poisoning, and that gout is thus quickly evoked. Garrod has fully established these facts, and all physicians now recognize them. The lead-influence, clearly through nervous agency, induces the measure of renal inadequacy which is, probably with correctness, acknowledged as a factor in gout. And the knowledge that this metal is capable of setting up special paralysis, epilepsy, coma, and other cerebral phenomena, is of the largest interest in relation to this subject.

I now approach a point in connection with the whole pathology of gout which merits much consideration. The connection of diabetes with gout has been recognized for some years.

I object to the term diabetes as applied to the special form of glycosuria associated with gout. The disorder is met with in certain members of gouty families, some having regular gout, and others manifesting less regular gout, or this alternating with glycosuria. I believe that many cases of temporary glycosuria are due to gouty conditions. The fleeting presence of glucose in the urine of many elderly persons may be thus explained. It has long been recognized that such an affection, which, in many instances, is undeserving of the name *diabetes mellitus*, for the simple reason that there is no *diabetes* in the strict sense of the term, is not really a grave one. *The presence of glucose is found to alternate with that of uric acid.* In the aged but little importance should be attached to the symptom. Charcot testified to this, some years ago, in his excellent lectures on the maladies of old people, his experience being gathered at that fertile school of

[1] Trousseau, Murchison, Paget *op. cit.*; H. Mayo, Philosophy of Living, p. 24, 1837.

study—the Salpétrière. In persons under forty years of age, however, glycosuria, even of gouty origin, is a most grave matter, and merits the closest attention, since it may eventuate in confirmed diabetes. It is the rule to find that the quantity of urine passed is not much, if at all, above the normal, but the specific gravity may range from 1.035 to 1.050. An anti-gouty treatment is called for, for the glucose may otherwise only give place to uric acid or increased azoturia, and the gouty habit has rather to be attacked than the glycosuria.

Dr. Lauder Brunton has called attention to this class of cases.[1]

The alliances of gout and diabetes are sufficiently intimate. In both the doctrine of heredity applies, and the nervous system is involved. The same habits lead to each, the same classes of person are affected, and the same exciting causes are potent to evoke both. A consideration of these facts naturally leads to the belief that the portions of the nervous system involved in each cannot be far apart from one another. The medulla oblongata, the sympathetic and splanchnic nerves have been found chiefly affected, and the spinal chord likewise in some instances. The point for the diabetic puncture in the medulla is believed by physiologists to correspond to the vaso-motor centre in the same structure.

Guided by these facts, and by the knowledge that the glycogenic function of the liver is under nervous influence, by the advancing theories which refer special arthropathies likewise to the same influence, and bearing in mind Dr. Buzzard's views, previously stated, in connection with the gastric crises so commonly associated with the arthritis of locomotor ataxia, I venture upon the hypothesis that the portion of the nervous system which is specially predisposed to the irregular mode of action known as gout, has its seat or centre in the medulla oblongata.

A point of difference between the arthritic affections which are now referred to nervous influence and those manifested in gout, is found in the fact that the latter appears to have an elective affinity, often unilateral at first, for the smaller joints, especially that of the big toe, while most of the others influence the larger ones. Herein, perhaps, lies part of the specificity of gout.

The trophical results of the latter are often seen impressed upon the physiognomy, and upon certain tissues, in a manner extremely definite and characteristic.

Thus, are found the large head, the thick hair, with tendency to early greyness, the large, full veins, the long uvula, the soft,

[1] Art. "Diabetes Mellitus," Reynolds' Syst. of Med., vol. v. p. 381, 1879.

smooth skin, the thickened extremity of the nose, and the lineated, brittle nails.

Lastly, I may add an argument from the therapeutical side.

The universally acknowledged specific action of colchicum in gout is known, owing to Garrod's researches, to be due to no power which it possesses of causing elimination of uric acid. Gouty inflammation is therefore influenced by it without reference to the secondary aberrant relations of uric acid. The active principle or alkaloid of the drug colchicina is a member of a nitrogenized group of bodies to which veratrina, strychnia, quinia, and morphina have close chemical alliance.[1] They all powerfully affect *the nervous system.* Colchicum acts very promptly, and affords often decided relief to the intolerable pain of the gouty process. When taken in health in small doses, Dr. Meldon and others have found that it induces a general glow at the surface of the body, diaphoresis, throbbing of the blood-vessels, and palpitation. Subsequently there is reduction in the force and frequency of the pulse. Dr. Meldon observed in his own case an invigoration of his mental energies. In larger doses, the effects are most marked along the whole tract supplied by the vagus, and thus cardio-vascular, gastric, and enteric symptoms ensue.

The peculiar benefit derived from this drug is not secured in any other form of inflammation, and thus it is plainly specific. Its cherished action is doubtless exerted upon the vaso-motor nerves.

The manifestly good influence of all agencies which cheerfully inspire the mental condition in the goutily disposed, must not be omitted from consideration amongst the *juvantia* both of prevention and cure.

It may be affirmed, in opposition to the views expressed as to the neuro-pathogeny of gout, that the disease is met with in persons, and under conditions, where no such high-strung state of nervous system as predicated exists. Sydenham declared that gout rarely attacked fools.[2] This is not quite in accordance with facts, at least nowadays ; but even in fools the great trophic processes of the body are dominated by a nervous mechanism. And if it be urged that such a conception is high-flown, and unnecessary for practical purposes and treatment, I would remark, that the therapeutic art is daily practised, and with good results, by

[1] *Vide* Lectures on Pathology and Therapeutics. London, 1867, p. 137. H. Bence Jones, M.D., F.R.S.

[2] The same may be said in respect of *delirium tremens*, which is most often met with in men of superior mental ability.

many who can give scant scientific reasons for their dealings. I will humbly venture to include myself in this category. In this case, however, I believe that the enlarged conceptions gained by due regard to the neuro-pathogenic element in gout cannot fail to avail us at the bedside.

I now proceed to discuss, secondly, the pathogenetic relations of uric acid to gout.

Of late years our knowledge of the physiology and intimate chemical relations of uric acid has received important additions. It is ascertained to be, like urea, a simple derivative of the disintegration of albuminous tissues or of albuminous food-elements (proteids). It is a crystalline compound, white in colour when pure, consisting of $C_5H_4N_4O_3$. Although it has been asserted, it is not proved, that uric acid is an antecedent of urea in the normal nitrogenous metabolism of the body. It occurs as a normal constituent of the urine, being excreted to the extent of a little over eight grains in twenty-four hours, chiefly in the form of sodium biurate, the acid joining part of the base of the alkaline phosphate of sodium existing in the blood. Whether this conjugation occurs in the general circulation or in the kidneys, is not certainly known. A prominent character of uric acid is its insolubility. It is a bibasic acid, forming with bases both neutral and acid salts or biurates. Ammonium and sodium urates are found in normal urine, the former being the less soluble of the two salts. Potassium urate is more soluble than either, and lithium urate is the most soluble of all. Neutral uratic salts are very unstable. Acid urates or biurates are the most stable salts. Neutral sodium urate is more endosmotic than the biurate sodium salt, and the latter is readily formed (and with it gouty manifestations) from the former, if the blood becomes from any cause less alkaline.

Uric acid requires 15,000 parts of cold water and 1800 parts of hot for its solution. Alkaline fluids readily dissolve it, also sodium phosphate. When excreted in excess in urine, it is deposited variously as rosettes, rhombs, or diamond-shaped crystals, stained by urinary pigments.

The ammonium urate of the urine is believed to be partly formed in the kidney. The sodium salt is that possessing pathological interest in gout, constituting the essential ingredient of tophi. It is met with in the blood of the gouty, but also under other conditions quite apart from this disease. It crystallizes in fine prismatic needles, and constitutes, sometimes with lime salts, the bulk of the gross uratic deposits met with in the tissues

of the gouty. These deposits possess the property of doubly refracting light when examined by the polariscope. Sodium biurate is soluble in 800 parts of cold water. Dr. Tichborne, of Dublin, has found that, at the temperature of the body, one part of uric acid is soluble in 1660 parts of water, and that sodium biurate is much more soluble at the same temperature.[1]

It is important to note that nowhere in health is uric acid met with as such. The presence of free uric acid anywhere in the body, or in any secretion, is a sign of disease. As is well known, in both the mammalia and herbivora the quantity excreted is small. In birds, especially the granivora, and snakes, the amount passed is large, exceeding, or entirely replacing, urea. Together with urea, the quantity is increased by nitrogenous or animal diet, and diminished by non-nitrogenous or vegetable diet. It is also greatly increased during febrile conditions.

Uric acid has been synthetically produced out of the body by Horbaczewski of Vienna, and by Dr. Latham of Cambridge.[2] Glycocine and urea heated together are found to yield it. Glycocine never occurs free in the body, but is derived from glycocholic acid, which, in conjunction with sodium, is one of the natural salts of the bile. The liver salts, glyococholate and taurocholate of sodium (bilin), are decomposed in the duodenum, splitting up respectively into cholic acid and glycocine, and cholic acid and taurine. The glycocine and taurine are absorbed by the portal vein, while the cholic acid passes off by the intestine. There is increasing evidence to show that the chief antecedents of urea are partly kreatine, a primary product of muscular and other disintegration, and leucine and tyrocine derived from the alimentary canal. It has been surmised that both urea and uric acid start from a body containing some of its nitrogen in the form of cyanogen, and that urea, as being more soluble, is adapted to the fluid urine of mammals, while uric acid is better fitted for the solid urinary excretion of birds and reptiles.

According to Dr. Latham, the occurrence of uric acid in the urine is due to defective transformation of glycocine into urea. Glycocine passes unchanged into the liver, where it is conjugated with urea derived from metabolism of other amido-bodies, leucine, tyrosine, &c., and is converted into hydantoin, which is readily soluble, and passes on into the circulation to combine

[1] Lancet, Nov. 19, 1887, p. 1097.

[2] Croonian Lectures, Roy. Coll. Phys., p. 57. London, 1886.

in the kidneys with other molecules of urea to form ammonium urate. A portion of this, he thinks, overflows into the circulation, all not being excreted, and combines with sodium in the blood to form urate of sodium.

It has been proved by Schroeder, in the case of birds, that uric acid is not specially produced in the kidneys, but in the tissues generally.

H. Ranke has maintained that the spleen is an important seat of uric acid-production, and he was led to this belief by the fact that in all cases of splenic enlargement there is increased excretion of the acid. This latter fact has been confirmed in cases of splenic and lymphatic leuchæmia, and it is also found that this excretion diminishes in intermittent febrile attacks, and is subdued by quinine. Deficiency of red globules carrying oxygen has been suggested as a cause for this. It may, therefore, be surmised that the spleen and blood-glands generally take part in the formation of uric acid, and that this process is associated with blood-formative rather than with blood-destroying function. Dr. Haig is of opinion that uric acid when retained in the body is largely held back by the spleen. The liver is, however, the organ in which, in health, uric acid is chiefly formed, and it is probably to derangement of function in this gland that we must look for over-production of this substance. As already stated, Murchison suggested that functional disorder of the liver not only caused deficient secretion of bile, but also interference with the normal metabolism of albuminous matters, so that uric acid, a less oxydized product than urea, was formed in excess.

The causes that lead to disturbance of hepatic function are, in the great majority of instances, those that lead to gout; but in many persons such functional disorder of the liver does not induce gout, at least in any classical form. It does, however, lead to many symptoms which have been long recognized amongst those of imperfect gout. Such are certain headaches, depression of spirits, forms of migraine, pains, palpitation, cramps, vertigo, and insomnia, all capable of removal by treatment addressed to the liver, which is commonly somewhat tumid and painful in these cases. It may fairly be asked why persons who thus suffer from what has been termed lithæmia yet develop no overt, but only incomplete gout? The reply is that such symtoms, though gouty in character, occur in persons who are not truly or completely gouty. In many of such cases, persistence in the habits leading to the hepatic distemper will, and indeed

does, ultimately lead up to overt gout. The tendency to such disorder is probably in many cases but a slightly marked predisposition to gout, which may be actually inherited. Murchison believed that this tendency to hepatic disorder was hereditary, and I agree with him. The urine in such cases deposits uric acid and biurates of sodium, ammonium, potassium, and lime. The presence of such deposits does not always indicate excess of production in the body. They may occur in pyrexia, or after exercise and sweating, simply from concentration of the urine. Oftener the cause is an error in diet, whereby more nitrogenized matter is conveyed to the liver than can be duly transformed there, especially of saccharine and alcoholic matters in combination, of fats and fruits, which interfere with due chemical transformations, and induce acid dyspepsia. The same result follows a gastro-enteric catarrh from the effects of cold. Diminished action of the skin in cold weather is another well-recognized cause, leading to increased acidity of the urine. Many of these are examples of temporary lithiasis, which may occur in persons having no claim whatever to gouty predisposition.[1]

Lithæmia, then, even when persistent, and not due to accidental causes, is not by itself gout. The muddy and loaded urine of the former state is commonly of higher specific gravity, and

[1] Sir William Roberts has lately confirmed some interesting experiments of Dr. Bence Jones, which indicated that the amorphous urate deposit is not wholly or chiefly composed of true biurates, but consists of quadurates, or a complex compound, in which biurate was united in loose combination with an additional equivalent of uric acid. Amorphous urate treated with water commonly, but not always, throws out uric acid, and leaves the associated biurate in solution. Roberts is inclined to believe that uric acid exists in urine in the form of quadurates, and he has found that the urine prevents the decomposition of these more or less rapidly, according to its density. Urine of low density permits rapid change, while that of medium density only acts slowly in throwing out uric acid from the deposit. This fact, which has been long known, was formerly attributed to acid fermentation. This inhibitory action of the urine is believed by Roberts to be due to its crystalloids—urea, chlorides, sulphates and phosphates of sodium, potassium, calcium, and magnesium; and he thus accounts for the non-precipitation of uric acid in the urinary passages and bladder, decomposition being delayed. The occurrence of uric acid in a free state in the urine as a biurate is thus attributed to subsequent changes in quadurates which take place in the urinary passages or after emission. "Quadurate is very unstable and susceptible to the disintegrating power of water. In the presence of alkaline bicarbonates it slowly takes up an additional atom of base, and is thereby wholly converted into biurate." The varying state of the urine from time to time as to reaction and concentration will affect the quadurates dissolved in it, and so, Roberts believes, lead to formation of gravel or stone. Abundance of salt in food or drinking-water has been shown to prevent calculous disease, and Roberts conceives that urinary pigments, for which urates have great affinity, also take part in preventing decomposition of these salts. (On the Amorphous Urate Deposit, *Medical Chronicle*, March, p. 441. Manchester, 1888.)

very different from that usually met with in the gouty, which is clear and bright, and apt sometimes to deposit uric acid. The fact that lithiasis is frequent in persons who have no claim to gout, as in children and temperate livers, has led to the belief that there is no connection between the tendency to this state and that to gout. I am sure, however, that it is unjustifiable to insist on this conclusion. I believe, with Murchison, that the tendency to hepatic disorder inducing lithæmia is hereditary, and it is certainly met with in the children and descendants of the gouty. Hence, I conceive of a rather close relation between the two states, so far, at least, that the tendency to lithæmia in early life may be an early expression of the gouty diathesis. In the large cities of the United States, lithæmia is alleged to be common, while gout is little known. I think it not unlikely that gout will become more common in that country in course of time. Gout is a disease of old and long-settled countries.

The lithiasis often seen in rickety and strumous children is probably due to deficiency of alkaline phosphates, as pointed out by Ralfe.

The tendency to persistent or severe lithiasis is often inherited, as is a gouty proclivity. In the families of those thus affected, or who pass gravel and develop urinary calculi, it is very common to find history of gout and gravel, so that it is impossible to regard these two affections as unrelated, and the one condition may most certainly precede, accompany, or follow the other in the same individual. Temporary cessation of gouty troubles may supervene on formation of calculus.

Where persistent lithiasis is well marked in early life, it naturally indicates, as in the case of gout, a strongly inherited tendency.

Gout and gravel are, moreover, apt to alternate in succeeding generations.

Uric acid is present in least quantity in the body in the highest conditions of health. In disease, urates commonly increase, and this is an indication of a lower level of metabolism, constituting, as Sir William Gull has remarked, a degradation to a lower animal type.

The fact that uric acid is met with (in the form of salts) in the blood both of healthy persons as well as in those suffering from morbid states other than gout, has made it difficult to believe that blood thus surcharged is alone to blame for all the disturbances recognised as gouty. This fact must be admitted; and, hence, a purely humoral doctrine is, I hold, inadequate to explain the entire pathogeny of gout.

We have now to inquire whether, and if so, how uric acid comes to be in excess in the system. It must first be stated that gout is not always evoked by high living. The peculiar habit of body existent in the gouty is not always dependent on nitrogenous excess for uratic accumulation. Without doubt, other conditions operate and lead to this. Gout will assert itself in a gouty individual under very varied dietetic habits. To explain the relation of uric acid to the attacks of gout, we have to conceive of this peccant matter as effective only when in solution in the blood or tissues. Uratic deposit is not, I believe, always the cause of gouty paroxysms. The deposits are often formed quietly, perhaps most often so. They also occur after paroxysmal attacks. Neither may we readily affirm that an actual excess of formation of uric acid is necessary for the production of gout anywhere. It is now proved that uric acid may be formed in normal amount and yet be retained in the body. The excretion in a given time may be less than normal, and at another given time be in excess of the due amount. It is also proved that definite symptoms result from its retention, which pass off with excretion of that which has been temporarily withdrawn from the circulation. Without actual excessive formation in the system, therefore, there may be, from defective excretion, a relative excess of uratic salts in the blood at a given time. With this arises, in certain individuals only, tendency to gouty manifestations. The kidneys are the excretory organs for uric acid, and in very slight degree, if at all, sites of its formation.

Unwonted muscular exercise is sometimes followed by increased excretion of uric acid. It is also not an infrequent cause of attacks of gout. There may be several factors in the production of the latter, such as fatigue, change of habit, injury to or overuse of joints, but it has been suggested that there may be actual increase in production of uric acid in the system as the result of unusual muscular exertion. Dr. Handfield-Jones has related an instance apparently illustrating this in the person of an Alpine climber. So far from exercise warding off gout, it induced both it and lithiasis, both conditions being absent during ordinary home-life.[1] Dr. Handfield-Jones argues in favour of gout being produced by excessive production of uric acid, as well as by retention of it, due to renal inadequacy. He also suggests that the hypersecretion of uric acid after great muscular exertion may be analogous to the paralytic secretion which ensues after section of the nerves of a gland.

1 Med. Press, Oct. 10, 1888, p. 358.

Dr. Haig [1] has contributed valuable facts respecting the retention of uric acid in the body and its irregular excretion, illustrating clearly in his own person that headache and malaise were dependent on retention, definitely and repeatedly induced, and that these symptoms were relieved by means which set free the retained acid. Without doubt, one relationship of uric acid to gouty manifestations appears to consist in the remarkable insolubility, as before noted, of this, the special peccant matter. Another relationship is that respecting the rate of its elimination from the body. Garrod demonstrated that urea is not excreted in any definite relation to the discharge of uric acid either in cases of acute or chronic gout. Dr. Haig has confirmed this observation in his researches on uric acid in relation to forms of headache induced by uratic retention.

This view may then be accepted, that the excreting functions of the kidneys for uric acid and urea are separate and independent of each other.[2] Garrod in his sixth and ninth propositions maintains the view that, among the causes exciting a gouty fit, is a functional failure of eliminating power for uric acid on the part of the kidneys. This has not received, as yet, any proof. As has been remarked by Dr. Haig, if organic renal failure existed, the urea-excretion would probably be equally affected together with that of uric acid; but this is not the case. In the earlier attacks of gout, at all events, the kidneys are presumably healthy, and, indeed, have been occasionally found so in cases where the joints have undergone uratic infiltration. In quoting the views of Dr. Ralfe, it was shown that he doubted Garrod's explanation of this part of the gouty process, maintaining, with many other observers, that diminution of uric acid in the urine was chiefly met with in cases of chronic gout with structurally damaged kidneys.

We have still to find a cause for uratic retention or non-excretion. Physiologists and chemists have not yet said the last word either about the production or the destruction of uric acid in the human economy. As has been pointed out, a larger field for its production than the liver, spleen, and blood-glands has been hypothecated. It has been suggested that under abnormal conditions uric acid may be produced in parts of the body not

[1] Practitioner, 1884, vol. xxxiii., No. 2; St. Barth. Hosp. Reports, vol. xxiii. p. 201, 1887; Med. Chir. Trans., vol. lxx., 1887. His researches go to show that uric acid is not produced in excess in the body, but that irregularities occur in its retention and excretion, thus giving rise to various symptoms.

[2] In support of the excretion of these two substances, uric acid and urea are usually found to be increased or diminished together. The relation in health has been found to be about 1 to 33.

usually concerned in its formation. Thus, Ebstein holds that the muscles, and possibly the medulla of bones, may take part in its production in the case of the gouty, and suggests that the disease may consist in a diathetic error of tissue-metamorphosis present in greater or less degree, perhaps latent in many predisposed to gout, and only capable of being evoked by certain determining causes. "Amongst the anomalies of tissue-change must be reckoned that of gout. Gouty individuals form uric acid in perverse localities in muscles and bones." If this view be accepted in the meantime, we may proceed a step farther, and conceive that with this perverted formation there may be also disordered tissue-metabolism, and the uric acid be insufficiently reduced, and thus thrown in excess into the blood. If Ebstein's view be discarded, it is still open to hold the latter suggestion, that, even with normal production of uric acid, there may be tissue-failure to dispose of it normally, and to reduce it as in health.

This is the view held by Dr. Ralfe, who maintains that, in the presence of a free circulation, uric acid is carried from its seats of production into the blood and gradually reduced to urea, whereas, in tissues outside the current of the circulation, the insoluble acid is not so readily carried off, and, on slight disturbance, is prone to be deposited.

There is much to be said for this conception of Ebstein as to undue formation of uric acid in unusual localities in the case of gouty diathesis. In particular, it affords an explanation of the fact that gout is something more than a functional disorder of the liver, which may lead to lithæmia as one of its results, but goes no further in establishing unequivocal gout. Without doubt, there are peculiarities of tissue in the gouty, and with these may very possibly be associated peculiarities of tissue-function and metabolism. This uric acid formative tendency has been regarded by some, notably by Laycock and Gull, as a reversion to a lower type of animal tissue-metamorphosis, wherein this substance is produced in place of more oxydized products thus rendered soluble and less noxious to the human economy.

Amongst the peculiarities of tissue in those goutily disposed has been observed a feebleness of capillary circulation at the periphery, a condition leading to disorders of chilblain-type, the vessels filling slowly after being emptied. The periphery is also very sensitive to external impressions.

The muscular and osseous systems are often highly developed, and, thus, if Ebstein's view be correct, there are large fields for production of uric acid in many cases.

If the liver and blood-glands be not entirely in fault as producing, primarily, undue quantity of uric acid, the former certainly appears to suffer from irritation by noxious products of early digestion, which may cause perverted metabolism, and thus induce, secondarily, undue formation of this substance. Evidence of this irritation and perversion of function is occasionally shown by the pale stools which occur in early stages of gout, deficient at least in biliary pigment, and sometimes associated with headache or hemicrania.

I think it may be fairly conceded that uric acid is apt to occur in excess, from time to time, absolutely and relatively, in the system of the gouty. It is certain that the disease occurs under the opposite conditions of over-indulgence and strict temperance both in meats and drinks. It is easy to explain excessive production of uric acid under the former condition, much less so to account for the excess under the latter. The only possible explanation is to be sought in the fact that there are specific differences in the tissue-metamorphoses in the two cases.

Hence, it may be assumed that in the gouty there is a failure of full physiological activity in the tissues, a "primordial vice of nutrition," as M. Rendu [1] terms it, leading to imperfect elaboration of the food taken.

Without doubt, the capacity of individuals to deal with certain aliments varies infinitely. These constitute dietetic idiosyncrasies, and they depend on modes of intimate tissue-potentiality. These vary even in members of the same family, and within the limits of health. Mr. Hutchinson has placed gout amongst the food-diatheses, and, as has been already stated, regards the inheritance of it as that of "a peculiarity of tissue."

We perhaps come nearer a complete understanding of this matter if we regard as present in the gouty a peculiar incapacity for normal elaboration within the whole body, not merely in the liver or in one or two organs, of food, whereby uric acid is formed at times in excess, or is incapable of being duly transformed into more soluble and less noxious products. Thus, by excess of ingesta excess of uric acid may be formed, and by failure of tissue-transformation, without excess of uric acid forming ingesta, excess of this acid may be thrown into the blood. With this failure of normal metabolism for uric acid

[1] *Nutrition retardante* of Beneke and Bouchard. *Vide* Art. "*Goutte*" in M. Dechambre's *Dict. Encyclopédique des Sciences Médicales.* This is a masterly exposition of the whole subject, replete with learning, and containing the most complete bibliography relating to gout ever compiled.

commonly co-exists a like incapacity for other complete transformations, so that other imperfect products are apt to be thrown into the circulation along with this special peccant matter.

I am of opinion that this incapacity for normal destruction of uric acid in the tissues depends on disturbed innervation. Dr. Ralfe has expressed himself in a similar sense.

Having now ascribed to perverted neuro-trophic function undue formation of uric acid, there remains to be shown cause for its abnormal retention in the system, which is allowed on all hands to bear intimate relation to the manifestations of gout. Diminished alkalescence of the blood is certainly the result either of undue formation or of abnormal retention. In health it is impossible to render the blood acid. Its alkaline state is believed to be due to the excess of alkaline bases derived from ordinary aliment. As Sir William Roberts has suggested, a meal "is *pro tanto* a dose of alkali, and must necessarily, for a time, add to the alkalescence of the blood." Direct experiments have proved the truth of this,[1] and of the effects of meals in inducing, as a consequence, an alkaline condition of the urine for a subsequent period. This "alkaline tide," as it has been termed, ebbs after a time, and with fasting the urine steadily resumes an acid reaction. As pointed out by Roberts, the reaction of the urine plainly reflects the condition, for the time being, of the blood, one function of the kidneys being to regulate the reaction of the blood. It is, therefore, possible to influence this condition of the blood by giving food, or, in another way, by directly administering alkalies, to produce alkalinity. With respect to acids, it is found impossible to acidulate urine except in the very peculiar way that this may be achieved by means of benzoic acid. All this is true of a healthy individual. In discussing this question in relation to headaches produced by retention of uric acid, Dr. Haig, in his able and suggestive research, inquires pertinently whether in *abnormal conditions* we may not meet with *variations* in the alkalescence of the blood and tissue-fluids, and of the liver and spleen, sufficient to produce fluctuations in the excretion of uric acid. He proved in his own case that animal food led to retention of uric acid, and that vegetable food promoted excretion of it, and he argues: "If an ordinary meal is a dose of alkali, a somewhat vegetarian meal, from which butcher's meat and beer are absent, must surely be a large dose of alkali," and, thus, both promote greater alkalescence of the blood and prevent uric acid retention.

[1] *Op. cit.*, p. 56.

Dr. Haig suggests, further, that the gouty somewhat resemble vegetable feeders in having less than the normal power of forming ammonia to resist acids, and prevent their taking alkali from the blood. By persistence in animal food and strong liquors, he thinks the alkalinity of their blood and fluids is so far overcome that urates will be less soluble in them than in the normal condition. This theory, I think, affords a valuable illustration of the tissue-incapacity already alluded to. Guided by this conception, which is founded on definite observations in several instances, we may fairly believe that in persons of gouty habit excess of nitrogenous diet, or definite additions of acids or acid liquors, such as beer and wines, or fruits, lead to retention of uric acid in the blood, and especially in the liver and spleen, because their tissues are less alkaline than those of many other organs. Dr. Haig suggests "that a dose of acid in these persons increases the acidity of the liver and spleen, and causes increased retention of uric acid in them, while a dose of alkali will diminish their acidity, and sweep out the uric acid accumulated there," thus giving rise to various symptoms.

Garrod's view as to retention was based on an assumed renal incapacity; but it is certainly not proved that such incapacity exists in the earlier stages of gout. It might, perhaps, be assumed as part of the tissue-peculiarity of the gouty. In considering the phenomena of a paroxysmal attack, the influence of the nervous system following shock, depression, or any violent interference, must not be left out of account as a possible determining factor for renal inadequacy. This is well ascertained in hysteria. Anuria, from suppression in such cases, has been described by Laycock and by Charcot. Laycock believed that a measure of renal inadequacy occurred in such cases of gout as were induced in early life by excessive venery and alcoholic stimulation, owing to reflected exhaustion of the nervous system of the genitalia, these organs being closely related embryologically. By the light of Dr. Haig's researches, we are justified in believing that temporary retention in the system, probably in the liver, spleen, and other glands, leads to such fluctuations in the alkalinity of the blood, and, coincidently, in the excretion of uric acid by the kidneys, as may fairly explain some of the humoro-pathogenetic relations of gout, and apparent arrest of renal function.

His facts are, that there is diminished excretion of uric acid before, and increased excretion of it after, the nervous disturbances induced by it.

With the induction of a well-established gouty habit, we must suppose a more permanent change in the natural alkalescence of the blood due to retained uric acid, and with this a tendency, under certain provocations, to deposition in different parts of the body.

The conditions leading to excretion are related to the alkaline tide of early digestion, and those concerned in retention are associated with later digestion, and the acid tide of this period and of sleep, as indicated by Dr. Haig.

According to Lecorché, the gouty state is due to the transformation of uric acid into acid biurate. This is brought about by such causes as generally lessen the alkalinity of the blood. He denies that gout is a malady induced by retarded nutrition, but is characterized rather by hypernutrition with exaggeration of molecular work.

The essential humoral feature of the gouty state is the presence in abnormal amount of uric acid salt in the blood and tissues. So long as this condition prevails, the patient is gouty, and unless the excessive amount be reduced by preventive and medicinal measures, this state is apt to be maintained or to recur.

It now remains to correlate the two pathogenetic factors concerned in the production of a paroxysm or overt attack of gout, namely, the nervous and humoral parts of this process.

It has already been shown that the nervous system of the gouty is peculiarly sensitive, and predisposed to instability in certain directions. This condition, together with the peculiar tissue-state of the gouty, itself, as I believe, dominated by trophic nerve-influences, affords, probably, a fairly complete conception of the malady in question. As already affirmed, neither condition by itself suffices to explain the recognized characters of gouty disease. Thus, the peculiar state of the blood has been shown to be insufficient by itself to set up the phenomena of gout, and the condition of the nervous system may be latent in respect of revealing (painful) symptoms of overt gout. We must then regard the nervous factor or element as dominating specifically the several definite features of gouty manifestations. We may assume that the chain of morbid events has its origin in irritation of the nervous system, or of some part or parts of it, by the peccant matter thrown into the circulating fluids of the body, and that, thus, misemployment of nerve-force occurs, determining definite paroxysms or local manifestations in one or more parts of the frame. At this point I must refer to the view expressed

in an earlier portion of this chapter respecting gout regarded as a diathetic neurosis, due to a central neurotic taint, and originating from prolonged toxæmia. This I termed primary or central gout. This view is conceivable as the result of inherited gouty propensity, but can perhaps hardly be admitted as all-explaining without acceptance of the further view as to a more widely spread complicity of other tissues in the manner already set forth. The morbid disposition may well exist in more marked degree in some instances in one than in the other direction, and in the most pronounced conditions we may regard both the nervous system and the tissues generally as involved for the production of gout.

In another class of cases, where the disease is, as it were, quiet, latent, or not paroxysmal, we may fairly suppose that some nervous manifestations usually present are from some cause in abeyance. For example, the most exquisite gout in respect of tophaceous deformity may grow up in various parts, constituting, as Todd believed,[1] a special clinical variety, without a single twinge of pain or symptoms of nervous disturbance. The nervous element in such a case may have been only so far abnormally evoked as to determine the special locality affected in this manner. We cannot doubt the influence of nervous force in any form of nutrition, whether normal or morbid, and we can conceive perturbations of it as exciting, if not determining, perverse trophic changes.

A complete conception of gout necessarily entails a consideration of the inflammatory processes associated with certain of its manifestations. We need not invoke any special factors in this connection. Gouty inflammation resembles most other forms of this disturbance, with perhaps one exception, viz., the well-known absence of tendency to suppuration. There are specific peculiarities, moreover, relating to suddenness of onset, intensity and fugacity of pain, and conditions attending such a process in parts which are non-yielding and non-vascular.

In respect of gouty as of other inflammations, we must admit the influence of perverted nerve-force in inducing alteration both of quality and quantity in nutritive fluids. Herein lies part of the specificity of the gouty process. There is, as I have already maintained, a special mode of nervous evolution dominating many of the phenomena of gout, and this marks off the peculiar diathesis as certainly and significantly as does the altered blood-state. We find, therefore, a twofold inheritance in respect of nerve-evolution

[1] Clin. Lectures, Urinary Diseases, p. 425, 1860.

and toxæmia, or tendency thereto, not always inherited in the same degree, or even equally in respect of the two states.

Where no inheritance is traceable, we may conceive that, as a result of the primary induction of toxæmia by over-indulgence, changes are set up in the nervous system leading to the specially perverted manifestations of nerve-force which constitute that part of the gouty pathogeny. And in our conception of the part played by the latter, we must not lose sight of the larger and more wide-spread influence of nerve-force upon intimate tissue-metabolism, which may be highly effective, though at present undemonstrable and almost inscrutable.

We must next seek to discover some of the special perverted modes of action determining overt gout.

It is an axiom in pathology that a change in any part may, by altering its relation to the blood, alter its mode of nutrition. In this manner injuries to parts lead to altered nutrition, and render them more vulnerable and open to inflammatory or other changes. In healthy persons such changes possess no specific characters, but in persons the subject of any diathesis, these changes will certainly be modified in some definite manner according to the specificity of the habit of body.[1] Thus come out the characters of specific disease, and to induce these there must be, as has been pointed out by Paget, at least two factors—the morbid matter in the blood, and the presence of a part of the organism specially adapted for this matter to effect its malign purpose.

To apply these views to the case of localized gouty manifestations is not a hard task. It is well known that parts which have been injured, strained, or overworked are just the sites in which gout is apt to appear. All such influences are calculated to impair and depress the nutrition of the textures involved. They are, therefore, more than other parts vulnerable, sensitive, and laid open to attack. The blood is the medium wherein the peccant matter of the disease lies, and a special elective affinity is, as it were, established between the weak part and the distempered blood.

We, thus, understand how uratic deposit is determined at certain points whose nutritional standard is lowered or altered for the worse. Amongst parts specially exposed to strain and hurts,

1 "Le traumatisme éveille souvent la prédisposition diathésique."—*M. Rendu.*

"An injured part may become the seat of gouty disease in one gouty. . . . Thus, in diseases recognized as specific, in those that certainly have a specific morbid material in the blood, we recognize a local injury or irritation as making a part susceptible or apt for the manifestation of the specific morbid changes."—*Paget, Morton Lecture,* 1887.

none suffer more than joints, and, hence, probably the explanation of the inordinate incidence of gout upon these structures, especially witnessed in the joints of the great-toe, the knee, and the hands. I believe that this view holds good both for imperfectly developed and for paroxysmal forms of gout. Other textures than joints are sites of gouty election; thus, we meet with uratic deposits in other tissues, but with especial frequence in such as have inactive circulation, such as sheaths of tendons, and the skin over various regions.

Many of the lesser, though painful, manifestations of gout are due, I believe, to temporary stasis, if not to deposition of uratic salts, even in viscera with active circulation, such as the liver, or in synovial sacs, nerve-sheaths, and lymph-spaces. No unequivocal proof of this is forthcoming, but its likelihood is established by the fact of their occurrence in gouty persons, and by the happy results of anti-gouty treatment which removes them. In the kidney the presence of urates in the adult may safely be taken as an indication of gout, and when found here, they will seldom be found absent from the smaller joints. Even in cases of this kind there may be no history of paroxysmal gout. The phenomena of an acute attack bear some resemblance to those seen in a specific fever. Indeed, the older physicians placed gout, nosologically, amongst the fevers.[1] The sudden paroxysm, the local signs, the crisis, and the subsequently amended health, are all comparable with the series of events witnessed in an exanthematous fever, and are fairly analogous as indicating the certain effects of distempered blood-state. The characters of gouty pyrexia will be given subsequently. Those relating to the paroxysm and its frequent suddenness now demand attention while discussing the pathogenic relations of the disorder. It has always been difficult to account for the explosive features of gout. These vary much in different individuals, and even in the same patient. A bad attack will establish itself within a few minutes, and an equally bad one will sometimes take many hours to reach the same degree of severity. Fagge regarded a paroxysmal attack in the light of an accident occurring in the course of an essentially chronic change in the joint affected. It may be believed that the conditions leading up to the attack have been some time previously in operation, the blood becoming more impregnated with urates. Some determining factor must now be invoked to explain how, as it were,

[1] The great Boerhaave believed that gout was contagious (Aph. 1255). *Vide* Translation of van Swieten's Commentaries upon Boerhaave's Aphorisms, vol. xiii. p. 27, 1775.

the train is fixed. The fact that paroxysms have been induced by a heavy meal, or by a single indulgence in certain liquors, is probably in part explained by the sudden addition to the system of more materials than can be dealt with by the organs and tissues engaged in metabolic functions, and these are probably, as has been shown in many cases, at the best, specifically impaired or inadequate in the gouty. The same result may also be caused by the effect of cold, throwing increased work on internal organs, which by checking perspiration diminishes the alkalescence of the blood, and so leads to precipitation of urates.

If the kidneys are healthy, it is not easy to suppose that, as has already been stated, any special functional inactivity exists leading to defective excretion. These organs may, however, be specially prone in the gouty to be nervously impressed. It may, however, be here noted that some liquors, especially wines, vary much in their effects on the kidneys. Those which promote diuresis are usually the least gout-provoking, and *vice versâ*—a point to be discussed in future under the head of dietetics.

I believe that, in order to explain the explosive characters of a gouty paroxysm, we must look to the nervous elements of the case. Not only are excessive diet and over-indulgence in liquors to blame as exciting causes. Were these factors alone potent, how then shall we explain the well-ascertained fact that the same phenomena supervene on mental causes, such as shock, or after fatigue and exhaustion, which can, and must, act alone through the agency of the nervous system? I have already endeavoured to show that this system is peculiarly disposed and impressible in the gouty, evincing instability and undue sensitiveness, and, hence, I am led to believe that to its influence is to be ascribed much that dominates the manifestations of the paroxysm. Hence, we may conceive the paroxysm as resulting from an interruption of what I would term the trophic equilibrium of the body. Thus, the tendency to nocturnal seizure, the extraordinary pain, and the other features already alluded to in the section on neuro-pathogeny. Hence, it is not the quiet deposition which determines a paroxysm in a part, but the presence of excess of uric acid in solution in the tissues which is thus, together with specially determined nervous influence, potent.

I have already discussed the line of action which is assumed as that of nervous influence upon joints, viz., the proposition which regards articular affections as due to irritative states of the spinal axis and sympathetic system, and have ventured to express my belief that this is essentially necessary for a complete conception

of the arthritic habit of body as evinced in both gouty and rheumatic diseases. The occurrence of one-sided manifestations of arthritism, *e.g.*, joint-affections, hemicrania, and neuralgia, which have been well-established in certain cases, indicate still further a dominating nervous influence. M. Henry Cazalis, of Aix-les-Bains, has directed attention to these cases, and he notes that these unilateral manifestations are most frequently right-sided.[1]

I have notes of several cases in which articular paroxysms occurred with greater frequency and intensity on one or other side of the body, no determining cause being made out in any case. I lay no stress on the facts. Acute attacks had occurred on both sides in many of the cases. There is, doubtless, some cause for the limbs on one side suffering more than those on the other, but there is at present no known explanation of the fact.

A mere physical theory of gout, such as is now much held in Germany, hypothecates local stasis of uric acid in certain textures and situations, and allows that in the more vascular parts this excess can be carried away by the vigour of the circulation, and taken into the blood in solution. In less vascular areas or non-vascular tissues, such as cartilages, and those of the latter, in particular, most peripherally situated, the force of the blood-current is too feeble to carry off such deposits, and, hence, persistent stasis with a gouty paroxysm.

On this theory, local influences have much to do with the determination of attacks, and it is not even necessary to believe that there is any absolute excess of uric acid in the system on the occurrence of a paroxysm.

Although very ingenious, I am not prepared to accept this purely physical view of the pathogeny of gout as sufficiently explanatory of all the phenomena. Neither is a purely chemical theory adequate for this purpose. It is incumbent, I believe, to invoke not only a chemical and a physical basis for gouty disease, but to include also, in a comprehensive view, the marked determining influence of the nervous factor in the problem.

It appears to be proved by Garrod that the inflammation in a gouty attack tends to the destruction of the urates in the blood-inflamed part. It is, however, difficult to believe that, as Garrod further maintains, this local change is sufficient to clear the system from the uratic excess present at such a time. The quantity

[1] *Note nouvelle sur l'Hémi-Rhumatisme, Jour. de Méd.*, Paris, 1 Mai 1887. *La Prédominance Hémi-laterale des Manifestations du Rhumatisme Chronique, Communication à l'Acad. de Médecine.*

deposited locally with each attack is probably insufficient to explain so much elimination as may be presumed to occur, for, along with destruction in the tissues due to inflammation, fresh deposit is laid down. After each attack the system is certainly relieved, and better health is established. Hence, it may fairly be assumed that the uratic excess in the blood is dissipated and disposed of. An argument in favour of such eliminant or destructive action being due to the inflammatory attack, or conditions attendant on this, may, however, be adduced from the fact that some of the most bulky deposits occur in parts which have never been the seat of acute paroxysms, or only of slight inflammatory attacks.

This point did not escape the acumen of Sydenham. He remarked, "In gout, however, it seems as if it were the prerogative of Nature to exterminate the peccant matter after her own fashion, to deposit it in the joints, and *afterwards to void it by insensible perspiration.*" He alludes to the relief afforded by morning-sweats after the pain and restlessness of a night of gouty suffering. Todd observed that sweating relieved the pain of gout.[1] Doubt has been cast on the eliminant power of the sweat-glands for the removal of uratic salts from the body. Garrod, in particular, denies this, having failed to find any uric acid in sweat procured from gouty patients after a Turkish bath. Dr. Tichborne, of Dublin, however, has succeeded in detecting it under similar circumstances, and maintains that the colloidal character of uric acid permits it to dialyse through animal membranes, this property being augmented by a temperature such as that of the body. I have, however, failed to find it in two cases examined by Tichborne's method.

I have, nevertheless, little doubt that the skin is a channel for removal of some of the excess of uratic acidity met with in gout.

It may be that the articular sites of predilection are determined by nervous (neuro-trophic) influences. Of this we are not yet sure. I think it certain that deposits may occur in joints long before any classical attack of gout supervenes in them, and such may, indeed, never be developed.[2] It is also certain that, in most instances, an attack of gouty inflammation leaves behind it uratic deposit. But gout can induce many other and grosser changes in

[1] Clin. Lectures on Urinary Diseases, p. 413. Lond., 1857. The proportion of uric acid in the urine has been shown to be diminished by diaphoresis. *Wilson Philip.*

[2] This was noted by Scudamore, *op. cit.*, p. 145.

parts beyond mere uratic deposition. These will be referred to under the head of morbid anatomy.

Uratic deposit is certainly found most abundantly in parts that are least vascular and peripherally placed in respect of the circulation. We must now discuss the relations of this deposition to the whole disease, whether in the latent or the paroxysmal form. It has been supposed that by this means the excess of uratic salt in the blood—a recognized factor in the case—is so far eliminated, or shut off, from the system. Garrod has demonstrated that gouty inflammation is always accompanied by deposit of urates in the affected part, and that this deposit is permanent.[1] It is also proved that this deposition may proceed without any inflammatory symptoms, as commonly recognized. Garrod believes that the deposition is the cause of the inflammation.

If this proposition be put forward without further qualification, it cannot be sustained, for the reason just mentioned. I believe that it holds good in many cases, and is, indeed, the only explanation forthcoming, at present, for explosive attacks. Even in cases where latent deposit has already taken place, I believe the supervention of a more pronounced gouty state will lead up to paroxysmal attacks in a part already the seat of quiet gouty change. The condition may therefore be very largely but a quantitative one. Hence, I cannot quite accept Dr. Ord's view that "the local processes are not dependent on these deposits." I would say, "are not *always* dependent," for I conceive that, with a large excess of urates in the blood, local inflammatory changes may be set up. A certain amount of deposition is tolerated, a larger amount is resented, and excites violent reaction. The conditions determining these phenomena probably relate to personal peculiarities, the degree of inheritance and range of the disorder, and they have to do with the age, tissue-state, and general vigour, or the reverse, of the individual. These personal factors, indeed, can never be lost sight of in any case.

When a gouty habit of body is established, the causes already enumerated tend to operate more readily and with less provocation. With failure of nervous power comes less reaction, and a more tedious and atonic process both of development and duration. Tissue-degenerations make progress, set up both by failing neuro-trophic influence, and by direct action of retained peccant matters. The kidneys, in particular, now become struc-

[1] From some examinations made after death, in cases where the history clearly pointed to gout in certain joints, I am disposed to question the unvarying certainty of permanent deposit.

turally involved, and a permanent inadequacy of their functions supervenes. This condition constitutes what is termed the gouty cachexia. But not in all cases does the gouty diathesis progress towards its corresponding cachexia. The disposition exists in very varying degrees of intensity. There may be only slight indications of its presence, or but a few overt paroxysmal attacks in a lifetime, and the habit of body may but modify any superadded morbid conditions, and not itself lead directly to death. Inherited gout is the most obstinate, because most fully developed. The two essential factors concerned in the production of the disorder may be inherited, probably, in varying proportion.

The tissue-defect, or uric acid-forming tendency, may be more pronounced than the neurotic element, and *vice versâ*. Circumstances of life, propensities, and habits may readily evoke, repress, or accentuate each of these, and, thus, determine the range of action of each in any given case.

In this manner we are enabled to explain many of the varied features of the disease which present themselves. Thus, a man may be gouty without having what is commonly called gout. There may be gouty disease of the kidneys without uratic arthritis, although in many, but not in all, of such cases, quiet deposits of urates may be found in certain joints. The paroxysmal (neurotic) element is in abeyance in such cases. Clinically, such cases are recognized as gouty by various features in their history and progress. The diagnosis is not rendered absolutely certain till the particular form of kidney-change and the deposits are manifested at the necropsy.

In persons under forty years of age, we are often justified in prognosticating at some future time the onset of regular gout by the special morbid tendencies exhibited, the irregular or incomplete gouty nature of the symptoms indicating what is in course of fuller development, unless effectually checked by a change of habits and by direct treatment. This is, happily, not an impossible achievement in preventive medicine, and, hence, the value of a due recognition of these manifestations and untoward tendencies in early life, an accurate knowledge of family history being amongst the most important facts to be sought.

I come, lastly, to consider the question as to the specific importance of uratic deposits as alone significant in any case of gout or goutiness. It may appear to savour of heterodoxy to entertain any doubt on this point.

The real issue is not as to the existence of urichæmia, which must be absolutely accepted in any given case, but as to the presence of deposited urates in some locality. It can hardly, I think, be doubted that lesions result from the action of uric acid in solution in the tissues, and that thus both acute and chronic inflammatory changes may be set up without the direct influence of uratic deposit as an alleged irritant in joints and in certain viscera, notably in the kidneys. Degenerative changes and necrosis also appear to be thus induced.

I think we here witness, as Ebstein puts it, the results of local uric acid stasis in the one case, and of general stasis in the other.

A study of the morbid anatomy of gout appears to justify the views held by Drs. Ord, Norman Moore, and others, that deposits never occur but in tissues which have already begun to degenerate.

I must express my belief, after much observation and long reflection on the whole matter, that the presence of uratic deposit is not absolutely indispensable for the determination of gouty disease or manifestation. I believe that all the essential elements of the morbid process may be present in cases without this particular expression of it in the form of what is termed "frank" gout. The facts on which I base my conviction may possibly not avail to carry proof to many. First, I would affirm that within the domain of pathology we meet with morbid states in very varying degrees of intensity. We have more or less, and we have always, the personal factor in each case, including the degree of inheritance, its modification, and the measure of vulnerable reaction in the textures specifically invaded or impressed. Secondly, I note that with very trifling degree of uratic deposit, a great deal of other recognizable gouty disease may be present in a given subject, as, for example, granular kidney (nephritis arthritica so-called), cardio-vascular change, and the like, leading to fatal result. Thirdly, I think it permissible to claim as gouty a case in which, without articular deposit, always supposing this to have been widely sought, interstitial nephritis and other lesions recognized commonly as gouty are present, and in which, during life, some of the irregular latent or incomplete manifestations of gout have been observed. If, in addition to this, there be found history of gouty ancestors or of gouty family, I maintain that it is not unwarrantable from this and the clinical features of the case to declare for gouty disease in such an individual.

Hence, without falling back on uncertainty, or making less

sharp the line of demarcation between gout and other joint-affections, I think too much stress has been laid on the fact of uratic deposit as the absolute touch-stone in the exact diagnosis. My belief is that there is much gouty disease—incomplete gout—as well as much overt gout, and I also incline to think that this is now thrown open to recognition by better differentiation of cases and improved diagnosis. It may also be the case that, at the present time, there are more frequently to be found some of these modifications of that more classical and overt disease which was described by most of our predecessors in Medicine who wrote about gout.

I may state that these views are held in the French school by Charcot and Lancéreaux, and they find support from Virchow and Ebstein in Germany. I lay stress on cases of chronic and incomplete gout, in elderly women more especially, where, with many truly gouty manifestations, articular changes take place leading to deformity, nodes, deflections and synostosis of phalanges. In such instances the kidneys may be found granular, and no uratic deposits be detected. The synostosis alone would afford to my mind the key-note of true gouty process. The clinical features of the case and the granular kidneys afford strong additional evidence. The absence of uratic incrustation may be explained by the incomplete development of the dyscrasia, and by insufficient production of urates to allow of deposit. Many observers would be content to call these changes "rheumatic" or "rheumatoid," but I must deny this element as the predominating factor, for the reasons just given. Morbid anatomy by itself is often apt to mislead, unless supported by previous and careful clinical study.

The latest researches into the nature of gouty disease as a whole plainly point to the very wide-spread character of the disorder. In its articular, abarticular, and visceral varieties may be found ample proof of this. There would appear to be no immunity from gouty processes in any tissue of the body.

A study of the disease as met with only in hospital practice is insufficient to furnish a complete experience of many of the most varied, if peculiar, characters of the disorder. These are, however, to be found abundantly amongst the upper classes of society, and private practice alone supplies the fullest materials for their observation. Many of the descriptions of the text-books are inadequate to portray the multiform features of gouty disease, and it may be that this arises in some measure from a necessarily

incomplete study of its manifestations, as observed mainly in hospital practice, and consequently for short periods of time.

Note.—It must be acknowledged that such light as modern knowledge enables us to throw on the general pathogeny of gout does not place us very far in advance of that held and taught by many writers of the last two centuries. We now, as formerly, invoke both humoral and nervous causation. The same ideas were really expressed, in the thoughts and language of the time, by Sydenham, the author of one of the most concise and classical treatises on the whole subject of gout, and himself a martyr for many years to the malady. He writes: "The more closely I have thought upon gout, the more I have referred it to *indigestion, or to the impaired concoction of matters both in the parts and juices of the body.*" This is an expression of humoral doctrine. Intemperance in food and drink is shown to impair digestion and lead to oppression of the system by a mass of humours. Next, for the nervous part of the pathogeny: "At one and the same time *the energy of the spirits*, which are the instruments of digestion, is diminished."[1] "The viscera are overworked, and then the spirits, which have been long giving way, are prostrated. If it were not so, if it were a simple weakness of the spirits, children and women and the victims of long illnesses could be equally gouty. On the contrary, however, it is the hearty and robust. These it attacks only during the decline of their best and natural spirits. When this takes place, a congestion of the humours supervenes. *From the two together the due concoctions are vitiated and prevented.*" For "*energy of the spirits*" we are to understand, in the thought and language of to-day, *nervous energy*. Sydenham here foreshadowed what we should now term the failure of tissue-metabolism, induced by, or associated with, impaired neuro-trophic energy, and took account for the production of gout of the two pathogenic factors of "peccant matter" and misdirected or perverted nerve-force. (The italics are mine.)

Again, in respect of the paroxysmal attack, he describes the vicious humours as increasing in bulk and virulence till "Nature" can no longer regulate them, and they break out, fall upon the joints, &c. We may venture to interpret "Nature" here to mean the neuro-trophic equilibrium of the system, which is thus upset.

Sydenham's conceptions appear in the light of to-day to be vastly nearer the truth than those of Cullen, who died a century after him. Stahl, from whom the latter drew his too purely nervous theories, was just coming into note at the time of Sydenham's death, but had not yet published, or perhaps formulated, his views. With Trousseau I would say, "Take it all in all, the theory of the great English physician is much more medical than the theories of modern chemists." I therefore claim the authority of Sydenham in support of a neuro-humoral pathogeny for gout.

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[1] R. G. Latham's translation of Greenhill's edition of Sydenham's Works. Syd. Soc. London, 1850.

CHAPTER IV.

THE MORBID ANATOMY OF GOUT.

"Though the terms arthritis and podagra would seem to limit the malady to the feet and the joints, we have seen it in almost every part of the human system."—Sir Henry Halford, Bart.[1]

I.—Articular.

In treating of the morbid anatomy of a particular disease, the physician may be pardoned if he refuses to accept as all-explanatory of antecedent clinical symptoms the teachings it may offer. While acknowledging to the full the imperative necessity of daily dead-house study for the practical physician, it must not be forgotten that autopsies often fail to reveal the whole truth in respect of the symptoms and features of disease as met with during life. Slight reflection forces us to concede this. As physicians, we have to deal with disease in the living, and not in the dead body, and thus have to contend with, and seek to modify, a whole realm of forces that cease with life. Our work relates therefore to *living* morbid anatomy, and not to the textures and fluids of the corpse. We have to seek knowledge of these perversions and distempers during life, and to try to gauge their degree, while, perchance, something may be done to influence them for good, and so promote relief or recovery.

The physician should tell of the physiological disturbances which are induced by disease, while the morbid anatomist should complement his story by demonstrating the textural ravages, coarse and fine, wrought by it.

The latter may, and sometimes does, discover changes in parts, which, although apparently exactly similar, have been brought about by very different morbid processes. Of these he can give no certain account, because the clinical facts are wanting. The physician supplies these, and, thus, only is the whole truth of the

[1] On the Treatment of Gout, Essays and Orations, 1831.

particular case likely to be learned. For anything approaching to the truth the labours of both are required. In the case of certain organs which are affected with gouty disorders, as, for example, the eye and ear, it is manifestly difficult to secure facts relating to morbid anatomy. There is much, too, in gout which is clinical, and not always susceptible of *post-mortem* proof.

In discussing the morbid anatomy of gout, it is incumbent to point out that, at least, two divisions of this large subject may be made. We have to deal in practice with gout that is primarily or mainly articular, and with gout or gouty disorders that are more general, and certainly less localized, in the joints. The former is the more common of the two. Cases exemplifying both forms are not infrequent. With some exceptions, it may be affirmed that primary articular gout is the less grave form of the malady, and admits of more satisfactory prognosis as regards longevity.

The morbid anatomy of gout relates to almost every tissue of the body. In cases of lesser degree, it may be affirmed that the changes are mainly, if not entirely, imposed on the articular textures. In cases of profound extent, where, in fact, the body is impregnated with gout ("*totum corpus est podagra*" of Sydenham), it is hard to find an organ or texture which is not variously impressed and changed in respect of its intimate structure as the result of the disease.

The touch-stone of gout being, according to most authorities, uratic deposition, it has been well-established that if in any part we meet with this, we are in the presence of unequivocal gout. The question arises, further, whether this is the sole manifestation in any given part. Now, deposits of urates may be regarded, for all practical purposes, as limited to but a few of the tissues of the body. The joints and the parts around and in connection with them are the chief sites of deposition. The evidence from morbid anatomy is sufficient to justify the belief that deposition is favoured by absence of vascular activity, and by the consistency and peculiar nutritional properties of the texture involved. Wherever the circulation is active and warmth constant, deposition is either impossible or greatly resisted With failing activity of circulation, and consequent degenerative changes, almost any tissue may become the site of deposition. Hence, in gouty cachexia, deposits increase and become more and more widely spread; and hence, too, an originally good constitution, and the vigorous circulation which accompanies it, both render the vessels less prone to decay, and the tissues better able to resist gouty degenerative and deposi-

tive changes. So much for the personal tissue-potentiality, which must be regarded in each case as it comes before us, and which goes far to explain many of the perplexing features of gout—as, for example, why this one has, and this one has not, deposit; and why, again, another is a prey to prodigious deposition. We are, perhaps, too apt to regard individuals as possessing identical proclivities, and as equally prone to manifest the same symptoms and reactions under similar provocations, whereas the degree of vulnerability is, in truth, most varied, and is affected by numerous inhibitory influences derived from mixed diatheses, blended strains, and tissue-peculiarities. These views apply not only to gouty, but to any morbid tendency, inherited or acquired, and I regard them as essential to be borne in mind, and very helpful in daily practice.

It has been taught that rheumatism attacks the larger joints, and gout the smaller. It would not be safe to dogmatize thus. With the exception of the hip-joint, which is rarely affected, it may be affirmed that gout commonly attacks both large and small joints.

The order of invasion of tissues by uratic deposits is fairly constant. Thus, diarthrodial cartilages are the first to be affected, then the ligaments, tendons, and bursæ. Next, the connective tissue and skin become impregnated. The order of invasion of joints is also often constant, beginning with the great-toe, metacarpo- and metatarso-phalangeal joints, the tarsus and carpus, and, lastly, the larger joints with no constancy.

The appearance of articular cartilage in which simple deposit of urates has taken place exactly resembles that which would result from smearing or splashing the surface with fresh white paint. If no secondary irritative changes have occurred, the surface is quite smooth, and the synovial fluid is natural in amount and consistence. This white, plastered, surface is sometimes singularly even and uniform, covering exactly the limits of all the cartilages forming the interior of a joint. This may exist without any signs of erosion or ulceration, and without any irritative overgrowth in its vicinity. There are examples of this in the Museum of St. Bartholomew's Hospital. Many joints in a body may be thus encrusted. Such specimens retain their appearance unchanged for many years. We have two in the Museum which Mr. Stanley placed there, and after seventy years' immersion in pure spirit [1] they are now quite exemplary. These

[1] Methylated spirit was not used till within the last twenty-five years.

It is important to note that prolonged immersion in *methylated* spirit is apt to cause complete removal of uratic deposit from cartilage. This is apparently due to

specimens were originally described as encrusted with carbonate of lime, and Scudamore in his book so described them in 1813. In 1884 I had them dismounted and re-examined, both chemically and microscopically, and on analysis they were found to present all the characters of gout with sodium urate deposit, calcium salts being present only in appreciable amount. Dr. Fuller referred to these specimens in his book on "Rheumatism, Rheumatic Gout, and Sciatica" (1852), and considered them as illustrative of a hybrid case of rheumatism and gout. Few would now be found to believe that these were other than examples of true gouty (uratic) arthritis.[1]

Instances such as these indicate that abundant uratic deposits may occur in joints without exciting irritating overgrowth. Why this should happen in some cases and not in others, it is hard to explain. Some personal factor or tissue-peculiarity may account for it. It has already been stated that deposits occur, sometimes to enormous extent, without exciting even pain, although the deformities thus induced are prodigious. This is termed quiet deposit.[2] It is certain that the bulkiest deposits occur in the upper rather than the lower extremities.

The changes in articular cartilages which result from gout have not been studied so carefully as they deserve. As a matter of fact, the joints have only of late years been inspected in necropsies of gouty subjects, and attention has been mainly directed to the question of uratic deposits.

Study of museum specimens shows that profound changes may occur in all the structures of gouty joints. So much so is this the case, that some observers have come to regard these as indicating the coexistence, or even the coalescence, of rheumatic

some special solvent—possibly an acid—in certain kinds of methylated spirit. Pure spirit of wine has no such action. Thus, within two years a large series of specimens, illustrating uratic arthritis, put up by Dr. Norman Moore, became practically valueless as indicating the truly gouty nature of the cases they came from, whereas specimens in our Museum put up many years ago in pure spirit are as valuable now as when fresh. Museum specimens may thus become a source of perplexity and error to the students of the future. Mr. Stanley's specimen, already referred to, was put up in pure spirit, which was always used in museums at that time.

Mr. Shattock tells me that this untoward effect on encrusted parts may be prevented by dissolving urate of sodium in methylated spirit. Tophi may be macerated in the preserving spirit, and after filtration this tincture or solution may be employed for encrusted specimens in museum collections, and prove trustworthy as a preservative of the characteristic features.

[1] *Vide* Notes respecting two Old Specimens of Gouty Arthritis in the Hospital Museum. St. Barth. Hosp. Reports, vol. xx., 1884.

[2] Drs. Moxon and Pye-Smith have noted the occurrence of slow deposition of urates in joints without symptoms. Fagge's Prin. and Pract. of Physic, vol. ii. p. 801.

changes. Hence, Mr. Hutchinson regards it as rare to meet with joints presenting only the characteristic deposit, and believes that all the associated tissue-changes are *rheumatic*, and thus he argues for a true "rheumatic-gout" as the outcome of all gross changes in such cases.

I cannot agree with him on this point. It is certain that very few specimens exist showing well-marked changes of chronic rheumatic or osteo-arthritis together with uratic deposits. Such specimens must be accepted as illustrating a true coincidence or a coalescence of two morbid conditions, and I see no reason for doubting that chronic rheumatic arthritis and true gout may co-exist and blend in the same individual. It would be strange if this did not sometimes happen; but as a matter of observation, it is rare to find such a conjunction. Mr. Hutchinson's views are so dogmatically set down on this point, and his opinions very justly carry so much weight, that I am concerned to combat them as forcibly as I can. He holds, with Charcot, that rheumatism and gout are the outcome of a basic arthritic diathesis, and thinks it a matter of habit, of dietetics, or of exposure to damp and cold, as to whether one or other, or both, of these troubles shall develop in any individual who inherits this diathesis. "Gout," he thinks, "is but rarely of pure breed, and often a complication of rheumatism. It so often mixes itself up with rheumatism, and the two in hereditary transmission become so intimately united, that it is a matter of considerable difficulty to ascertain how far rheumatism pure can go. . . . When this complication exists, it shows its power, we may suspect, by inducing a permanent modification of tissue, and it is to this modification that the peculiarities in the processes (transitory rheumatic pains in joints, fasciæ, and muscles, chronic crippling arthritis, destructive arthritis with eburnation, lumbago, sciatica,) are due. Hence, the impossibility, under many conditions, of discriminating between gout and rheumatism."[1]

The evidence that is to settle this vexed question is as yet not all forthcoming. The sources of this evidence are obviously threefold: first, the antecedent history; secondly, the clinical symptoms; and thirdly, the morbid anatomy. Now, the difficulties in the way of getting a trustworthy antecedent history are enormous, and this holds good almost equally in every rank of life. It has been my habit to seek for this evidence untiringly in all my cases, and the results are for the most part unsatisfactory. Still, with care, it is possible in many instances to unravel the family and

[1] Pedigree of Disease, 1883, p. 126.

personal histories so as to allow the fact of goutiness to be either admitted or rejected. Failing the history, recourse must be had to study of the existing clinical symptoms and the effects of treatment. The latter, in particular, seldom fail to throw light on the nature of any given case. The ultimate appeal is to morbid anatomy. Hitherto, in the case of gout, the touch-stone of the process has been the presence of uratic deposit. As already stated, Mr. Hutchinson regards it as rare to meet with simple deposit in joints apart from other changes. My experience is quite otherwise, so that I regard this as a common occurrence, without any associated structural changes. The gross lesions referred to by Mr. Hutchinson as due to rheumatic influence, I believe to be the result of gouty arthritis, and I should require strong evidence to convince myself to the contrary. In a case presenting other truly gouty characteristics, I am not prepared to insist on the necessity of discovering deposited urates in every joint, since I believe that, as a result of urichæmia, many and varied textural lesions may occur without such deposit, and I am not alone in this opinion. The variety of cartilage which is chiefly involved in gouty processes is that known as articular or hyaline cartilage. This has a transparent ground-glass-like basis. The cells are spherical or oval, with a single nucleus, and are usually arranged in pairs contained in a single capsule. In the neighbourhood of the bone the capsules contain more than two cells.

With respect to the mechanism of cartilage-nutrition, reference may be made to Dr. Albert Carter's observations on the diaplasmatic system of vessels which he believes to exist in non-vascular parts. He found in articular cartilage certain reticulations (plasma channels) passing from the margins of the lacunæ, which were filled here and there with granules.[1]

These have not been noted by other observers. From conversations with distinguished anatomists and histologists, I gather that there is at the present time no certainly demonstrated mechanism for the intimate textural nutrition of cartilage.

Where articular cartilage adjoins the synovial membrane and capsule of a joint, the cells are branched, and pass insensibly into the connective tissue-cells of the synovial membrane.

In studying the morbid articular changes due to gout, it has been necessary first to examine the diarthrodial cartilages in persons at various ages and in those of the healthy. Dr. Wynne and

[1] Professor Turner has kindly referred me to this paper. Jour. Anat. and Phys., vol. iv. p. 97, 1870.

I have found that in normal adult cartilage three zones of cells are perceptible: (Fig. 1)—(*a.*) A superficial one of flattened cells, in section short spindles, lying parallel to the surface in three or four layers; (*b.*) a middle one of lacunæ, containing one or two cells, scattered sparingly through the matrix, and tending to lie horizontally, parallel as they approach to the surface; and (*c.*) a deep zone of lacunæ, larger and more numerous than in (*b*), containing various numbers of cells, and lying perpendicularly. There is no layer of epithelial cells lining the free surface. This usually disappears early in life, but may be found in adults at the margins, where they are not subject to pressure.

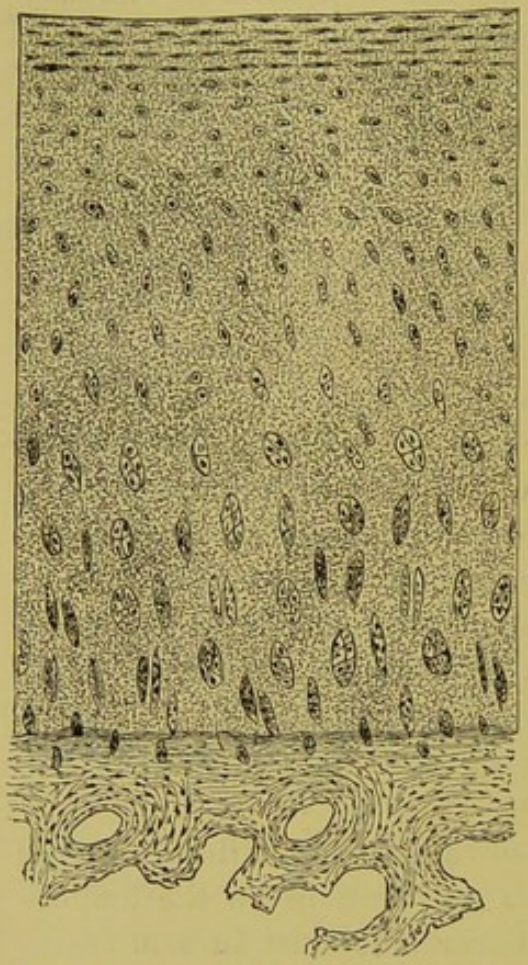


Fig. 1.—Human Articular Cartilage, from head of a metatarsal bone (Normal.)

There is no investment of synovial membrane on the free surface of articular cartilage. The superficial zone is beset with spindle-shaped cells lying in a matrix, which at the periphery exhibits a transition into fibrous tissue, continuous with that of the synovial membrane.

In persons not the subjects of gout, senile changes occur:—(*a.*) The superficial zone of flat cells disappears;[1] (*b.*) the cartilage-cells may proliferate, so that the middle and deep zones become indistinguishable; (*c.*) the matrix may exhibit fibrillation (*vide* Fig. 2); (*d.*) erosions may occur, which may even lay bare the bone.

As I have already stated, there is no investment of synovial membrane on the free surface of articular cartilage. The superficial zone is beset with spindle-shaped cells lying in a matrix, which presents also different characters from that in other portions of the texture. Thus, it has a fibrous aspect, and is striated horizontally for about one-sixth of the depth of the entire cartilage.

This fibrous appearance is sometimes clearly seen in sections where fibres are seen partly detached from the free surface in

[1] The superficial zone of flat cells has been found in the cartilages of the knee in a man aged sixty, in quite a normal condition.

ribbon-like bands. The round cells of the proximal layer become transformed into spindle-shaped ones towards the free articular surface.

The mechanism of nutrition of articular cartilage is still, as I have stated, a moot-point with anatomists. My friend and former preceptor, Professor Sir William Turner, of Edinburgh, is in the habit of teaching that "the encrusting cartilage of diarthrodial joints derives its nourishment in the adult partly from the vessels of the periosteum which reach its periphery, and partly from those of the synovial membrane, which not only reach its peripheral edge, but extend for a very short distance on its free surface, where

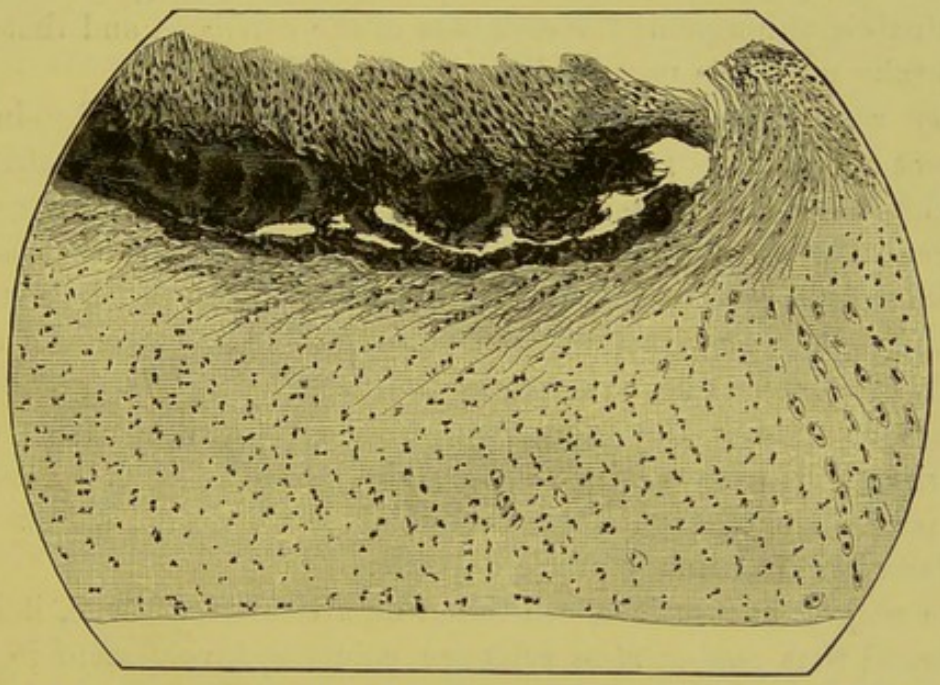


FIG. 2.—Articular Cartilage (Great Toe-Joint). Encrusted with sodium urate, which appears black by reflected light. Fibrillation of matrix and proliferation of cartilage-cells. The latter changes sometimes occur in old persons independently of gouty influence.

they form a definite vascular border—the "*circulus articuli vasculosus.*" Professor Turner denies that the cartilage is nourished by vessels derived from the sub- or superjacent bones.

Some differences of opinion exist as to the exact relation of uratic deposit to the several elements of articular cartilage. This subject has been carefully studied by several observers. The cells are held to be the centres of primary deposit by Cornil and Ranvier, by Charcot and Rindfleisch, and are believed to be actively concerned in it. William Budd[1] also conceived that there was some relation between the deposit and the cartilage-cells, which he regarded as the original foci of it. He remarked that "needles

[1] W. Budd, Researches on Gout, Med.-Chir. Trans., vol. xxxviii., 1855.

begin to radiate from these, not in relation to any cartilage-cells, but to the whole central mass; hence, the dynamic relations (if any) between the cell and the deposit are superseded by the common physical influences in action around it."

Other observers have conceived that the deposit occurs quite indiscriminately and irregularly. It is naturally a matter of difficulty to determine the truth in this matter. The appearances afforded by study of sections of the cartilage so infiltrated justify the opinion that this deposit, in crystallizing, pushes its way without special regard to the component elements of the tissue, and acts in respect of it as if it were an indifferent or homogeneous medium. My own impression is that the deposit progresses indiscriminately throughout the elements of the cartilage, and that the cells take no active part in directing or determining it.

My colleague, Mr. Bowlby, supports the view of Ebstein in respect of urates being only deposited in cartilage already damaged. He has observed that the articular cartilage is generally fibrillated and eroded.[1] He thinks it probable that some of the salt may be formed by disintegration of the cartilage itself, and, thus, agrees with Cantani,[2] who regarded the uric acid dyscrasia of gout as due to disturbed nutrition of this and of other tissues composing the joints, and with Robin,[3] who holds that gelatinous structures may disintegrate into uric acid. I admit the possibility of such a transformation, but I do not accept this explanation of ordinary uratic deposits.

In respect of uratic encrustation of articular cartilage, it is to be noted that *post-mortem* evidence points to involvement in this fashion of joints which have not been during life the seat of overt gouty attacks. In studying joints which are infiltrated in an early stage or in a lesser degree, it is observable, with some constancy, that the cartilage is more profoundly affected on prominent and central portions. Two reasons have been assigned for this. One is, that the prominent parts are those on which most pressure and friction are exerted, and therefore most likely to be injured and faulty in structure; and the other is, that the cartilage is in nearer relation to nutrient vessels at its periphery, and therefore less well-nourished and more vulnerable in its central portions. It is almost certain that uratic deposit is most favoured in parts which are least vascular and warm, and therefore most peripheral or distant from the circulatory centre.

[1] Surgical Pathology, p. 311, 1887.
[2] Quoted by Ebstein, *op. cit.*
[3] *Dictionnaire de Méd.*, 1865.

Ebstein declares that he has never seen urates crystallize in normal tissues. It is certain that such deposits have been found many years after a single attack of acute gout has occurred in a joint.

The changes induced by gout in articular cartilage have been referred to two stages, the infiltrating or depositive one, and the irritative or reactive inflammatory one. Study of a large number of gouty joints does not appear to warrant this classification. It seems probable that both changes proceed together in many instances, and it is certain that the irritative or inflammatory stage may be absent so far as overgrowths or gross changes are concerned. Deposition of urates may continue after the irritative action has begun. It has been urged that the latter stage is akin to the changes of chronic rheumatic arthritis which simply ensue on some irritative provocation, humoral or neurotrophic.

I am not prepared to deny this absolutely, though it must be allowed that such irritative changes often form no part of the gouty process, and in no case of gout do they ever reach the degree of development found in rheumatic or rheumatoid disease. I must again remark, that identity of morbid change is no proof of identity of the exciting cause in any given case. The same reasoning is applied to the deflections and distortions of the digits. This I accept, for these changes are often identical in gouty and rheumatic cases, and are sometimes entailed by the pain, but more often by the chronicity of the processes, however induced, in the affected joints.

Uratic deposit is not always found in the superficial layers of articular cartilage, but may exist on the under-surface of the cartilage, which is often swollen, or degenerated. (*Vide* Plate, Fig. 2.) A common site for this is the inter-condyloid space of the femur.

In the examination of many sections of non-gouty diarthrodial cartilage, it is common to find the distal layer worn away in adult or advanced life. This is proved by the absence of the spindle-shaped cells, which at this part lie horizontally, or parallel to the free surface. This layer is well seen in the cartilages of the young. The lacunæ usually found at the free surface of adult, or of gouty, cartilage are more or less round, and are such as occur in the centre of this structure in health, forming the middle zone.

With the able assistance of my present senior house-physician, Dr. Wynne, who has expended much time, skill, and study on my

behalf in respect of the minute anatomy of very many gouty joints, the following points have been determined :—

(1.) That the most frequent site of deposit is at the surface, extending about $\frac{1}{20}$ inch into the cartilage. (*Vide* Fig. 3.)

(2.) That, as a rule, the deposit has no special relation to the

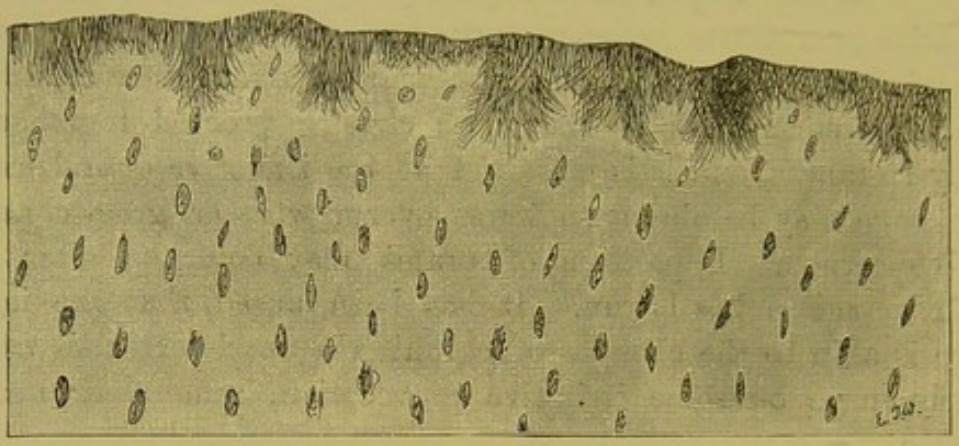


FIG. 3.—Articular Cartilage encrusted with Sodium Urate, which is deposited in the usual site at the free margin (knee).

cells, but in some cases the crystals of sodium urate are more numerous in and around the cells. (*Vide* Fig. 4.)

(3.) A less common site is in the deeper layers of the cartilage, starting in some cases from the bone. (*Vide* Plate, Fig. 2.)

In respect of the cartilage :—

(4.) In all cases the superficial zone disappears.

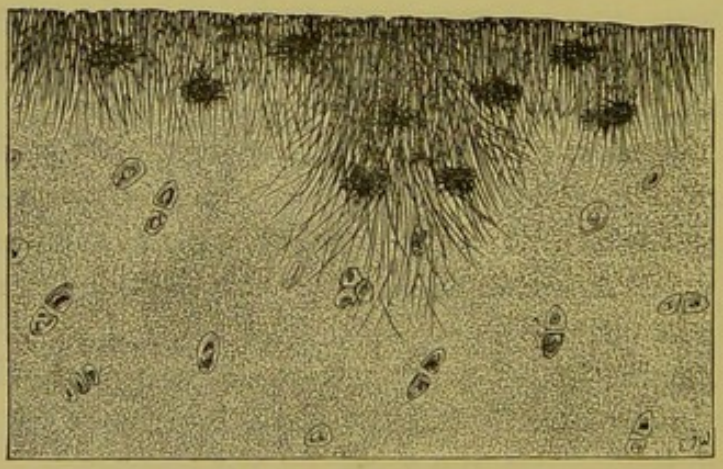


FIG. 4.—Illustrating Uratic Deposition in Cartilage-capsules.

(5.) As a rule, on washing out urates, no change is found in the cartilage, except that it is slightly more granular than elsewhere. An appearance is sometimes seen as if the cartilage were fibrillated in the direction inwards taken by the penetrating crystals, but we have never been quite sure whether this may not have been due to insufficient maceration, and, so, dependent on remains of crystals.

(6.) Treated with an acid (HCl), destruction of cartilage at seat of deposit was found, but appeared not to be due to the deposition, being only met with when the latter was very abundant.

(7.) Quite exceptionally, an appearance of a funnel-shaped cavity, as figured by Ebstein, and described by him as necrotic, was met with proceeding inwards from the free surface. This is probably not indicative of necrosis, because no signs of irritative change, such as might be expected, were to be found in the immediate neighbourhood. The appearance may be accounted for by the washing out of the urates, which in these situations are

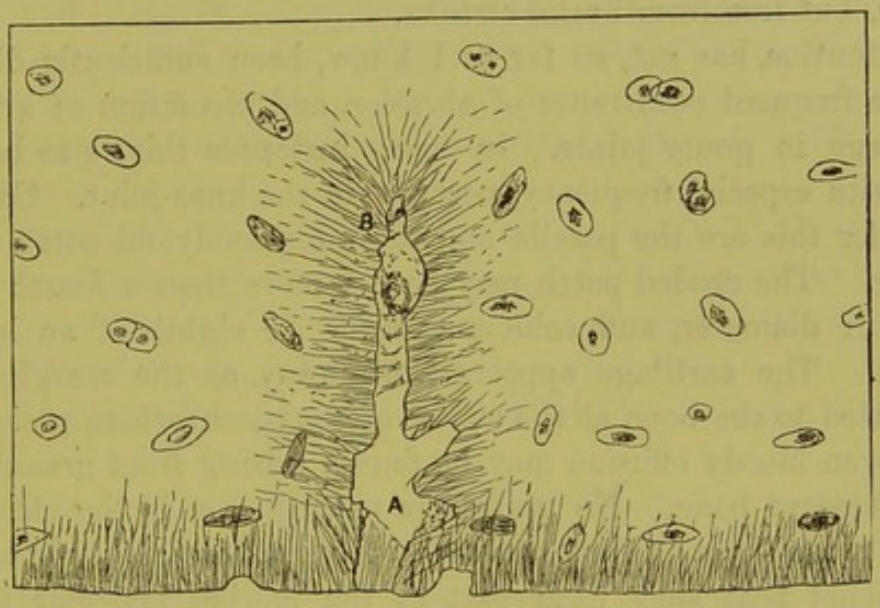


FIG. 4A.

(A.) Showing a (? necrotic) patch, as described by Ebstein, in cartilage from gouty knee-joint; (B.) Urates partly removed by distilled water. (This appearance was not present in all the sections examined, and only occurred where the uratic deposit was dense).

abundant, causing the involved matrix to crumble away, owing to failure of support.

Our general conclusions, therefore, are that there is no special microscopical condition of cartilage peculiar to gouty deposit; that the common site of deposit is at the free surface, but that it may occur at any point, and that the cells are not *foci* of deposition.

Although uratic deposition may occur in any tissue during life, its most frequent site *post-mortem* is in the cartilages, because means for its removal are there least efficient.[1]

[1] The encrustations are always, in my experience, formed by urates in slightly curved needles, sometimes lying parallel, or radiating outwards. In the cells they are prone to form tufts, which bristle with needles like teasel-burrs. (*Vide* Figs. 3 and 4.)

According to Dr. Ord, this acicular form of crystallization should not occur in the substance of a colloid substance such as cartilage, and he thinks a spheroidal form is what might naturally be expected in such a texture. He remarks: "It may be assumed that the needles are to be regarded as crystals, though their appearance of flexibility, their remarkable tenuity, and the absence from them of angularity of

Uratic deposit is not always found as an enduring evidence of the existence of past attacks of gout in a joint. Garrod has affirmed this to be the case. The following case may be cited in proof of the contrary. A man, æt. forty-three, was under my care suffering from chronic pulmonary phthisis and interstitial nephritis. He was a brassfounder, and had used lead. There was no blue line on his gums. He had drunk freely of beer. He died of acute bronchitis. He had had two attacks of gout in the right great toe-joint. At the autopsy neither toe-joint contained a speck of uratic deposit. The kidneys were large, granular, and cystic, but free from uratic streaks.

Attention has not, so far as I know, been sufficiently directed to the frequent occurrence of abrasion and ulceration of articular cartilage in gouty joints. In many instances this is to be seen, and with especial frequency, perhaps, in the knee-joint. Common sites for this are the patella and the inter-condyloid notch of the femur. The eroded patch may be not more than a fourth of an inch in diameter, and seldom exceeds an eighth of an inch in depth. The cartilage appears worn away at the margins, and ulcerated to the bone at the centre of the patch, where vascularity and even bloody effusion may be found, arising from granulations of subjacent bone. No urates are usually seen in the vicinity of these ulcerations. They are so frequent as to impress one with the belief that they form part of the morbid articular process apart from mere uratic encrustation, and, so far, they support the views of Ebstein already alluded to.[1] These points are not to be confounded with the grooved lines of erosion met with in chronic rheumatic arthritis, but they are significant of irritative change.

In the great toe-joint, erosions are met with not infrequently, and in this situation such changes are common as a result either

section are departures from the typical qualities of the crystal. They are to my mind crystals with definite colloidal affinities." * Again, "The needle, though a crystalline form, is not by any means the true or perfect crystalline form of urate of soda. The true form is a short, six-sided prism. The needle of urate of soda occurs where uric acid would be found in spheres, and urate of ammonia in molecules. But it also occurs where uric acid would be in crystals—that is to say, where no colloid save colloidal manifestations of itself exists."

[1] It is only right, however, to state that such erosions are not very infrequent in the same situations in joints of persons who present no overt signs of gout. In the dead-house at St. Bartholomew's the joints of a large proportion of the cases are regularly examined. Erosions are, however, more frequent in gouty than in non-gouty joints.

* On the Influence of Colloids upon Crystalline Form, W. M. Ord, M.D., 1879, p. 67.

of old injuries, or of premature decay of the cartilage. As these erosions are found in middle life, they must be classed either as prematurely senile changes, or as specifically gouty.

In an elaborate and very valuable contribution to the Morbid Anatomy of Gout by my colleague, Dr. Norman Moore,[1] the following are some of the conclusions arrived at in respect of the changes in joints, derived from study of eighty cases:—

1. That degenerative changes are usually present in the same joint, or in other joints of the same body, or in both, where urate is present even in a single joint.

2. That deposits of urate of soda, like other degenerative changes, are usually more or less symmetrically arranged on both sides of the body.

3. That it is commoner to find a deposit in the joints of the legs than of the arms.

4. That a deposit may be present in nearly all the joints of the lower limbs, and absent from those of the upper limbs.

5. That a deposit is commoner in the metatarso-phalangeal joint of the great-toe than in the phalangeal joint.

6. That however abundant in and below the knees, a deposit is rare in the hip-joint.

7. That a deposit is often found in the great-toes and knees when absent in the ankles, but not in the ankles, when absent in the toes and knees.

8. That when present in the ankles, some deposits may be usually found in the ligaments of the foot.

9. That the elbow-joint has deposit when one is present in the wrist.

10. That the sterno-clavicular joint rarely contains deposit.

11. That the articulations of the larynx rarely contain deposit.

Dr. Moore found that extensive deposit may exist in the articular cartilages without any external deposit, such as tophi in the ear, and that it is comparatively rare for the latter to be present in such cases. It is, further, to be noted that urates may be absent from the interior of nodular joints, while specks of them may be found externally in their ligaments and adjacent tendons.

The immunity of the hip-joint is remarkable. Garrod records a case and depicts the appearances in a man, æt. fifty-four, where deposit occurred on the head of the femur, in the acetabulum, and in the ligamentum teres. In chronic rheumatic arthritis the hip-joint is especially liable to be involved, such cases being often termed "hip-gout." In these there may be

[1] St. Barth. Hosp. Reports, vol. xxiii., 1887.

profound bony changes following on absorption of the cartilages, with permanent crippling as a result.

The shoulder-joint is singularly free from uratic deposit or gouty changes.

The order of frequency of uratic deposit in the knee is, according to Dr. Moore, the articular surface of the patella, inter-condyloid groove of femur, condyles, and lastly, the surface of the tibia.

Ebstein maintains that deposits occur chiefly in parts where tissue-change is least active, and where the conditions for separation from nutrient fluids are, thus, most favourable. By direct experiment he proved that uric acid has a highly injurious effect on certain tissues, and especially upon the cornea.

Deposits occur in the marrow of bone, usually, but not always, in the neighbourhood of encrusted cartilages, and they sometimes appear to be due to direct destruction of the bony lamina by the continued pressure of intra-articular accumulations.[1] (*Vide* Plate, Fig. 2.) Cases are, however, met with where the articular cartilage is intact, and yet deposit has occurred in the bone. In the Museum of St. Bartholomew's Hospital are several specimens illustrating this. Dr. Wilks found in a man, æt. forty-six, who had suffered for sixteen years from gout, and had tophi round the digital joints, uratic deposit in the centre of the first phalanx of the ring-finger. This finger was amputated by Mr. Bryant for pain caused by disorganization of one of the joints.

The circulation is probably too active in this situation to allow of deposition, and, thus, marrow takes its place with other tissues and organs which are for the most part exempt from deposit, with the exception just mentioned.

It is noteworthy that the bones of the gouty manifest, when dried, a more fatty appearance than bones taken from the subjects of rheumatic, strumous, or other diathetic disease. There is nothing specially to be remarked in respect of this greasy appearance in recent bones from gouty subjects. Sometimes they appear oily, and there is possibly a greater amount of fat in the medullary portion than is normally the case. Analyses of gouty bones by Marchand, Lehmann, and Bramson have shown diminution of earthy salts and increase of fat. M. Budin[2] (quoted by Rendu) found rarefaction of spongy tissue and islets of ostitis, granulo-fatty transformation of osteo-blasts (marrow-cells) and dilated

[1] In a case reported by Féréol (*Union Méd.*, p. 827, 1869) deposit was found in spongy tissue of a phalanx unconnected with the joint. (Quoted by M. Rendu, *op. cit.*)

[2] *Bull. de la Soc. Anat.*, 1875, p. 712.

vessels, surrounded with crystals of margarine. These changes are probably largely dependent on a chronic gouty cachexia, and are hardly to be expected in recent or less grave cases. The bony changes occur late, and subsequently to involvement of cartilage and less yielding structures, and are, therefore, so far evidences of chronicity. An exception must be made to this statement in respect of the primary affection of bone which occurs in deforming gouty arthritis, when nodes such as those described by Heberden [1] and Haygarth [2] are produced. These consist in overgrowth of the natural tubercles of the distal digital phalangeal bones. I am convinced that amongst cases referred

[1] *Nodi digitorum.*—This term was applied to the knotty or knobby state of the terminal phalangeal joints by Heberden. It is commonly taught that these are not of gouty origin, and Heberden denied that they were so. "Nihil certe illis commune est cum arthritide; quoniam in multis reperiuntur, quibus morbus ille est incognitus." * Heredity is strongly marked in respect of these nodes. An eminent member of the profession thus relates his own case to me. "I am up to a certain point, though in a very minor degree, a living specimen of 'digitorum nodi,' of which, however, I am aware of three generations, at least, of ancestors who had them in a much more confirmed form, and associated in too many instances, as they are in the case of two of my brothers and one sister, with really serious contractions in the palmar fascia. But no one of us all, so far as I know, ever had a fit of the gout, and this holds true, I believe, of all the three generations. Rheumatism, more or less defined, appears in one of my brothers, and glycosuria in another;† but none of us has ever had rheumatic fever, and this also applies to all three generations, as also the immunity, I believe, from valvular heart-disease. It is very interesting that in a collateral branch of our cousinhood, descended from our great-grandfather, gout appears in close association with the digitorum nodi and the palmar contraction; but then the father of these was gouty and probably earned it, our connection with them being through the mother, who probably brought in the digital deformities. I may add that the nodes run mostly, with us, in the female line, and, so far as I know, they have come on without any kind of painful symptoms, and usually only after the middle term of life."

These nodes are met with in persons in very advanced life. Charcot has noted that in women thus affected, cancer of the mamma and uterus is not an infrequent event, and my own experience is somewhat confirmatory of this. When occurring in men, without any pronounced gouty or rheumatic concomitants, they may support a general prognosis for longevity. In women they often coexist with hemicrania, asthma, severe headaches, and other troubles, which are properly recognized as gouty manifestations of the sex. They may precede by many years overt gouty attacks. Dr. James Begbie, of Edinburgh, who had large consulting practice amongst the upper ranks in Scotland, was convinced of the occasional gouty nature of Heberden's nodes,‡ and recorded several cases in proof of this. He noted that they were seldom or never seen on the fingers of the industrious labourer or hard-working mechanic, but found chiefly among the upper classes or the luxurious and well-fed of their dependents.

[2] Clin. Hist. of Diseases, Part I., Acute Rheumatism and Nodosity of the Joints, 1805.

* Commentarii, cap. xxviii., 1802.

† This brother has since had a severe attack of gout in left great toe-joint.

‡ Contributions to Practical Medicine, Edinburgh, 1862, p. 27.

to the latter categories are some of unquestionable gouty nature. These may arise from the second to the fifth decade, are most common, perhaps, as a gouty manifestation in women about or after the menopause, but are not uncommon in men and even in children. A well-marked instance is depicted in Fig. 5, where the diagnosis of rheumatoid arthritis was made during life by my colleague, Dr. Gee, who kindly permits me to record the case. Unequivocally gouty changes were found after death with small scattered uratic deposits.[1] True synostosis occurred in several of the phalangeal joints. This is shown at A in Fig. 6, where the joint has been vertically bisected. No line of junction is seen nor any remains of the compact layers of bone. Garrod[2] records a case of synostosis of first joint of great-toe after a few attacks of gout. Scudamore[3] relates the details of a dissection by Brodie of a gouty old woman, in whom several joints of the fingers, as well as the right wrist and elbow, and several toe-joints, were ankylosed. "Chalky matter" was found on the bones where the cartilage had disappeared, and "exostoses" (lipping) were present at the edges of the knee-joint.

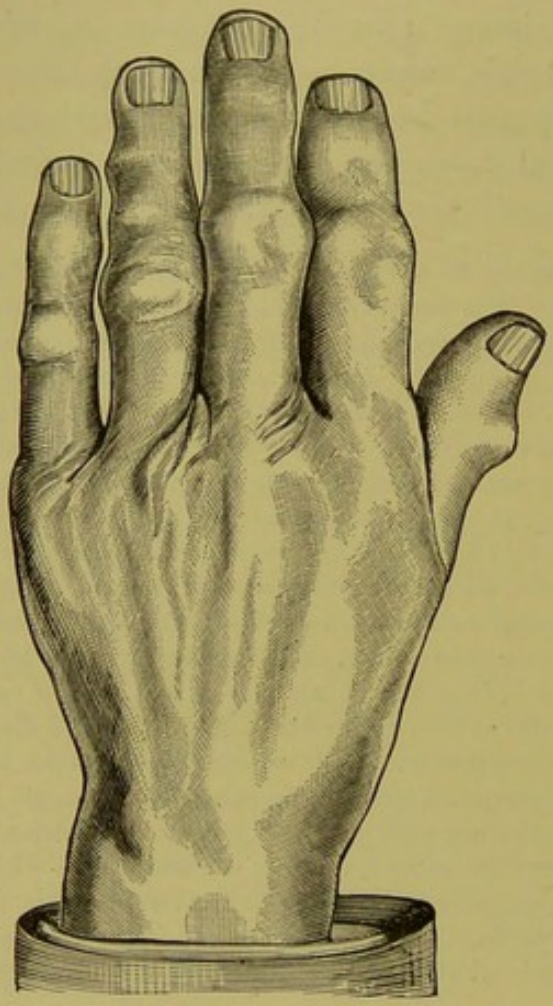


FIG. 5.—Left Hand of E. G. Drawn from a plaster cast taken during life. Illustrating *nodi digitorum* of truly gouty nature.

[1] *Heberden's Nodes in a Case of Gout—Jaundice—Death from Cancer of the Liver.*—E. G., nurse, aged 63. No history of rheumatism, or of gout, known of in the family. Never had rheumatic fever. Began to have what she called "gout" in the joints of the fingers ten years ago. Attacks first came in the interphalangeal joints of the thumbs. Has had it many times since, and it has affected from time to time all the joints of the fingers. Since the attacks began, patient has noticed a gradual increase in size of the joints of the fingers. Soon after the attacks of "gout" began in the fingers, she had similar attacks in the metatarso-phalangeal joints of the great-toes, and since then in the ankles and knees. No enlargement of these joints was noticed by the patient. No tophi. Heberden's nodes, both hands. Bones of metatarso-phalangeal joints of both great-toes slightly flanged (lipped). Nothing noticeable found in the other joints. (The hand is preserved in the Hospital Museum.)

[2] *Op. cit.*, p. 194.

[3] *Op. cit.*, p. 48.

In some examples of interstitial nephritis, so-called cases of primary renal gout, such nodular arthritis is met with unassociated clinically with uratic deposit (*vide* p. 99). The enlargement consists in overgrowth of the natural tubercles of the phalanges, which become, with the heads of the bone, somewhat expanded. The process is often gradual and almost painless, though there may be occasional uneasiness and burning sensations in these joints.

It is probable that Heberden denied the appellation of gout to those nodules because they occurred without the ordinary classical manifestations of gouty arthritis, and were met with in persons who presented none of the recognized and overt signs

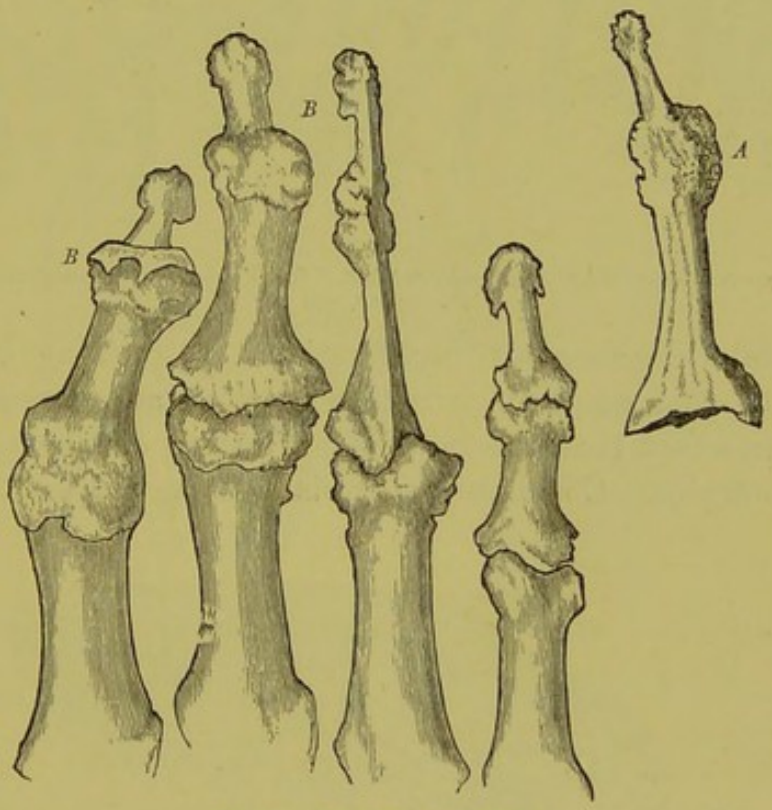


FIG. 6.—Right Hand of E. G.
(*A.*) Illustrating true gouty synostosis; (*B.*) Nodi digitorum.

of what, in his time, was alone regarded as true gout. It is certain that nodes undistinguishable from these occur in persons who are not gouty. Their gouty nature, in any given case, is determined by other concomitants, which may be discovered by those who know what to look for. These nodular enlargements are often red, and prone to become hot and painful from various causes. Fugitive achings may occur in them after dietetic errors.

Chronic irritation of articular cartilage leads in any case of arthritis, however induced, to the formation of exostoses or ecchondroses — so-called "lipping" — which arise beneath the synovial membrane at the edges of the cartilages, and round

the heads of the phalanges or other bones, as in the femora, patellæ, or tibiæ. In gouty arthritis these are less exaggerated than in truly rheumatic disease, and, as already noted, seldom

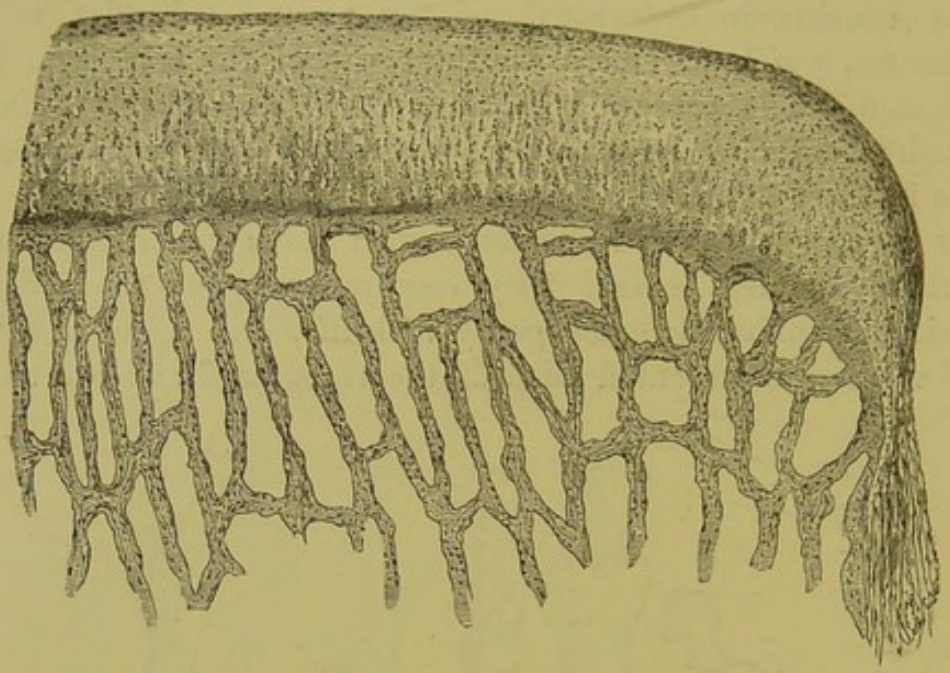


Fig. 7.—Section of Margin of Healthy Adult Knee (Femur), for comparison with Figs. 8, 9, 10, and 11.

lead to stalactitic (osseous) proliferation. In opening joints thus affected, everted edges of irritative exostosis are witnessed, often more translucent than the naturally investing cartilage, and with sinuous margins. Commonly, there is associated deposit of urates,

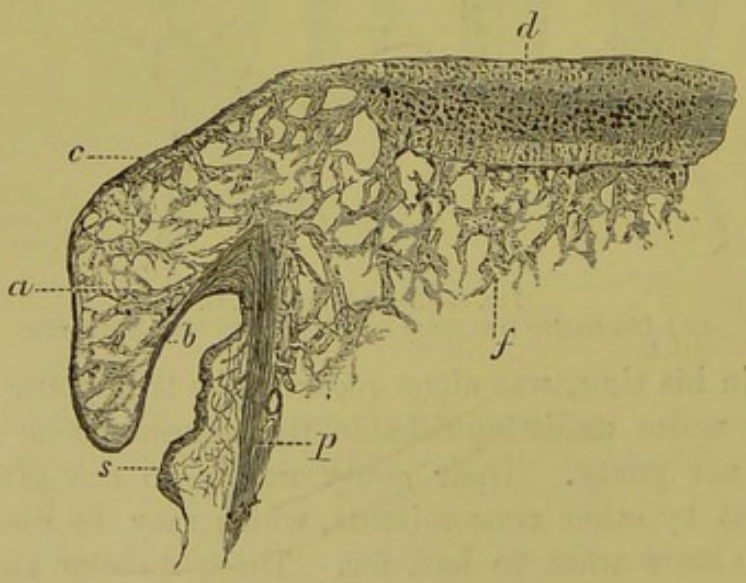


Fig. 8.—Section at Edge of Femur showing Gouty Exostosis or "Lipping."
(*a.*) Osteophyte of ill-formed cancellous bone; (*b.*) Reflection of synovial membrane (*s*); (*c.*) Limit of cartilage cells; (*d.*) Articular cartilage of femur; (*f.*) Cancellous tissue; (*p.*) Periosteum.

more or less, but not always, and this is rarely seen over the exostosis, but more often towards the centre of the cartilages. It may, however, occur in proliferating cartilage, occupying the primary capsules, which include numerous secondary ones.

As pointed out by Cornil and Ranvier, this formation of marginal outgrowths is inevitable in any form of chronic arthritis. There is less in the scrofulous than in the gouty form, and most in the rheumatic variety. Proliferation occurs at the borders of the articular cartilage, and atrophy at the centre. Cornil and Ranvier affirm that the former process is caused by the fibro-synovial investment of the edges, already described, whereby the proliferating elements of the cartilage, fibrous villi and cells, are shut in, and must, perforce, accumulate, instead of being cast off

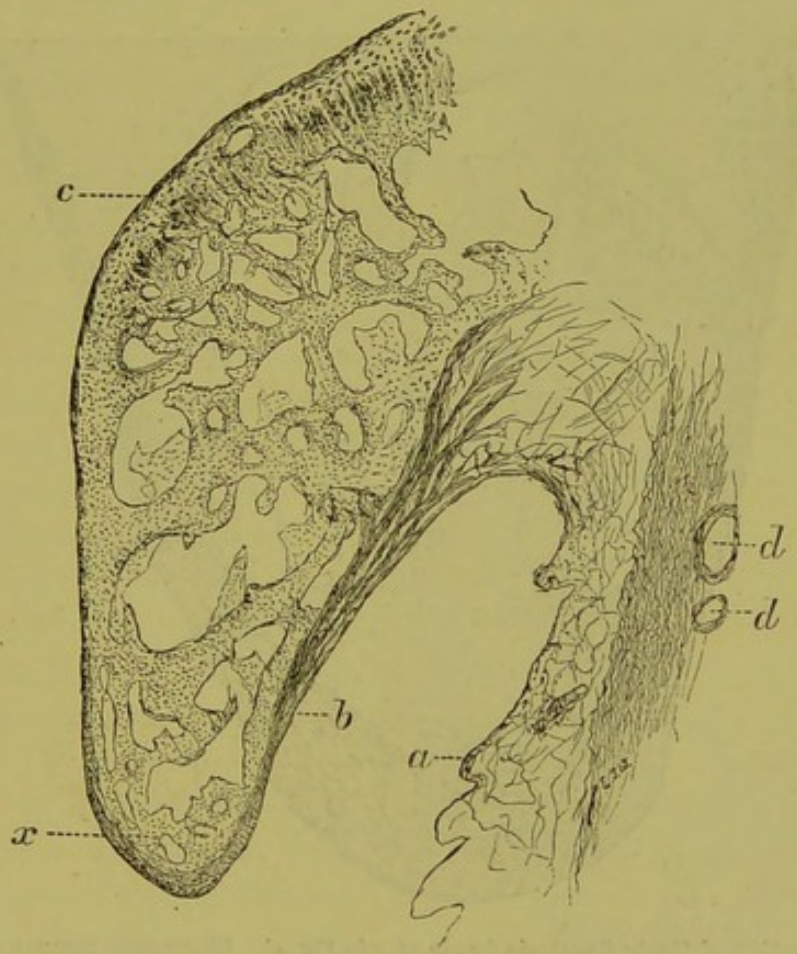


FIG. 9.—Section of Gouty Exostosis.
(*a.*) Synovial fringe; (*b.*) Prolongation of same over outgrowth; (*c.*) First appearance of cartilage-cells; (*d.*) Vessels of periosteum.

into the cavity of the joint, as occurs in the central or non-synovially invested portions. In this manner nodules are produced in articular cartilage, on synovial fringes, and on tendons and ligaments, fibrous synovial investment at these points surrounding the irritative overgrowth.

Dr. Wynne has made a careful study of the intimate structure of these marginal outgrowths, both in gouty and chronic rheumatic arthritis, and I am indebted to him for the details and illustrations which follow.[1] (*Vide* Figs. 7–11.)

[1] *Vide* Note on a Point of Difference in the Pathology of Gouty and Rheumatoid Arthritis, by E. T. Wynne, M.B., Cantab. Lancet, May 11, 1889, p. 933.

Marginal outgrowths in gouty arthritis are true exostoses.—The peculiar enlargement has much coarse resemblance to that met with in cases of chronic rheumatic arthritis. It is not *directly* connected with uratic deposition, and the enlarged portions are commonly void of such deposit. It is apparently due to an overgrowth of cartilage; but this tissue does not extend to the summit of the outgrowth, and is replaced by a yellow, translucent texture, which offers great resistance to the knife. Examined microscopically (*vide* Figs. 8, 9, 10), this latter

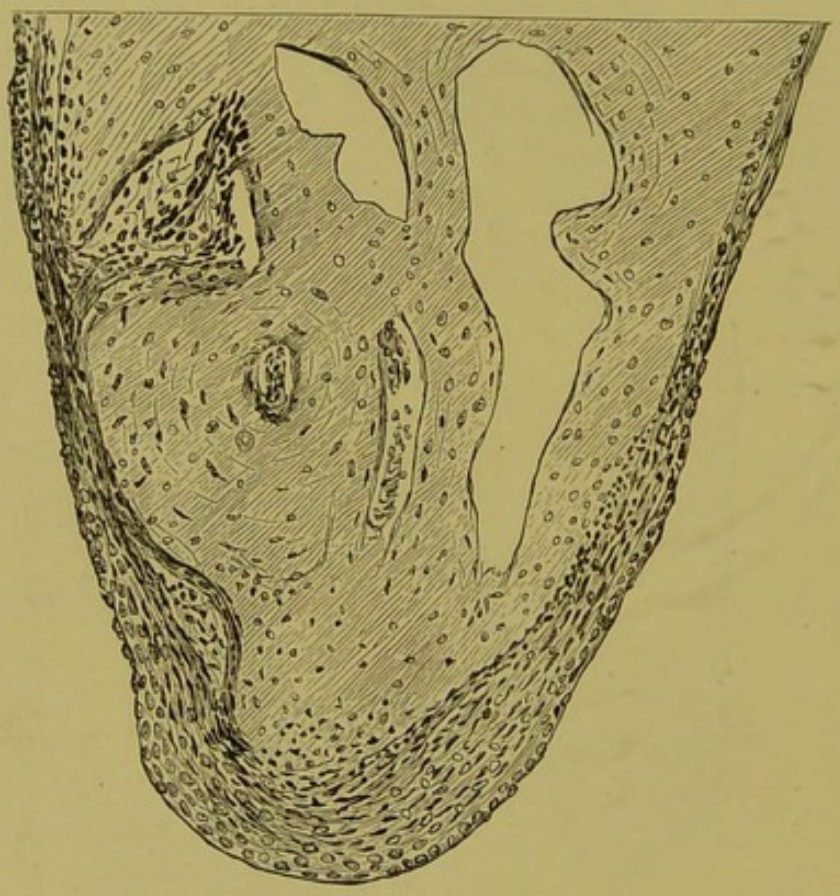


FIG. 10.—Section of Gouty Exostosis (made at *x* in Fig. 9). Illustrating absence of encrusting cartilage, the bony outgrowth being invested by fibrous tissue prolonged from the synovial membrane.

substance is found to be composed of bony matter, the epiphysis appearing to be locally hypertrophied, and pushing the encrusting cartilage before it. The latter is seen to terminate abruptly at the summit of the protuberance, when it becomes continuous with a thin layer of fibrous tissue derived from the periosteum and synovial membrane. Beneath this fibrous tissue is found spongy bone, identical, and continuous, with the cancellous texture of the subjacent bone. These appearances are constant in true gouty arthritis.

Marginal outgrowths or "lipping" round joints affected with

chronic rheumatic arthritis are due to ecchondrosis.—In chronic rheumatic arthritis there are found ridged or lipping outgrowths due to overgrowth of cartilage, usually fibrillated, which may in their deeper parts be calcified, but rarely show true bone-structure. In the case of gouty arthritis, therefore, this "lipping" outgrowth is a true *exostosis*, while in the rheumatic variety of this enlargement the change is a true *ecchondrosis*. (*Vide* Fig. 11.) Dr. Wynne suggests that, in the latter, the change is probably due to a nervous dystrophy, while in the gouty form the overgrowths result from the irritative action of uratic deposit in the vicinity, or from the abnormal presence of urates in the blood circulating through the bone. These results constitute a new contribution to the intimate morbid anatomy of gout and of chronic rheumatic arthritis, which reflects great credit on the painstaking observations of Dr. Wynne. In particular, they contradict the teaching of MM. Cornil and Ranvier, which has up to this time been generally accepted. These observers regard the changes in question as due, in each case, to true ecchondroses. They must henceforth be differentiated.

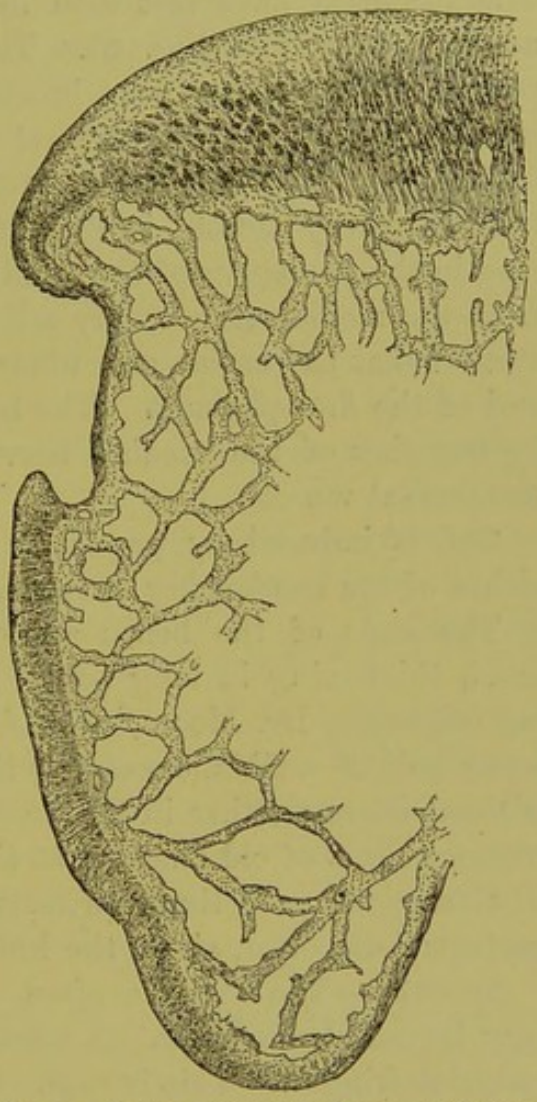


Fig. 11.—Section of Ossifying Rheumatic Ecchondrosis (Lipping), showing complete investment by cartilage, which is undergoing fibrillation.

Erosion of the cartilage may occur, and atrophy from continued motion in a joint admitting free play. It is rare to meet with it in those, such as the carpal or tarsal joints, which move but slightly. This happens, perforce, in gouty and rheumatic arthritis, but greater ravages occur in the latter. In joints thus affected, pain on motion is felt during life, and crackling is also audible. Pain is not always present, unless there is active gouty inflammatory process at work. The knee is perhaps most frequently thus affected.

Eburnation appears to be extremely rare as a result of gout in a joint, and, indeed, the measure of bony changes anywhere

in true gout seems to be in relation both to the severity and chronicity of the attacks in the part.

It is noteworthy that true bony ankylosis (synostosis) is met with as an occasional result of gouty arthritis, while it is almost, if not quite, unknown as a sequel of chronic rheumatic arthritis. In the latter, false ankylosis is common enough from proliferative periarthritic growths, or from fibrous or fibro-cartilaginous change. The former I have met with in the great-toe [1] and in the phalangeal joints (*vide* Fig. 6). The cavity of the joint may be replaced by new spongy bone in which a white line (seen on section) indicates sometimes the original articular surfaces.

The joint may be filled with urates separating the two bones.

It is worthy of note, in respect of the varied deformities occurring in the phalanges of the hand, that their innervation is derived from the median nerve. My colleague, Mr. Walsham, has demonstrated that the radial and ulnar nerves do not go beyond the end of the first phalanx. The terminal extremities are animated by branches of the median nerve which pass onwards from the metacarpal muscles.

Ostitis induced by proximity of infiltrating deposit may be either of the condensing or rarefying varieties.

The ends of the bones are often enlarged. There may be much thickening in long bones, as in one instance, recorded by my colleague, Dr. Moore,[2] who found new bone (osteoma) in the lower half of a tibia, exceeding the diameter of the normal bone, without change either in the medullary cavity or periosteum, and without signs of old fracture at the part.

Urates are sometimes present in the fat outside of joints, perhaps most often about the knees.

Synovia.—The synovia often contains specks of urates, and may be unduly vascular. I have met with spicules of urates on synovial fringes. This is rare. I have found the synovia alkaline in a gouty knee-joint, with granular flocculi and acicular sodium urate crystals in it. With the thread-test I got negative results in the clear fluid. It contained no glucose. In another case, where the synovia was very abundant in an encrusted knee-joint, I found the reaction slightly acid. The fringes may become much hypertrophied and infiltrated with fat (dendritic lipoma). Deposits may be found in every component tissue

[1] Mr. Shattock has reported an example of true gouty synostosis of the great-toe (Path. Soc. Trans., p. 280, vol. xxxix. 1888.) He kindly showed me this specimen. Uratic deposits were found around the joint.

[2] Path. Soc. Trans., vol. xxxiii., p. 275, 1882.

of a joint, and are met with in connective tissue, intermuscular connective tissue, in nerve-sheaths, periosteum, prævertebral fascia, tendons and their sheaths, ligaments, and in fibro-cartilage.

Axial distortion of digits ("seal-fin" type of hands).—A noteworthy point frequently to be observed in gouty arthritis, as in most other forms of chronic arthritis, is the peculiar deflection of the digits to the ulnar side of the fore-arm. The toes sometimes assume a distortion to the outer side of the foot. It has been taught that this is a specific indication of chronic rheumatic arthritis, but this is certainly not true. I think the deflection is more often found in rheumatic than in gouty cases, but I have met with it equally well-marked in both. Chronicity is certainly signified by it.

The cause of this axial distortion is to be found in the influence of the extensor muscles of the wrist and fingers. The movements of adduction at the wrist are more free than those of abduction, and, therefore, the muscles when unchecked tend to draw the fingers inwards. I believe, with Charcot and others, that there is a reflex action on the musculo-motor nerves excited by irritation of the sensory branches in the affected joints leading to spasmodic contraction, which in course of time induces the characteristic distortion of the digits referred to.[1]

In the case of the deflected or abducted toes, it has been sought to prove that these result entirely from badly-shaped boots. Mr. Arbuthnot Lane, in particular, is dogmatic on this point.[2] I cannot agree with this view. It is certain that most feet are distorted and compressed to some degree by wearing boots, but the cases in which abduction is considerable are almost

[1] Dr. Herringham seeks to explain the ulnar deflection of digits as a result of atrophy of the abductor indicis muscle, which leads to this displacement of the index finger, itself pressing, in turn, on the other digits. Against this view must be placed the fact that such atrophy is most inconstant, and, therefore, not present in all cases.

Direct experiments have been made to determine the influence of muscular contraction in inducing deformities of joints. M. Valtat made caustic injections into the joints of dogs and guinea-pigs, and found afterwards contractures of muscles bordering on these articulations. He witnessed contractures first induced by arthritis, and subsequent atrophy of the muscles, and attributed this to over-excitement of reflex activity of the spinal chord from prolonged peripheral stimulus. As analogous instances, he mentions articular spasm caused by irritation of the conjunctiva, and anal fissure maintained by spasm of the sphincters. He regards contracture as the analogue of hyperæsthesia, occurring when the motor portion of the chord is stimulated (Lecture by M. Charles Richet, Lancet, May 21, 1881, p. 816).

[2] Path. Soc. Trans., vol. xxxvii., p. 433, 1886. Mr. Lane attributes the "so-called" disease—chronic rheumatic arthritis—entirely to the results of transmission of pressure.

always, in my experience, the subjects of rheumatic or gouty habit. The distortions are very frequent in women, even in ladies, who seldom wear such strong boots as alone could compass the extreme changes referred to. If boots alone were to blame, such distorted feet should be much more common than they are. In extreme cases the great-toe is everted almost at a right angle, and overlaps all the other toes. Cases of unequivocal gout

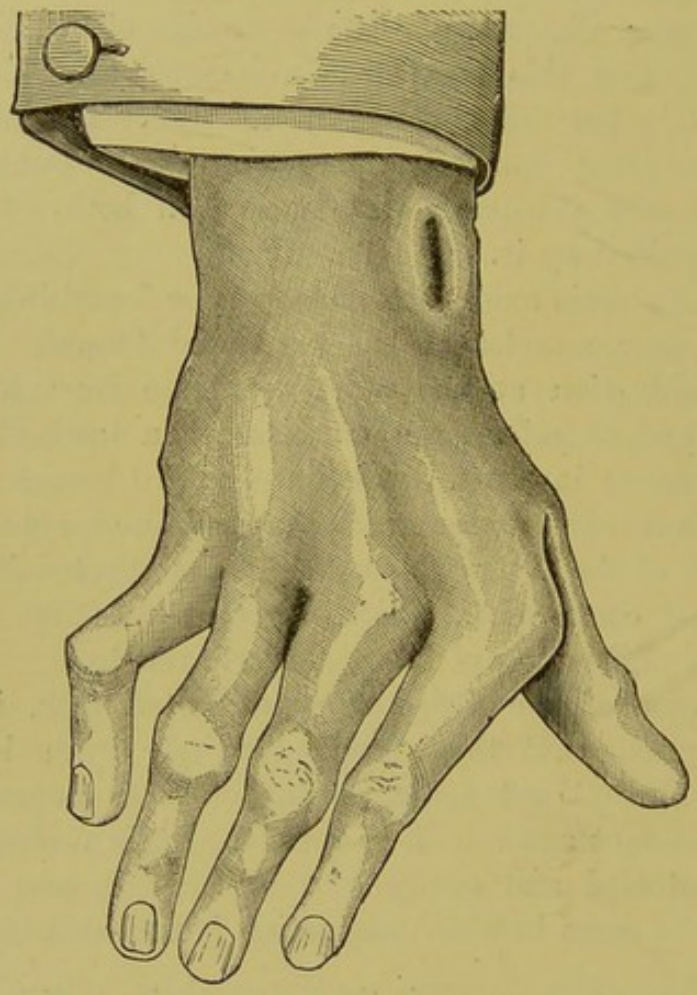


FIG. 12.—Tophaceous Gout of Right Hand. Deflection of digits to ulnar aspect. On the wrist a scar of a large "chalky" deposit which had been treated by incision.

are often seen where, with involvement of small joints, the digits are quite straight in their axial lines, and it is commoner to see merely displacements of the phalanges in one or two fingers. Thus, the terminal bones may be bent in or out, and a frequent change is a deflection outwards of the last phalanx of the fore-finger, and one inwards of that of the little finger in the same case. I have observed this with some frequency in chronic gouty hands of women.[1] (*Vide* Figs. 13, 14.)

[1] "Adduction is effected to a greater extent than abduction in consequence of the mode of disposition of the lateral ligaments, and with greater power in consequence

Flexion into the palm of the first phalanx of the middle finger, and of the distal phalanx of the same digit, is somewhat common in men, and the bones of the ring-finger may be similarly involved. These changes are seen with and without much uratic deposit (Fig. 15).

One of the most commonly affected joints is the metacarpo-phalangeal of the forefinger. It is rare to find deposits elsewhere, if this joint be free.[1] All the types of deflection described by Charcot as met with in chronic rheumatic arthritis, or combina-

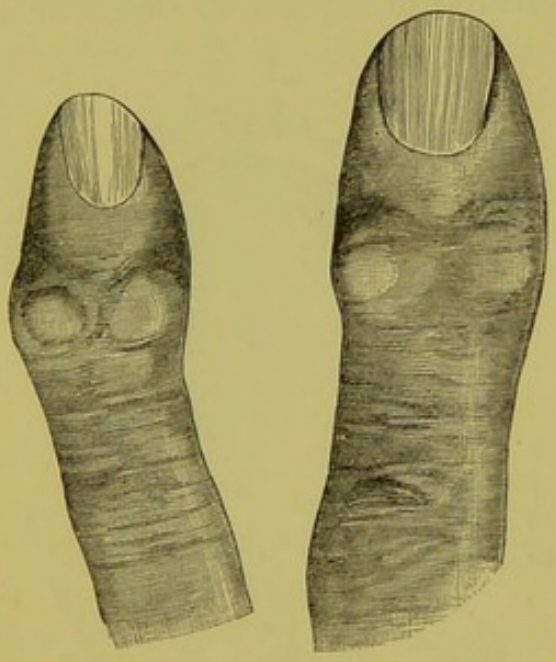


FIG. 13.—Gouty Heberden s Nodes. Illustrating common forms of terminal phalangeal deflection. Forefinger and little finger of a woman, æt. 70. "Crab's-eye" cysts over the joints are also depicted.

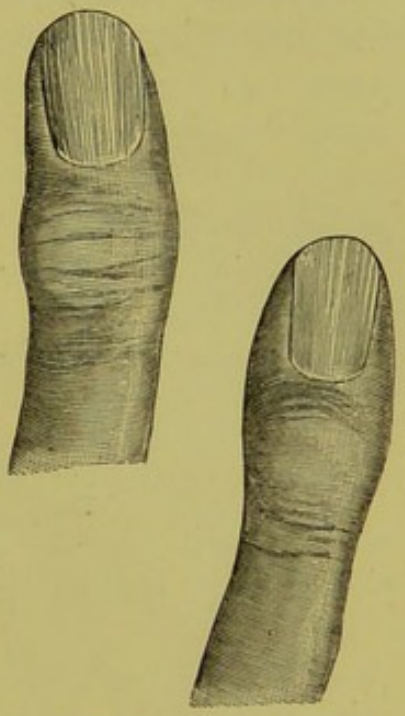


FIG. 14.—Illustrating Nodular Swellings (Heberden's Nodes) due to gouty arthritis on the fore-finger and little finger of a lady, æt. 50.

tions of them, may be found in the gouty; and the same law, doubtless, explains them in both diseases.

Hæmorrhage may occur into the joints. Dr. Pye-Smith has recorded an instance where this was met with in the knees, hips, great-toes, ankles, wrists, and one elbow. The shoulders were free from bloody effusion. The blood was dark and recent, but

of the leverage afforded by projection of the cuneiform and pisiform bones on the inner side of the wrist. Thus, the hand assumes the position of adduction and the little finger becomes inclined to the ulna when, from disease or other cause, the muscles lose the influence of volition, and exercise an uncontrolled sway over the part."—*A Treatise on the Human Skeleton*, p. 427. Prof. Humphry, Cambridge, 1858.

[1] In one case with deposits in great-toes, right knee (only one opened), and left ear, this joint was free from deposit, ibut the cartilage was much eroded. This occurred in a man, æt. circ. forty-five, who died from cerebral hæmorrhage, and had granular kidneys.

in one hip was rusty and brown. In some joints the synovial membrane was infiltrated with blood. In this case the choroid plexus was deeply discoloured, owing to infiltration of the pia mater with large altered blood-discs, which had not, however, disintegrated.[1] Such an example recalls somewhat the appearances met with in the joints in some cases of hæmophilia.[2]

The synovial membrane is sometimes greatly congested, and thrown into folds, which project into the joints. Fagge recorded a notable example in which loose flakes of lymph, much synovial fluid, and, in one joint, thin watery pus were also found, the latter burrowing into the adjacent thenar muscles of the thumb.

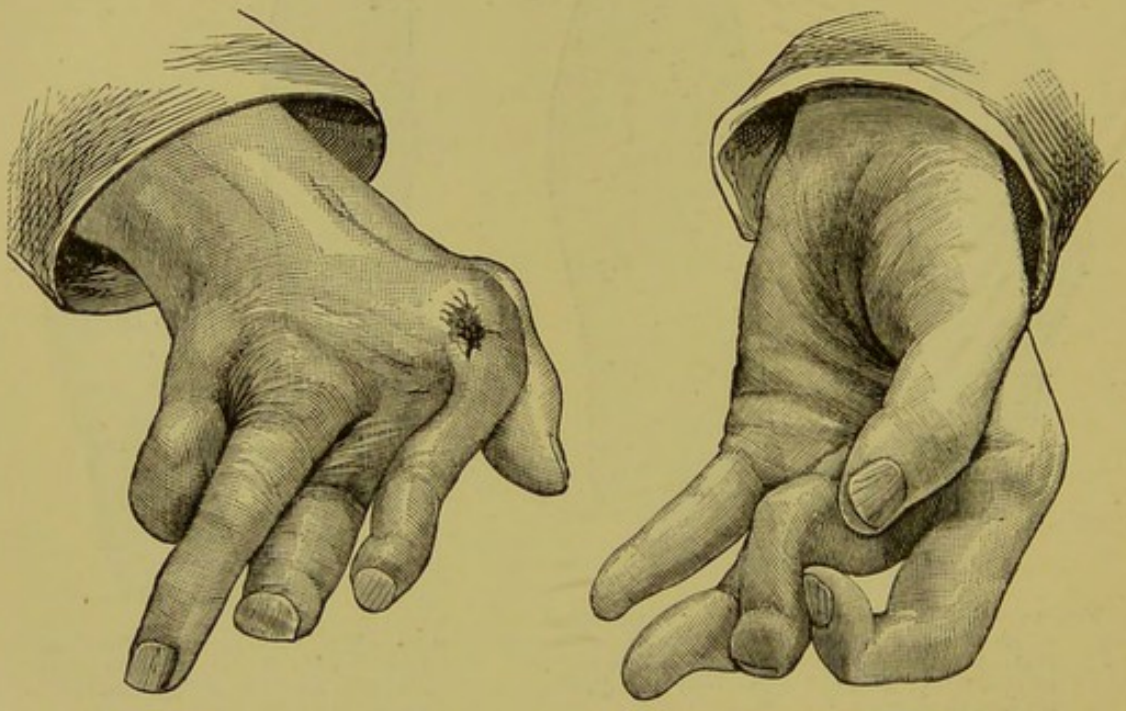


Fig. 15.—Tophaceous Gout of Hands, illustrating deflection and torsion of digits and phalanges,—"seal-fin" type.

Dr. Goodhart found very similar conditions in another case.[3] These appearances are very uncommon.

Suppurative Arthritis in Gout.—Pus is rarely found. Amongst the cardinal distinctions of gouty arthritis are its aseptic course and the absence of suppurative tendency. In bursal sacs impregnated with urates, inflammatory changes going on to formation of pus may be occasionally met with. The bursa over the olecranon is the most frequent site for this, and here it may perhaps be induced by injuries. Scudamore[4] met with four cases

[1] Path. Soc. Trans., vol. xxvi. p. 162, 1875.

[2] Dr. Barlow has related to me the case of a young man who had hæmophilia, with epistaxis, hæmaturia, and effusions into the joints, and, subsequently, uratic tophi in the ears.

[3] Path. Soc. Trans., vol. xxvi. p. 164.

[4] *Op. cit.*, p. 146.

of suppuration as the termination of gouty inflammation, the result being modified in each case by an attendant secretion of urates. Norman Moore [1] found puriform fluid in a gouty knee-joint in a man æt. forty-six. Mr. Stephen Paget has related a case of purulent gonarthritis in a man æt. forty-nine, long gouty, and quotes another example, recorded by Mr. Rivington, of suppuration involving the wrist-joint in a man æt. sixty-two.[2] Mr. Hutchinson has met with suppuration in the great toe-joint, with uratic deposit. Sometimes a circum-articular abscess breaks into a joint, septical matters being thus introduced from without.

Gangrene.—Gouty inflammation has been known to pass into gangrene, with sloughing of the integuments about the great-toe, and without associated glycosuria. Gangrene is not infrequent as a result of acute gout, or of injury to peripheral parts in the subjects of chronic gouty glycosuria. Gangrenous gouty inflammation may result from enfeebled powers in the aged, and in gouty cachectic subjects. Dr. Quain has related to me an instance of this kind which proved fatal in an elderly gouty subject.

Bursal Cysts.—Over the nodules, and also just above the nail, at the last phalangeal joints, may sometimes be found small cystic swellings of the integument. These have been likened rather aptly to crabs' eyes. I have hitherto only met with them in women in middle or advanced life. Paget and Garrod have described these. I believe they are due to small and, perhaps, adventitious synovial bursæ. Sometimes they burst, and there issues from them a clear, viscid fluid, in which I have not detected uratic salts. When they are tumid there is some pain and heat in them, and pressure yields a delicate crunching sensation to the finger, such as is found in ordinary inflamed bursæ. I have never observed these in any but Heberden's nodes of gouty origin, and only rarely in these. Their appearance is depicted in Fig. 13. In time the contents become dry, and the nodule hardens. They may subside for months, and reform as before.

Dupuytren's Contraction.—Contraction of the palmar fascia, known as Dupuytren's contraction, is more apt to occur in gouty than in other persons. If pressure alone were the cause of this, we should more frequently meet with it. Pressure is the exciting cause, but is mainly potential in the gouty or arthritic habit of body.[3] It is not usual to find other overt gouty changes in these

[1] Path. Soc. Trans., vol. xxxiii. p. 274.

[2] Clin. Soc. Trans., vol. xx. 1887, p. 232.

[3] Vide *De la rétraction spontanée et progressive des doigts dans ses rapports avec la goutte et le rhumatisme goutteux.* Par A. L. Menjaud, Thèse, Paris, 1861.

subjects, and hence it is to be regarded as one of the manifestations of incomplete gout. It is often hereditary. The plantar fascia is less often affected in this manner. The integuments become adherent to the fascia, and thus puckered, because blended with it in parts. The sheaths of the flexor tendons are also involved, but the tendons and joints are not implicated.

Uratic deposits attain at times enormous size. The largest are invariably around some joint, and the upper extremities furnish the most marked examples. (*Vide* Fig. 16.) Section of these

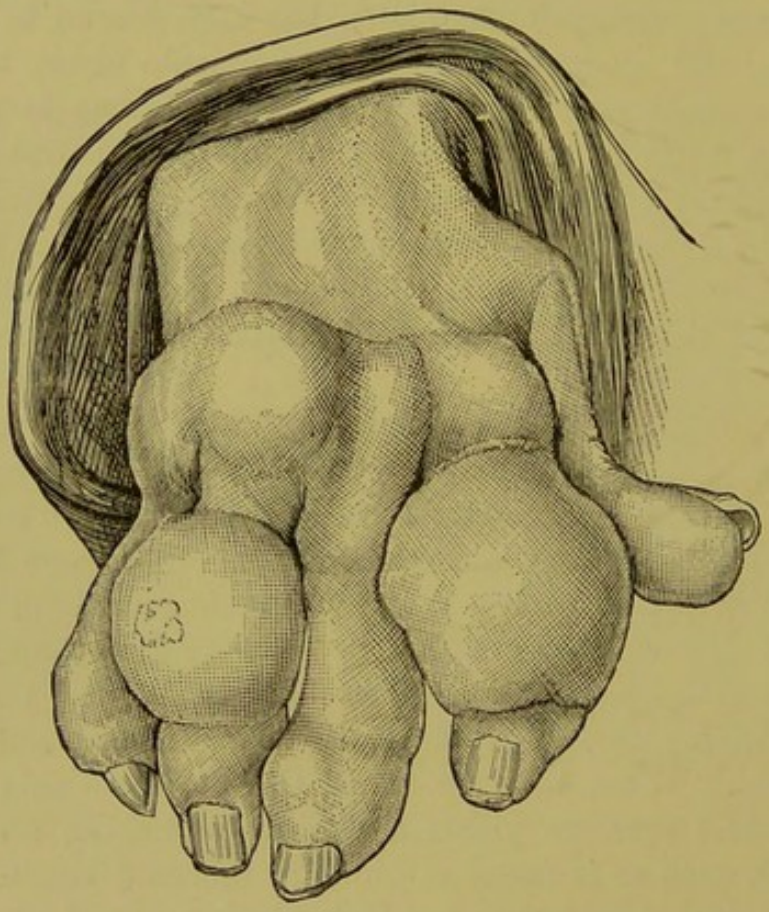


Fig. 16.—Tophaceous Gout. Both hands were symmetrically affected Man, æt. 60. Enormous tophi.

tumours shows that many of the changes induced are due to long-continued mechanical pressure, leading to absorption of much normal texture. As these deposits approach the surface, they tend occasionally to burst through the skin, and great relief to all the troubles engendered is afforded by a flow of pultaceous cretiform urates, and the deformities greatly subside. In this fashion many ounces of urates are got rid of, and patients sometimes bring with them parcels of these collections.

II.—Abarticular and Visceral Gout.

Deposits in internal organs are of extreme rarity. Cases have been described which were supposed to illustrate this in the cardiac valves and inner tunic of the aorta, in the bronchial glands and tubes, and the meninges of the brain. Few of these observations have been made since the chemical reactions of uric acid have been better understood. In some of these instances only slight traces of the acid were found, associated with lime salts commonly met with in the situations mentioned. Lancéreaux, Bence Jones, Dr. Sansom, and Dr. Sydney Coupland have found urates in concretions of the mitral and aortic cardiac valves, and so has Bramson in plates from the arch of the aorta.

Respiratory System.—Crystals of uric acid have been detected in the sputa of a gouty patient by Dr. J. W. Moore of Dublin,[1] and by Lecorché.[2] Garrod met with encrustation of the arytænoid cartilages in one case. This must be very rare. Virchow detected a "tophulus" in the posterior part of the right vocal chord.[3] Litten, in an account of *post-mortem* appearances in a very gouty man, aged forty-one, describes uratic deposits in the crico-arytænoid ligaments, which extended in the form of broad white stripes between the articulating surfaces of both the cartilages, and nearly filled up the joints.[4] Dr. Norman Moore has reported a case of saturnine gout in which small deposits were found in both vocal chords, none being found in the crico-arytænoid joints.[5] In this case there was deposit in the pia mater over the anterior cerebral lobe, a half by a quarter of an inch in extent, the dura mater not being adherent to it.

Patches of congestion are sometimes found on the vocal chords during life with symptoms of catarrh, and are met with in gouty persons without any overt exposure to ordinary causes of catarrh.

Bronchitis.—There are found signs of irritation, such as chronic congestion of the bronchial mucous membrane, in cases of gouty cachexia. Laryngitis of gouty origin is rare, but not unknown.

Bronchitis is very common after middle-life in gouty persons. It may alternate with articular gout, or with some skin-affection, as psoriasis or eczema. With this is associated hypertrophous emphysema, resulting mechanically from cough, its primary cause being degeneration of the small vessels and capillaries, which

[1] Irish Hosp. Gazette, July 15, 1873.

[2] *Op. cit.*, p. 319.

[3] Archiv, vol. xliv.

[4] Virchow's Archiv, vol. lxii. p. 132, 1876. (Dr. Semon has kindly given me this reference.)

[5] Path. Soc. Trans., vol. xxxiii.

become obliterated and fatty, together with the epithelium of the alveoli, thus leading to atrophy and rupture of the walls of the latter.

Emphysema.—According to Dr. Norman Moore, emphysema is as constant a lesion in the gouty as is interstitial nephritis. It is *par excellence* the pulmonary lesion of the gouty. With emphysema is associated hypertrophy of the right ventricle of the heart, which is long compensatory of the difficulties of the circulation in the pulmonary artery. As in most of these cases we have to do with chronic nephritis, it is hardly possible to determine in each how much of the existent morbid anatomy is dependent on the renal mischief alone. The occurrence of uric acid in the sputa of a gouty bronchitic patient has been already alluded to.

Pneumonia (Arthritic Pneumonia).—Gout is known to cause pneumonia. Some authors deny this, but there is no room to doubt the fact. The cases occur in persons of gouty habit, and are not always preceded by arthritis. Cold is, perhaps, the commonest exciting cause. The pneumonia is lobular, but may be patchy and "*ambulans*," affecting both lungs. It does not form part of the arthritic attack after the manner of a rheumatic pneumonia. The physical signs are such as are usually present, and the sputa are often rusty, and even bloody in the aged and exhausted. Several attacks may occur at long intervals, be recovered from, and leave the lungs unimpaired. Herpes labialis is not uncommon in these cases. The pneumonic symptoms may be rapidly relieved by onset of articular gout.

Catarrhal pneumonia is sometimes met with.

Congestion with œdema is found at the bases of the lungs in cases of cardiac dilatation and failure.

Embolic Pneumonia.—Embolic pneumonia results in cases of gouty phlebitis.[1] It may prove fatal, or be recovered from. The base of the lung is most commonly involved. The sputa are mucous and very bloody, like currant jelly. Branches of the pulmonary artery may be plugged first in one and then, later, in the other lung. The emboli may soften and become puriform, and may thus become sources of general septical infection.

This form of pneumonia may also be patchy, and involve both lungs.

Pleuræ.—Pleural effusion I believe to be infrequent in the earlier stages of gout, and I can hardly support Fraentzel's statement that attacks of pleurisy are common in gout.[2] Garrod

[1] *Vide* Cases reported by Tuckwell, St. Barth. Hosp. Reports, vol. x., 1874.

[2] Ziemssen's Cyclopædia, vol. iv. p. 597.

mentions the occurrence of a species of dry pleurisy, which may also attack the diaphragm, causing violent spasmodic cough. Charcot considers that these are probably cases of simple pleurodynia. In examining the bodies of those who have died from gouty cachexia with degenerate hearts and kidneys, effusions are not infrequently found in the pleuræ. Garrod found uric acid in pleural effusions. Pleuritic adhesions are somewhat commonly met with in autopsies of the gouty. Ebstein records the case of a man, aged sixty-three, the subject of articular gout, who suffered from intense bronchitis with emphysema. After a sharp fit in a foot and a hand lasting five days, violent pain came on in the left pleura with a moderately large effusion, proving fatal in two days. At the autopsy there was found interstitial (uratic) nephritis, and hypertrophy of the bladder and prostate, the latter suppurating, and evidently the cause of pyæmia. There were hæmorrhagic pleural effusion, abscesses in the left lung, and commencing suppurative pericarditis. The left ventricle of the heart was hypertrophied. Uratic deposits were present in both great-toe-joints.

Digestive System—Tongue.—The tongue presents no noteworthy objective features. But the gouty may suffer from deep-seated pain in the tongue, which, as Paget has declared, may sometimes cause apprehension of cancer. This neuralgia seldom lasts more than a day or two, and is more often met with in cases of irregular and incomplete gout. A peculiar sense of heat and burning is sometimes experienced.[1]

Psoriasis of the tongue is apt to occur in the gouty. It may vary in extent from a small patch to one covering the greater part of the organ. In colour these may be bluish, and, if thin, are shiny and glistening, with the "snail-track" character. Thicker patches are white and rough, and are termed leucoplakia. They have been well-described and depicted by my colleague, Mr. Butlin.[2] Excessive smoking is probably the most frequent cause. According to Paget, the hard palate is sometimes thus affected, but not, as is the case in syphilitic psoriasis, the buccal membrane or lips. In cases of acute and chronic gout, according to Dickinson,[3] the tongue presents, as to furring or coating, a stippled character, with whitish dots, or a partial coat, being generally moist.

[1] *Vide* Art. "Tongue," Quain's Dictionary of Medicine, 1882, p. 1638.

[2] Diseases of the Tongue, 1885, p. 126.

[3] Lumleian Lectures, Roy. Coll Phys., "The Tongue as an Indication in Disease," 1888, p. 36.

Throat and Pharynx.—The gouty throat is like no other. The pillars of the fauces, especially the posterior pair, the velum, and the uvula, are very red and glazed. They appear as if freshly brushed over with glycerine. Some dilated venules may often be seen coursing over parts of the membrane. The uvula is greatly enlarged and elongated, sometimes seeming to fill up the gap between the pillars. It has often an œdematous border, or edging, and tip. Sometimes it is so big that the condition of the pharynx can hardly be observed. The surface of the latter is not so smooth as that of the fauces. It is coarse, with red, glairy prominences upon it, and depressions here and there covered with greyish, slightly adherent patches of mucus, and it has sometimes enlarged venules upon it. In elderly people the redness is less marked in some instances, but the large uvula and glairy membranes are readily recognized.

A case of granular pharyngitis has been reported by N. Gueneau de Mussy,[1] in which masses of concretion, consisting of carbonate and urate of lime, were discharged several times daily. These issued from mucous follicles which presented white points.

Angina tonsillaris, very painful, but not suppurating, may, in the gouty, suddenly yield to an acute articular attack.[2]

Parotid Glands.—Gouty parotitis is occasionally met with. Garrod, Rotureau, of Paris, Teissier, of Lyons, and Debout D'Estrées,[3] of Contrexéville, have noted unequivocal cases. Metastasis to the testis has not been observed in this association.

Sulpho-cyanide of potassium has been found in excess in the saliva by Dr. Fenwick in gouty patients, especially before an acute attack; also in patients suffering from "bilious headaches" who belonged to arthritic families.[4]

Symptoms referable to the œsophagus will be described elsewhere.

Stomach.—Not much is known as to morbid anatomy in gout of this organ. Deposits of urates are unknown in its mucous or other coats. Congestion and catarrhal states are found in cases where renal disease exists, and may fairly be referred to as concomitants of chronic nephritis, and not directly gouty. Gout in the stomach is often mentioned, but not often observed. The attack is sometimes determined by irritating food. Doubtless, angina pectoris in the gouty has been mistaken at times for gastric pain.

[1] L'Union Méd., No. xviii., 1856. (No mention of "gout" occurs in this report.)
[2] Case recorded by Sir H. Halford, *op. cit.*, p. 108.
[3] Med. Chir. Trans. Lond., vol. lxx., 1887, p. 217.
[4] Med. Chir. Trans. Lond., vol. lxv., 1882, p. 127.

The most trustworthy case is one reported by Moxon,[1] in which erosion of the mucosa was found with submucous hæmorrhages and adherent pellicles of lymph, and I have knowledge of another in which the symptoms were severe, with hæmatemesis probably arising from erosion. The patient, æt. forty-two, a medical man, had had regular gout, inherited from his father. In this case M. Charcot concurred in the diagnosis.[2] Many functional gastric disturbances occur in the gouty, such as gastralgia, vomiting, sometimes incoercible, and pyrosis. These usually give way to articular symptoms, and leave nothing for the morbid anatomist to discover. Great flatulent distension may occur, with intense pain, and this, together with collapse, may constitute the leading symptoms in gout of the stomach.

Pancreas.—No noteworthy changes have been observed in this organ. Cancer is sometimes present in it in the gouty.

Intestines.—Professor Hayem has recorded a case where enteritis occurred, the villi being strewn with small uratic encrustations. In other cases the enteritic symptoms have appeared to be dependent on associated nephritis.

Severe colic, tympanitic distension, enteralgia, and diarrhœa are sometimes distinctly referable to gouty disorder ; but little is known in respect of the morbid anatomy of such cases, since they rarely prove fatal.

Hæmorrhoids.—In the rectum hæmorrhoids are common, as the result of portal venous congestion and constipation.

Cutaneous System.—The skin is not uniformly affected in persons of gouty habit. Cullen says : " Gout attacks especially men of robust and large bodies, men of large heads, of full and corpulent habit, and men whose skins are covered with a thicker *rete mucosum*, which gives a coarser surface." In advanced cases it is common to meet with a peculiar soft and satiny state of skin, with smoothness as if the integument had been oiled. This is very noteworthy in the cases of even labouring men. The crippling associated with the fixation and pain in the joints has, however, usually prevented any laborious pursuit for some time. This smoothness is found especially marked on the limbs. The facial integuments may be coarser. The capillaries are injected in persons of the vascular arthritic type. They fill slowly when emptied. The nasal integument may become thick, coarse, and

[1] Trans. Path. Soc., 1870.

[2] In the Hunterian Museum there is a preparation of a gouty stomach thus described :—" A specimen of a portion of the œsophagus and stomach of a person who died suddenly of gout in his stomach. There was considerable inflammation, even in some places to the extravasation of blood."

unctuous, and also unduly vascular, with enlarged ramifying venules, especially, but not always, in tipplers. These appearances have been regarded as typical of the meat-eating, beer-drinking Englishman, the classical subject of gout. I found three tophi in the skin of the nose in a man aged sixty-two. There were none on the ears. But gout attaches itself to almost all diatheses, and in the pale, sallow, or "bilious" subject (olive-complexioned arthritic) (*Laycock*), with little-marked integumentary vascularity, these changes (gouty cachectic) do not so proceed. Painful follicular inflammations in the alæ of the nose have been noted with some frequency in the gouty. These do not suppurate, but are apt to recur again and again. Skin-diseases due to gout will be subsequently treated of. The ears are often large, and present undue hardness in the cartilages, sometimes in the form of plates of almost bony consistence.[1] Tophi may or may not be associated with these.[2]

According to my statistics, in one third (49 in 150) of all well-marked cases of gout, the ears present tophi on the helix, antihelix and its fossa, and the lobule. In many cases where deposits exist in joints, there may be entire absence of tophi in the ears or other peripheral parts. With less frequency deposit is found in the skin away from joints, and here almost indiscriminately. Thus, tophi have been found in the alæ of the nose, in the integument of the trunk, perineum, and penis, over the ulna and tibia, and are common over the olecranon and patella. The palmar integument and pulps of the fingers are not infrequent sites.

These tophi sometimes burst and discharge, or may be picked out, especially from the ears, in small chalky masses. In one case they disappeared from the ears, and had not returned after three years, the patient having given up beer in the meantime. Tophi are very rare in the ears of women. I demonstrated an instance of this in a series of gouty cases at the International Medical Congress in London in 1881.[3] In a remarkable case under my care in 1888, where tophi were most widely diffused, they occurred in streaks on the eyelids, much resembling xanthoma.[4]

[1] To these plates I would apply the term *porosis*, which comes down from Galen in connection with gout.

[2] *Vide* a case recorded by me in Clin. Soc. Trans., vol. xvi. p. 258, 1883. Dr. Barlow has made two similar observations.

[3] Transactions, 1881. Report on Congress Museum, p. 124.

[4] "In mercatore podagrico diu et misere afflicto ex toto corpore, per poros, adeo ut etiam palpebræ oculorum non exemptæ fuerint, ejusmodi materiam gypseam, circa poros cutis mox in tophos mutatam prodiisse observavit."—*F. Plater, Prax. Med.* tom. ii. p. 598.

These were chemically tested, and proved to be uratic (*vide* Plate, fig. 1). When they are seen on the face and ears, they have sometimes to be distinguished from milium and small sebaceous cysts. Tophi have been found in the sclerotic tissue of the eyeball. They tend to produce irritation of the skin, which becomes purplish, thin, transparent, and glossy, and, lastly, ulcerates. A fungous base is seen with uratic discharge. Such ulcers occur on the hands, feet, and legs. In the latter situation they may be complicated with varix, and are apt to be painful.

Sub-acute gout sometimes occurs in the ears, and I believe the indurations of the cartilage I have noted to be the result of such attacks. Laycock described the lobule of the ears in persons of the gouty habit as "soldered," that is, not pendulous. It is often plump and vascular,[1] and tophi may occur in it.

Hair.—The hair is thick in early life, but often tends to grow

1 *Physiognomy of the Goutily Disposed.*—Taking the principles as laid down by Laycock, the peculiarities of those thus affected fall under the head of the sanguine arthritic diathesis. (That careful observer did not fail to note the modifying influences of gout upon struma and other cachexia.) Thus may be compared the physiognomy of the diathesis and its associated cachexia (developed in time):—

Blood-vessels numerous; heart large and powerful; blood-corpuscles numerous; skin over malar bones highly vascular (florid complexion); skin fair, firm, oleaginous, perspirable; eyes blue; hair thick, not falling easily; teeth massive, well-enamelled, regular, even, undecayed in advanced life; malar bones flattened; head symmetrical; nasal bones well-formed, nose aquiline or of mixed form; lower jaw massive; lips symmetrical.

Form.—Figure for the most part tall; thorax broad at the summit; ribs well-curved; abdomen full; muscles firm, large; limbs large, robust; gait erect, well-poised. *Nutrition* active; digestion vigorous; appetite great for animal food and alcoholic stimuli. *Respiration* deliberate, deep; circulation vigorous; animal heat abundant; locomotion active; aptitude for exercise and outdoor amusements. *Reproductive* powers active; innervation abundant; the mental powers vigorous and enduring.

Physiognomy of the Sanguine Gouty Cachexia.—Blood-vessels largely developed over the malar bones and varicose; skin oily, yellow from subcutaneous deposit of fat; hair thick and white; teeth numerous, discoloured, crusted with tartar; lips bluish, nose reddish, hypertrophied; arcus senilis; abdomen pendulous; limbs thick; joints nodose; nodosities on the ends of the fingers, lobes of ears, fascia of muscles, and tendons; respiration hurried, wheezing; pulse intermittent, irregular; stomach flatulent; digestion acid; urine loaded with lithates; temper irritable; mind sometimes enfeebled.

The local diseases of the arthritic cachexia are principally seen in adult males past the age of forty-five. They consist especially in chronic inflammation of the muscular and articular tissues; in calcification of the basilar and coronary arteries, and of the cardiac valves. These changes give rise to hæmorrhagic apoplexy, angina pectoris, cardiac hypertrophy and dilatation; and to secondary pulmonary affections, as emphysema, pulmonary apoplexy, and asthma. Irritation of the mucous surfaces may give rise to nephritis, pharyngeal and laryngeal coughs, and diarrhœa.*

* Med. Observation and Research, 2nd edit., pp. 96–98.

thin on the vertex before the third decade, and may leave a shiny poll with a well-defined ring of hair below. In some cases there is early greyness in the goutily disposed, and abundant grey hair may be maintained through life. In others the hair retains luxuriance and good colour to the seventh or eighth decade, even with profound articular disease and deposits. These facts are founded on many careful observations, hence, no special type can be described in respect to the nutrition of the hair. The marked divergence must be explained by factors, personal or inherited, in each case. Greyness goes with general tissue-failure, as a rule, but this is found to vary much even in the same family, and it must be borne in mind that in some persons certain textures tend to decay, and die out sooner than others. This is equally true of the teeth, and yet in each case longevity may occur.

The composition of tophaceous matter or "cutaneous gravel," as Trousseau termed it,[1] has been found to vary in respect of the sodium and calcium salts of uric acid, according to the site whence it is taken, but about fifty per cent. of it is uratic, and sodium chloride is present to the extent of ten per cent. Calcium phosphate and animal matter make up the remainder. The consistency varies, according to its age, from that of cream to that of ordinary chalk. Sir A. Garrod gives me his experience to the effect that tophi consist essentially of crystallized sodium urate, which can be dissolved in distilled water and re-crystallized, and he believes that lime is only an accidental admixture. In cardiac valvular concretions occurring in the gouty, there appears to be only slight impregnation of lime-salts with urates.

Sir Andrew Clark has told me of a case in which analysis of tophus-matter yielded a large amount of calcium oxalate, and he believes that calcium urate in spiculated crystals is common as a deposit. A specimen of tophus from the patellar bursa of one of my patients was kindly examined by my colleague, Dr. Russell, in the Laboratory at St. Bartholomew's Hospital, and was found to consist essentially of sodium urate with a mere trace of calcium salts.

The skin is usually active in respect of sweating, though when exercise is lessened from any cause, and in cold weather, it may be very inactive. It has been maintained that urates do not pass off from the sweat-glands. Garrod has emphatically declared this, but Drs. Meldon and Tichborne, of Dublin, are equally confident in affirming the contrary. The latter has indicated the best method for detecting uratic salts in sweat.[2] I have had

[1] "Tartar of the blood."—*Sydenham.*

[2] Brit. Med. Journal, November 19, 1887, p. 1097.

several experiments made to determine this question, so far without any success. The relief afforded in gouty cases by regular use of Turkish baths is attributed to the free action of the skin. These are probably effective in much the same way as is regular muscular exercise, and, in great measure, they may replace this, especially if followed up by shampooing.

Golding Bird found uric acid in the contents of the vesicles of gouty eczema, and James Begbie recorded an instance of pemphigus in which uric acid was found in the fluid of the bullæ.

The skin-diseases to which the gouty are especially obnoxious are eczema and psoriasis, and these will be discussed elsewhere.

The bursæ over the joints, especially of the knuckles and phalanges, are apt to be loose and enlarged, and are recognized sites for uratic deposits. That over the olecranon is sometimes distended to the size of a large orange, and nodules of urates can be felt in it, as well as increase of synovia.

Nails.—The nails are observed to be coarse and fibrous, striated and fluted, or lined vertically. *Vide* Figs. 13–16. This peculiarity is well-marked in most persons of the arthritic diathesis, and hence is found in rheumatic persons. The nail-substance is apt to grow thick and brittle, and especially so after attacks of gout. The nails may be shed after severe local attacks.

The transverse depressions, described by M. Beau and by Dr. Wilks, are seen in due time after gouty as after other illnesses, indicating a temporary failure and depression of nutrition of the whole body. As an entire nail takes six months to grow, the site of these furrows indicates the date of the past illness with singular exactness. Sometimes a white line marks the attack.

Teeth.—The teeth are especially noteworthy in persons of gouty habit. They are, as a rule, well developed, with strong and hard enamel which is rather yellow in colour. They resist decay and are firmly set in their alveoli. In time they become more or less worn down, so that the pulp-cavities begin to be visible.[1]

Buck-teeth are not uncommon, one of the lower central incisors being thrust out of rank, as first noticed by Laycock. I have observed these characters in a large number of cases, and am confirmed as to their correctness by dental surgeons.[2]

[1] Pye-Smith quotes the authority of Mr. Moore for the facts that this condition may be caused by that formation of jaws which gives an edge-bite, also in cases where the enamel is thin on the summits of the teeth.—*Fagge, Prin. and Pract. of Med.*, 2nd edit., p. 490.

[2] *Vide* "The Characters of the Teeth in Persons of the Arthritic Diathesis," a paper read by myself before the Odontological Society of Great Britain, in Soc. Journal, 1883, p. 193.

A tendency to shed sound teeth has been noted with some frequency in middle or later life in goutily disposed persons, and they are more than others liable to occasional and fugitive attacks of pain in several sound teeth at a time, with a sensation as if these were starting from their sockets, being tender to bite upon.[1]

Without doubt, many varieties may be met with in the characters I have laid down. These are explicable by the fact that other causes are at work unconnected with gouty influence, and with other commingled diathetic states are found the tendencies and results of those states. This statement holds good for all tissue-characteristics that may be affirmed of the gouty. I am only concerned here to express the prominent and typical features that may be observed, and to describe the character that pertains to the several tissues as impressed by the gouty habit of body.

My statements are chastened by the discipline entailed by long-continued and careful inquiry on these points.

It may be confidently affirmed that no uratic deposits are met with in connection with the jaws, teeth, or gums, notwithstanding contrary statements.

Tooth-grinding, as a peculiarity of the gouty, will be subsequently referred to.

Eye.—Iritis is met with in the gouty, and is believed to be a manifestation of this habit. It may be very insidious, and is prone to relapse. Gouty persons are more than others apt to suffer from glaucoma, which, as Mr. Nettleship remarks, was formerly described as "arthritic ophthalmia." This is commoner in women during pregnancy or soon after the menopause. Males thus affected are often subject to hæmorrhoids. Mr. Hutchinson [2] has described cases of hæmorrhagic retinitis.

Hæmorrhagic Retinitis.—This occurs usually in one eye, and is of sudden onset in the gouty or in persons so predisposed. It is to be differentiated from albuminuric retinitis. Small flame-shaped patches of hemorrhage are scattered abundantly over the fundus. Those which are punctate have striated margins, and none appear as blots. Haziness about the disc is observed, but no glistening, white deposits as met with in renal retinitis. The veins are large and angular from turgescence, and rendered indistinct by blood-effusion into their sheaths. The arteries are very small. Of fifteen cases noted by

[1] *Vide* cases reported by Mr. James Rymer, Jour. Brit. Dent. Assoc., August 1887, p. 499.

[2] Clin. Soc. Trans., vol. xi. p. 132, 1878.

Hutchinson, eleven occurred in men and four in women. The youngest patient was forty-four, the oldest seventy-two, and seven were over sixty years. Gout was positively present in six, and was strongly probable in four or five others. In one there was saturnine gout, with much albuminuria; and in another there was no gouty history, but diabetes existed, which was probably the cause of retinitis. In ten cases only one eye was affected, and in five both. In about one-third of the cases there was slight albuminuria present occasionally. There was no dropsy.

As Mr. Hutchinson observes, it is hardly justifiable to separate this group of cases abruptly from other forms of retinitis associated with renal disease and diabetes, since kidney-disease is so often a concomitant in gouty cases. The main points to be noted are the unilateral character, the left eye being most often affected, the very numerous flame-shaped hæmorrhages, and the absence of white deposits. The extravasations may recur for a long period.

The influence of the cardio-vascular system must be considered in relation to the occurrence of retinitis. Hæmorrhages may arise under the influence of strain, as in stooping or coughing. Mr. Hutchinson is of opinion that this form of retinitis is due rather to venous obstruction, such as thrombosis of the retinal vein, than to arterial disease, the former being a recognized gouty lesion, and better explanatory of the suddenness of the attacks than a theory of arterial embolism or aneurysm.

I am inclined to regard these hæmorrhages as being sometimes akin to the subconjunctival bleedings, epistaxis, and other leakages from small vessels which are very apt to occur in those of gouty habit.

Cases of insidious irido-cyclitis, leading to secondary glaucoma, have been noted by Mr. Hutchinson as sometimes occurring in the members of gouty families during early adult life.

Conjunctivitis, episcleritis, and sclerotitis are recognized as of gouty origin. Garrod found uratic deposit in two cases on the conjunctivæ.

Glaucoma.—Mr. Brudenell Carter has noted the tendency of iritis or keratitis in arthritic cases to spread to the anterior sclerotic (vascular) zone around the cornea. He is of opinion that a large number of examples of supposed gouty or rheumatic ophthalmia are nothing more than cases of subacute or remittent forms of glaucoma, the pain being tensive and not specific.

The point to be noted, however, is the tendency for those

arthritically disposed to suffer from this special class of ailment with greater frequency than those not thus impressed.

Optic Neuritis.—There are no proofs that optic neuritis is ever of gouty origin. Mr. Hutchinson, however, believes that he has met with cases.

A case of destructive inflammation of the eyeball in a gouty man is mentioned by Mr. Stephen Paget as having occurred in Mr. George Critchett's practice.[1] There had been history of gout for twenty-three years. At last he had inflammation of the right eye, which recurred more than once; then came acute destructive inflammation, with suppuration of the globe, the sclerotic gave way, the lens escaped, and the globe collapsed.

Blocking of the duct of a Meibomian gland leading to a projection on the inner aspect of the tarsal cartilage, and thus irritating the conjunctiva, is alleged by Brudenell Carter to occur most often in the gouty, the retained secretion being rendered more irritant by chalky deposit.[2]

Lymphatic System.—The lymphatic system has been held to be free from any changes in gout. The glandular portion cannot be said to be involved, but there is clinical evidence of sub-acute gouty inflammation of lymph-spaces in certain regions, due to uratic deposit and influence. Dr. Buzzard has called attention to this.[3]

Spleen.—The spleen cannot be said to be specifically affected. In many cases it is enlarged and hard in texture, and may reach from 15 to 22 ounces in weight. Sometimes it is found to be soft. Infarctions are met with occasionally, and adhesions may exist between it and the stomach and diaphragm. The capsule is not infrequently thickened. Most of these changes are not improbably in relation with the associated cardiac and hepatic conditions.

Adrenal Bodies.—No changes have been noted in the suprarenal glands.

Ear.—But little has been recorded respecting the morbid anatomy of the ear in gout. The auditory meatus is sometimes found red, glazed, and as if recovering from eczema. According to Hinton, "a peculiar irritation of the meatus with dull redness, swelling, and watery discharge resisting local remedies are very characteristic of gout." Mr. Hutchinson prefers the term seborrhœa

[1] Clin. Soc. Trans., vol. xx. p. 234, 1887.

[2] Holmes' Surgery, p. 692. So far as I am aware, this has not been proved chemically.

[3] Diseases of Nervous System, p. 69, 1882.

"The fasciæ and other fibrous structures are nothing but lymphatic pumps, pumping up the waste material from the muscles and sending it on into the lymphatic trunks."—*L. Brunton, Disorders of Digestion*, p. 231, 1886.

for these cases.[1] In a chronic form this may last for many years, leading to hypertrophy, and so to stenosis of the canal, admitting barely a small eye-probe.

Sir William Dalby has kindly reported to me instances of this kind. Attacks of acute gout may occur in the external canal, yielding to specific treatment. This form, according to Dalby, does not involve the membrane or tympanic cavity, but the swelling may be so great as nearly to occlude the canal. The auricle is sometimes thus affected.

Tophi have already been referred to as occurring in the pinna. More rarely they occur on its posterior aspect. If the ear be held out against sunlight or a beam of lamp-light, the distribution of the deposits is well seen. The large, so-called, calcareous deposits in the tympanic membrane often seen, can in no way, so Dalby believes, be connected with gout. They occur sometimes in children, and are commonly cicatricial results of perforation.

Multiple hyperostosis of the canal has been attributed to gout, but, according to Dalby, without sufficient reason. Adhesive changes and thickenings of the ossicula, when met with, can only be regarded as results of inflammatory processes, and not as certainly gouty in most cases. Uratic deposits have not been found on the ossicula. I have already noted the liability to induration of the cartilage of the auricle in gouty men, and the occurrence of flattened nodular masses in them, which I term porosis.

It is probable that some forms of senile deafness are attributable to gouty changes in the ossicula.

Muscles.—There is no recognized morbid change in the muscular system. Uric acid has been found in the muscles. Tendons become involved in association with the joints, uratically encrusted and stiffened.[2] The muscles are the sites of painful gouty attacks and of subjective symptoms in the gouty. These will be elsewhere described. That much so-called "muscular rheumatism" is really gouty, I feel sure.

Nervous System.—In respect of the nervous system, it may be affirmed that there is, as yet, no morbid anatomy in the gouty. The changes due to cachexia in long-standing cases are not now in question. Clinically, there is good reason to believe that gout may induce neuritis in almost any nerve-trunk with motor,

[1] Med. Press and Circular, January 25, 1888, p. 77.

[2] Rupture of the tendo Achillis is stated by Gairdner to occur chiefly in persons of gouty habit. It is an accident of advanced life. *Op. cit.*, p. 26.

sensory, and vaso-motor symptoms. Dr. Buzzard has directed attention to the manner in which this is probably set up through the lymph-spaces in connection with the nerve-bundles wherein uratic deposits may form. Neuralgia is especially common in the gouty. Cases of sciatica, and affections of the circumflex, median, and portio dura are now well-recognized, and have been referred to gout by Todd.[1] Dr. Buzzard[2] relates a case of pseudo-infantile paralysis due to neuritis. Dr. Ormerod has recorded cases of tingling and numbness in the arms in connection with gout and knobby joints.[3] I have seen many such. Perineuritis is probably the lesion, and there may be much pain.

More important than these, however, are cases of gouty paraplegia, which, though rare, occur in men past middle life, sometimes suddenly as by metastasis from a joint. The paralysis may be complete and involve the sphincters. Such cases, happily, recover, and leave no subsequent changes. Occasionally, the paralysis yields suddenly to onset of arthritic gout.

It is rare to find uratic deposit in the nervous tissues or their investments. They have been detected on the cerebral meninges in a few instances, and sodium urate was found by Cornil in the cerebro-spinal fluid. On the spinal meninges deposits have been found by Albert and Ollivier. The latter has recorded an important case in a gouty man, æt. forty-five, in which fulgurant pains of locomotor ataxia were supposed to exist. There were constrictive pains round the neck, chest, and abdomen, with radiation down the limbs. Uratic deposits were found outside the spinal dura mater. A patch of whitish granulations was found on this membrane, extending from the third cervical vertebra to the sacral canal, being thickest in the mid-dorsal region and extending along the sheaths of the spinal nerves. This was only separable by tearing. The chord and other membranes were unaffected. M. Rendu, who relates this case, remarks that the dura mater is the least vascular of the meninges.

Charcot[4] found uric acid in the sub-arachnoid fluid of a gouty woman by means of Garrod's thread-test.

Graves believed that gouty degeneration of the spinal chord occurred sometimes, following neuralgia, neuritis, and perineuritis spreading centripetally. He remarked that "there is no reason why gout should not attack the spinal marrow and its investing

[1] Clin. Lectures, Nervous Diseases, pp. 69, 72.
[2] Nervous Diseases, p. 39.
[3] St. Barth. Hosp. Reports, vol. xix. 1883.
[4] *Arch. de Phys.*, 2 Ser., vol. v. p. 455, 1878.

membranes in the first instance, or in consequence of metastasis.[1] All that can now be affirmed, half a century since this was written, is that such cases are of extreme rarity, and that little has come to light in respect of their morbid anatomy.

Kidneys.—Perhaps no part of the morbid anatomy of gout is of more importance than that relating to the kidneys. A large conception of gout demands attention not merely to the changes associated with, and dependent on, uratic deposition, as has already been set forth in these pages. We have to deal clinically with other manifestations than these, and with none of more profound significance than those affecting the kidneys. I must here reaffirm my belief that, either by the influence of the gouty poison directly, or by inherent tissue-proclivities peculiar to those goutily disposed, changes occur in various organs and parts which can in no overt way be shown to be due to uratic deposits. This is, in truth, part of the pathology of gout. The constant experience of the dead-house is against such direct deposition in the majority of cases of so-called gouty kidneys. A more important question to determine in such cases is the presence of uratic deposit elsewhere in the body, and especially in some joint. It is still too common to hear small granular kidneys called "gouty" kidneys. I entertain no doubt that forms of interstitial nephritis occur which owe no dependence on gout, but I am equally convinced that the kidneys may be primarily or mainly affected in some cases with gouty disease which leads to chronic nephritis and a small, granular condition of these organs. This opinion is founded partly on clinical considerations and partly from a study of the ætiology of these cases. If it can be shown, as I think it can, that in gouty families certain members suffer from the primary articular form of the disease, while others show signs of renal change alone, apart from joint-affections, and the autopsy reveals the characteristic conditions of such nephritis as is common in chronic gout with joint-lesions, I think the case is proved.

The abiding weak point in the argument is the difficulty which arises in consequence of incomplete examination of the joints for evidence of deposit. This may perhaps be fairly conceded, because it is certain that quiet deposits may occur in joints without giving any token of their presence during life, and hence there is no history of overt gout in such instances. Again, it is equally certain that gout may occur in joints and leave behind no

[1] *Vide* two cases reported in his Clin. Med., reprint from 2nd edit. by Neligan, p. 367.

uratic deposits. An appeal to such statistics as are alone of value for this purpose gives the following results:—

Drs. Ord and Greenfield[1] found, in a series of cases examined with a view to determine the presence or absence of renal disease in association with uratic deposits, that in two-thirds of these hospital cases of gouty affection of the great toe-joint, there was a definite co-existence of contracted granular kidney, and in the remaining third there were affections of the kidney closely allied thereto. *There were at least eight, and probably nine, out of ninety-six cases of renal disease in which no uratic deposits were found in the joints. Of these, two were examples of extreme granular contraction, two of marked contracted granular, two of slightly granular, and one of mixed granular and tubal nephritis.*

Dr. Norman Moore[2] gives a table of forty-nine cases of chronic interstitial nephritis in males, and shows that uratic deposits were present in twenty-two cases. The youngest was twenty-eight years of age, the oldest sixty-six, the mean age forty-nine years. He gives another of sixteen cases in females, in which urates were present in five cases. The youngest was thirty-four and the oldest sixty-six years, the mean age fifty-one years. In nine cases of chronic parenchymatous nephritis in males, he found deposits in the joints in two cases at the ages of thirty-eight and forty years. In two cases in females he found no deposits. With respect to the former cases, he remarks that "chronic interstitial nephritis is not invariably accompanied by deposits in the articular cartilages, though usually accompanied by some traces of degeneration in some of the articular cartilages."

Dr. Pye-Smith records ten fatal cases of gout in men.[3] Excluding two cases in which malignant disease was the immediate cause of death, the mean age was about forty-eight. There was interstitial nephritis in all the cases, save the cancerous, and cerebral hæmorrhage occurred in two cases, one aged thirty-eight and the other sixty.

In sixty-nine fatal cases of granular kidney Dr. Dickinson found sixteen dependent on, or coincident with, gout.[4] The change he regards as gout of the kidney. No examination of joints is recorded.

Virchow[5] is inclined to believe that there can be a gouty nephritis without either classical gout or uratic depositions.

[1] Trans. Internat. Med. Congress, London, 1881, p. 107.
[2] *Loc. cit.*, p. 292.
[3] Guy's Hosp. Rep., 1874.
[4] Pathol. and Treat. of Albuminuria, p. 149, 1877.
[5] *Op. cit.*, p. 149.

It is certain that gout may occur in the ordinary articular form without implicating the kidneys, which may remain healthy even into advanced life. In such cases there is usually a fine constitution, great resistance and vigour of the tissues; and the progress of the disease is kept at bay and overcome by the vital organs. In primary renal gout the general health is poor, and a progressive cachexia works its special ravages, cutting life short prematurely. In frank gout of long duration the kidneys commonly undergo gradual cirrhotic changes, but exceptional cases are met with, as stated above.[1]

The changes induced are essentially chronic, insidious in origin, and not recognized till mischievously advanced. I believe that the gouty habit is alone the potent ætiological factor in a considerable proportion of all cases of interstitial nephritis, the form in which this manifestation occurs.

The influence of lead-impregnation and of alcoholic intemperance on the production of granular kidney is undoubted.[2] Lead and alcohol are both not infrequent factors in the history of many cases of gout, but these specific relationships will be subsequently discussed. It need only be stated here that there are no specific ætiological differences traceable in the morbid anatomy of granular kidney. M. Lancéreaux[3] is convinced of the identity of the changes in the kidneys found in cases of ordinary and of saturnine gout. He has described a variety of interstitial nephritis due to, or rather consecutive to, alteration in the arterial system, associated with arthritis, indistinguishable from the chronic rheumatic form of the disorder. He therefore believes that there are at least two forms of granular kidney. The latter variety is that now recognized as a part of the constitutional cachexia of arterio-capillary fibrosis, which I am disposed to believe is, in many instances, a manifestation of gouty inheritance.

To differentiate the two varieties described by Lancéreaux, it would be necessary to record the clinical symptoms of the arthritis more in detail, and to examine many of the joints in a larger number of cases than appears to have been done.

The term "gouty" has been used synonymously by some authors to signify "granular." Todd proposed, in 1846, to apply this term to such contracted conditions of kidney as were associated with a decided gouty diathesis. He gives particulars of two cases of

[1] *Berlin. klin. Wochenschrift*, No. 1, 1884.

[2] Dr. Fenwick found deficiency of sulpho-cyanide of potassium in the saliva in cases of lead-poisoning. This is the reverse of the condition found in gouty subjects. *Op. cit.*, p. 124.

[3] *Vide* Lancéreaux, Trans. Int. Med. Congress, 1881, vol. i.

true gout in which streaks of lithate of soda were found in the tubes of the cones of the kidneys.[1]

The view that the small red granular kidney is peculiar and due to gouty affection alone, is not sustained by experience. It is true that in this form the deposits of uric acid and urates are commonly found, situated either amongst the granulations on the surface, or in streaks in the pyramids. Other forms of granular kidney are, however, met with as the result of gouty influence, and hence it is not possible to pronounce that any particular variety of cirrhosed or contracted kidney is specifically significant of gouty disease.

Dr. Dickinson has shown that kidneys rendered granular by gout commonly advance to the most extreme degree of the disease, because, from the enduring nature of the cause, they have time to develop the utmost extent of granulation compatible with life.[2]

Uric acid, yellowish in colour, is found deposited in small granular or crystalline particles in the cortical and pyramidal portions of the kidneys. They are commonly scattered in the cortex, but in linear arrangement in the medulla, sometimes thickly set at, and encrusting, as it were, the apices of the papillæ. Microscopically, these deposits are found both in the tubules and in the intertubular stromal tissue. Uratic salts may be found in the same situations. The existence of such a deposit, even in granular kidneys, is not by itself certain evidence of gouty disease, and we are here reminded that the uric acid diathesis, although closely related to, is not the same condition as, the gouty.[3]

Uratic depositions are found in the pyramidal tubular system in very young children, but never, according to Klebs, in those whose lungs are unexpanded, and hence it is presumed that digestion has begun, and has been carried on under the influences of defective respiration.[4] Fagge directed attention to the fact

[1] Clin. Lect. (xii.) on Certain Diseases of the Urinary Organs, 1857.

[2] *Op. cit.*, p. 157.

[3] As Sir James Paget * has remarked, "Many children and young people, whom you cannot reasonably accuse of gout, produce large excess of lithic acid in the urine; and the lithates are the most common constituent of urinary calculi in the children of the poor, among whom one would suppose the gouty constitution most unlikely to occur. In the children of the middle and upper classes, in whom the inheritance of the lithic acid diathesis may be expected, calculus of any kind is one of the very rarest of diseases."

[4] Atlas of Illustrations of Pathology. Résumé of Renal Pathology, Prof. Greenfield, p. 12, fasc. ii. New Syd. Soc., 1879.

* *Op. cit.*, p. 377. Lond., 1879.

that uratic deposits in the kidney are commonly met with in Germany, where gout is rare. Castelnau, Garrod, Charcot, and Dickinson are of opinion that the deposit is in the matrix, but Garrod allows that it is also found in the tubules.

Cornil and Ranvier state that urates are primarily deposited in cells, which are the centres whence the free crystals spring, and which play an active part in the phenomena of simple deposit.[1]

According to Senator, amorphous uratic deposit is first found in the tubules and their epithelium, extending later into the interstitial tissue, and becoming crystallized. Greenfield states that these deposits are commonly found in the connective tissue of the cortex, and but rarely in the tubules.

There appears to be a common belief, especially on the Continent of Europe, that the contracted kidney of gout is constantly associated with, if not somewhat dependent on, uratic deposit.

My own experience is not singular in this country in indicating that such is not the case. These deposits are, in truth, somewhat rarely found in the kidneys of the gouty. If this statement is true for observations made in England, it may fairly over-ride Continental opinion drawn from far smaller fields of study.

In Dr. Moore's eighty cases, deposits were found in the pyramids in six cases, and in the tubules (specifically mentioned) in six cases. Hence, in hardly one-seventh of these, which were all instances of well-marked uratic arthritis, did tubal or interstitial deposits occur. As remarked by Ebstein, gout can only be the cause of calculous disease in those cases in which intra-tubular deposits occur. In many cases, however, calculous formation is an incomplete phase of gout, and may precede, accompany, or follow articular troubles. Sydenham's own case was an example in point. It is most frequent to find in these cases that the gravel-phase has preceded the articular one. With onset of interstitial nephritis, it is conceivable that intra-tubular deposits may be subsequently washed out by the free secretion of urine under high-pressure.

A distinction has been made between cases in which uric acid is found free as gravel, and those in which indiscriminate deposit of urates is found here as in other textures. *Intra*-tubular deposit of uric acid is held to signify gravel. *Inter*-tubular infiltration with urates is considered more distinctly gouty. Deposits certainly occur in both situations. They are infrequent in the secreting tubules of the cortex. The kidney

[1] In calcification infiltration begins in the ground-substance.

furnishes almost the solitary example of a highly vascular structure in which deposit is met with.

The morbid anatomy relates to all the component tissues. In a well-marked case, the organ is shrunken and more or less indurated. Its colour is red. The capsule is thickened and adherent. The surface is rough and granulated, and small cysts are commonly seen on it. Some of the granular eminences are yellowish or grey in colour, and are as large as mustard-seed. To this form of kidney Bright applied the term "contracted." Such kidneys are not always small, and may be larger than natural. Small kidneys thus affected weigh about 3 oz. The granulations are surrounded by depressions, and in stripping off the capsule portions of them may come away with it.[1]

On section there is often much fat in the pelvis. The pyramidal cones are not distinctly marked off from the cortex. The cortex is much wasted, it may be unevenly; and so, in parts, the pyramids may almost come to the surface. Numerous small cysts may exist in it, full of yellowish gelatinous fluid. That portion between the pyramids is less apt to waste than the superficial layer, and suffers later in time than the latter. The pyramids undergo little change, but may waste to a slight extent.

These are the naked-eye characters of the so-called "small red granular" or "gouty" kidney. But, as has been stated, large and mixed granular forms may be found dependent on gout, associated with some degree of tubular nephritis. Large vessels may be seen in the cortex with rigid walls. The arteries are usually hard, atheromatous, and narrowed in calibre.

The essential and grossest change in this form of kidney is the chronic inflammatory sclerosis, which mainly affects the interstitial matrix or supporting tissue of the organ. A round-celled granulation tissue is formed, which proceeds to increased formation of connective tissue. This is believed by Greenfield[2] to begin in the vessels and glomeruli of the peripheral portions of the interlobular arteries. These become thickened in their *intima* and sheaths, while fibro-muscular hypertrophy occurs in the middle coat. The glomeruli atrophy in parts, and their associated tubules likewise waste, owing to compression from the fibroid transformation of the connective tissue. This leads to the fibroid depressions seen on the surface, which are sometimes joined by

[1] Adherent capsules are not always met with in kidneys presenting signs of interstitial overgrowth. Sometimes the capsules strip off fairly well, or completely, when microscopic examination reveals a good deal of cirrhotic change.

[2] *Op. cit.*

newly-developed vessels from the capsule, and thus rendered deep-red by contrast with the paler and prominent granulations. This process spreads gradually but irregularly throughout the cortex. New development of connective tissue takes the place of the wasted elements, with accumulated leucocytes, and according to the extent of this is the size of the organ in any particular instance. This sclerosing process is not uniform, but is apt to occur in certain areas. The tubular system as a whole escapes primary disorder, but suffers compression secondarily in the affected areas from intertubular fibrosis, which may also lead to the formation of cysts. These are now believed to be due to distension of tubules or Malpighian capsules, resulting from complete obstruction of their lumen. The glomeruli become thickened, and their vessels dwindle into small tufts. Tracts are found, as described above, in which the tubules have been destroyed or have greatly atrophied. The sclerosing process causes the glomeruli to be drawn together in parts. The tubal epithelium is apt to accumulate and distend the tubes in places, and is often fatty.[1]

The vascular changes are primarily those of endarteritis obliterans in the larger arteries. The capillaries are destroyed with the tubules by compression.

In treating of the morbid anatomy of gout, the changes in the cardio-vascular system associated with the condition of the kidneys cannot be studied apart from the latter. These are the concomitants of interstitial nephritis, however induced. They consist essentially of hypertrophy of the left ventricle and the arterial tunics. The mechanism of these changes has been a much-debated subject. The most acceptable doctrine refers the cardiac hypertrophy to thickening and contraction of the arterial tunics as well in the kidneys as in the system at large, leading to high arterial tension throughout the body. The changes in the arteries are proved by microscopical examination, the high tension is ascertained by the finger or the sphygmograph, and both progress steadily with the progress of the chronic nephritis. The ventricular hypertrophy is entailed by excess of work in forcing the blood through obstructed vessels. It is certain that obstruction in the arterioles can produce high arterial tension, and this may suffice to induce ventricular hypertrophy. But in chronic sclerosing nephritis there is probably an additional source of obstruction due to laborious circulation, in the capillaries, of blood, which is

[1] For many points in the foregoing description I am under obligation to the able *résumé* by Professor Greenfield already referred to.

rendered impure by defective renal function. Some of these impurities are believed to stimulate directly the muscular walls of the heart and arteries, and so to lead to hypertrophy. It thus appears that the renal lesion is sufficient to account for the associated cardio-vascular changes.

The views of Gull and Sutton respecting the systemic degeneration termed by them "arterio-capillary fibrosis," are not, in my opinion, displaced by the explanation just given. This disorder is conceived to be widespread, and the kidneys but take a part in it. The series of changes already described cannot be controverted. They will probably retain their place in pathology, and Dr. George Johnson's name will ever be honourably associated as the earliest interpreter of them. I believe in the existence of a systemic arterio-capillary fibrosis as a definite tissue-lesion, and some of the forms of granular kidney are ætiologically related to it. How far gouty disease takes part, if at all, in it, I am not now prepared to state; but such a view deserves consideration in determining the outcome and transformations of the gouty habit as modified by inheritance, and by other conditions.

Dr. Mahomed described a form of chronic Bright's disease without albuminuria, the kidneys being red and granular, and the changes being chiefly vascular, including thickened arteries, glomeruli, and fibro-hyaline intertubular thickening. Cardio-vascular changes with high arterial pressure were present. He collected sixty-one cases. Of these, six were associated with gout and accompanied with great cardiac hypertrophy, high arterial tension, and non-albuminous urine.[1]

Lardaceous disease is very rarely associated with the nephritis due to gout, perhaps hardly in two per cent. of all cases. The inhibitory effect of gout on struma may perhaps account for this.

Changes in the Heart, Arteries, and Veins.

The condition of the heart varies according to the stage of gouty disease present when death occurs. Degeneration is most frequent in the later periods, when gouty cachexia has supervened. The changes relate to the pericardium, muscular walls, valves, and great vessels.

The Pericardium.—It is difficult to determine the direct relation, if such exist, between gouty and ordinary pericarditis in the subjects of interstitial nephritis, in whom alone it practically

1 Trans. Int. Med. Congress, 1881, vol. i.

occurs. James Begbie recorded the case of a lady, æt. twenty-seven, who was gouty, and came of gouty family, who died of pericarditis, and he stated that he had known at least two other instances.[1] I have not met with an example.

In the cachexia of gout with granular kidneys, pericarditis may occur not infrequently, and is usually fatal. In sixteen of sixty-eight cases of granular kidneys from all causes, Dr. Dickinson met with recent pericarditis, and if false membrane and adhesions had been reckoned in, the number would have been far larger. Dr. Norman Moore found signs of pericarditis—effusion, lymph, and adhesions—in twelve out of eighty cases of true gout, in all associated with granular kidney. Garrod found uric acid in pericardial effusion in gout.

Cardiac Walls.—Hypertrophy of the left ventricle figures as the leading change here. It is practically almost always present in greater or less degree, as associated with the renal changes already described. Rarely, no hypertrophy is found, but, instead, fatty degeneration and dilatation. The measure of hypertrophy is regulated by the general nutritional state of the individual, and by the amount of involvement of the kidneys and arterio-capillary vessels in the general sclerosis which prevails. The nutrition of the cardiac walls is in relation to the presence or absence of pericardial adhesions, which may lead to myocarditis, softening, or fibroid change, and to the permeability of the coronary arteries, which is often diminished by endarteritis or by atheromatous and calcific deposits. Changes in the other cavities depend on the competence of the left ventricle as secured by its nutrition, or as affected by co-existing valvular defects, and on the presence or absence of bronchitis and emphysema. The apex is often formed entirely by the left ventricle, the right appearing very small beside it. There is commonly associated dilatation of the left side. Fatty degeneration may ensue on profound atheroma of the coronary arteries. The weight of the heart is usually greatly increased. The average weight in forty-nine cases examined by Dr. Norman Moore was 16½ oz. In some the weight was over 20 oz., and in one reached 26 oz. (The natural weight is 11 oz. for the male, and 9 oz. for the female.) Softening or partial fatty change is more commonly seen. Ebstein believes that the heart is imperfectly nourished by blood rich in urates. He found uratic nodules in the heart's muscle in one case, some as large as a hemp-seed, and many smaller ones. In the vicinity of these were small-celled infiltrations, necrosing changes due to action

[1] *Op. cit.* Edinburgh, 1862.

of uric acid. No other observer has met with cardiac deposits of urates.

Endocarditis.—This is practically unknown in an acute form as a gouty lesion.[1] The changes affecting the endocardium are confined to chronic sclerosing lesions of the valves, and mainly of the mitral and aortic curtains. Thickening of the chordæ, with shortening, atheromatous patches, and calcareous nodules are found as the result. The tricuspid valve is rarely involved, and the pulmonary cusps most rarely. In the only case of the latter known to me, and reported to the Clinical Society,[2] there was reason to believe that some congenital affection of the valve had occurred, so that one cusp had disappeared, and an aneurysmal pouch had formed below its proper site. This was in a gouty man. Ebstein, on the strength of uratic infiltration having been met with on the valves, conceives that gouty endocarditis may exist. But he gives no facts in support of his view, and there are none known to me. The fact is, that uric acid is only found in very small quantities in this situation, and merely impregnating the ordinary calcareous salts met with in ordinary cases of the kind.

That the sclerosing changes are really due to gouty influence, and not to associated alcoholic habit, is proved by their occurrence in strictly temperate gouty individuals. Dr. Norman Moore remarks that "it is common to find uratic deposit in the joints of those persons whose aortic valves show chronic degenerative changes, with calcification, and who therefore belong to the class of patients likely to have had angina pectoris."

Aorta, Arteries, and Capillaries.—Dilatation of the aorta, with loss of elasticity, and atheroma in varying amount, even to calcific change, are met with.

Smaller arteries gape on section, and show thickened walls in most places. These changes are especially well seen in the cerebral vessels, and hence, under the influence of the powerful left ventricle, are prone to rupture, and induce a very common ending to all such cases, viz., apoplexy. As has been pointed out by Sir William Jenner, such vessels are not always brittle. They may be classed, indeed, under two heads—those that toughen, and those that become brittle. This is probably dependent on textural peculiarity of the individual. In respect of tendency to rupture, it has been noted by my late colleague, Dr. Southey,

[1] Sibson observed one case in which pericarditis and endocarditis occurred in a man with saturnine gout. The condition of the urine is not recorded. *Works*, edited by Dr. Ord, vol. iv.

[2] Trans. Clin. Soc., vol. xxi. p. 18, 1888.

that apoplexy does not usually occur until the left ventricular wall has begun to soften and decay.

Respecting the relationship between gout and cerebral hæmorrhage, it is shown by Dr. Norman Moore that in thirty-two cases of the latter in males, uratic deposits were present in thirteen instances, or in somewhat less than one-third of the cases. The earliest age in which these were associated was twenty-eight, and the oldest sixty-six years.

In ten fatal cases of gout, Pye-Smith found two resulting from cerebral hemorrhage.

Murchison noted that arterial atheroma supervening early in life, and diseases of the aortic valves, which are not congenital, and are independent of rheumatism and injury, are met with chiefly in the subjects of the lithic acid dyscrasia or of gout.

Sections of small arteries show great thickening of the internal longitudinal and external circular muscular coats, and the outer fibrous coat is also thickened. With respect to this endarteritis, although it is commonly associated with granular kidneys of all varieties, it must be affirmed that it is of especial frequence in persons of arthritic habit, and thus occurs very markedly in certain families.

Aneurysm is rare in the gouty,[1] and so, too, is gangrene from arterial embolism and thrombosis. Deposits of urates have been found in the renal arteries by Dr. Moore. He affirms that "urates are present in the joints of a large proportion of those persons over forty years of age who die of cerebral hæmorrhage."

The changes in the capillaries relate to thickening and brittleness, whence hæmorrhages and ecchymoses in various parts, as in the bladder, nasal mucous membrane, and the conjunctivæ.

Veins.—Morbid anatomy tells little in respect to these. Clinical observation tells more. The venous troubles of the gouty, though serious, happily most often end favourably. Phlebitis is well-recognized as a gouty ailment, with thrombosis, and the clots may be dislodged and cause sudden death by impaction in the heart, pulmonary artery, and lungs.[2] Schroeder van der Kolk has recorded a case in which the walls and valves of the veins were thickly infiltrated with urate of lime. The veins are apt to be large and full in gouty persons of sanguine type, and varix is not uncommon in them. Large veins may become suddenly and spontaneously blocked, sometimes permanently so, as in the

[1] In N. Moore's tables is a case in which an aneurysm, the size of a walnut, burst into the pericardium. The vessels were generally atheromatous. Man, æt. 44. Kidneys granular and cystic.

[2] *Nederland Lancet*, 1853.

axillary and iliac veins. The superficial crural veins are perhaps the most frequent sites for this, and the thrombosis may be patchy, extending up or down the limb. At times, great pain accompanies this process, of which I have seen one well-marked instance; more often only a little aching and uneasiness is felt. The veins are felt like cords, and œdema occurs, or not, according to the degree of mechanical impediment. Such cases are very tedious, and recurrence is common.[1] The occurrence of phlebitis in an elderly person without any evident external cause, according to Paget, warrants suspicion of gout, and this is perhaps the most common form of idiopathic phlebitis.

The frequency of hæmorrhoids has been already alluded to.

Liver.—The morbid anatomy of the liver in gout should surely demand attention, since few organs in the body have been more believed to be in fault in this malady. In truth, there is but little to be told concerning the liver in those dying directly from gouty cachexia.

Many of the changes are doubtless due to associated heart-disease. Chronic congestion, induced by alcoholic indulgence or over-eating, may lead to the capsular thickening sometimes met with. Pseudo-cirrhosis is due to venous remora resulting from cardiac obstruction in many cases. Fatty degeneration is not uncommon in cases of gouty cachexia, and the liver may thus be much enlarged in volume. The organ has been found small with thickened capsule, which may be adherent to adjacent parts.

Whether true cirrhosis can be induced by the gouty habit, apart from other and more common causes, is still a vexed question in pathology. My own opinion is that this may occur, but it is not easy to furnish absolute proof of it. Murchison[2] noted the frequency of cirrhosis in connection with gout, and remarked that "the condition of liver which develops gout renders it liable to suffer from alcohol, even in small quantity." He refers to cases of cirrhosis which have been preceded for years by lithic acid dyscrasia and dyspepsia, in which alcohol bears no part, and which have been termed chronic gouty hepatitis. Trousseau alludes to such cases.[3] Murchison reported the case of a girl, æt. twelve years, who suffered from interstitial hepatitis as the result of a chill which ended in cirrhosis. Both parents were gouty.[4] It is, at any rate, certain that all cases of cirrhosis are

[1] *Vide* Prescott Hewett, Clin. Soc. Trans., vol. vi., 1873. Paget, *op. cit.*, p. 376, and St. Barth. Hosp. Reports, vol. ii. p. 82, 1866. Tuckwell, St. Barth. Hosp. Reports, vol. x. p. 23.

[2] Lect. on Dis. of Liver, 2nd edit., p. 283, 1877.

[3] Clin. Med., vol. iv. p. 381.

[4] *Op. cit.*, Appendix, p. 631.

not due to alcoholic influence. Various irritants may excite it. It may occur in animals. Dr. Norman Moore's tables show that in the majority of cases of cirrhosis of the liver, uratic deposit is not to be found in the joints. In twenty-three cases, in both sexes, he found urates in the joints in but three instances in men, aged forty-one, forty-three, and sixty years. Ebstein has recorded cases of hypertrophic cirrhosis in gouty men without portal venous obstruction.

Degenerative joint-changes are sometimes met with in cases of cirrhosis of the liver. Erosion of cartilages, eburnation of bone, and outgrowths (lipping) may occur without uratic deposit. Alcoholic intemperance is presumable for most, if not all, of these cases. The special significance of this association of joint-change with cirrhosis of the liver would appear to be that more widely spread lesions, indicative of degeneration, are to be found than is commonly believed in chronic alcoholism. These changes only afford a parallel to the articular manifestations of gout in respect of their far-reaching character, and their determination to joints. Uric acid disturbances do not prevail in such cases, as a rule, because they are not gouty.

Biliary Calculi.—Biliary calculi are not often found in the bodies of those whose tissues show manifest signs of gout. In Dr. Moore's eighty cases, calculi were only found in three instances, twice in men aged fifty-four and sixty-two years, and once in a woman aged fifty. In the families of gouty persons, and especially in women, there is, however, not infrequently clinical history of biliary colic. Biliary and renal calculi have long been known to co-exist. I am inclined to regard the occurrence of biliary calculi as one of the occasional manifestations in women of imperfectly developed gouty habit. Uric acid is stated by Charcot to have been found in the gall-bladder in the form of calculi. This is on the authority of Frerichs. A reference to the original observation, however, shows that this was very doubtful. The source of the calculus examined was not certainly determined, and it was probably of renal origin. I have no knowledge of any example of the kind.

It is certain that, in many cases in which gall-stones exist, there is family history of ailments of a gouty nature, as well as of true gout, asthma, migraine, neuralgia, lithiasis, and tendency to urticaria. The habits of life leading to gall-stones are such as induce gout,—to wit, high living, anxiety, mental tension, and sedentary pursuits.

It has been shown that biliary colic is somewhat rare after

the age of fifty, while the formation of calculi in the gall-bladder tends to go on without dislodgment with advancing years, and they are often found in bodies where their presence has been unsuspected during life. They are seldom found in association with cirrhosis of the liver induced by spirit-drinking, and are more common in persons who are intemperate in malt liquor, which is notoriously gout-inducing.

In hot climates they are not common, and the same holds good for gout. Persons leading active, open-air lives are, as a rule, exempt from gout, lithiasis, and biliary calculi.

The opinion, therefore, as to the significance of gall-stones as a gouty indication in any individual is, like many others in respect of this disorder, formed upon clinical rather than upon *post-mortem* data.

I would just note in this connection the occasional occurrence of cancer of the gall-bladder and ducts, induced by irritation of calculi, which is met with in gouty persons, especially in women.

Genito-Urinary System.

Bladder.—Cystitis is plainly a gouty trouble in certain cases, and may occur in persons who, though gouty, have never had regular gout. Todd described examples, and regarded them as analogous to gouty pneumonia, bronchitis, or gastritis. It may occur by sharp metastasis. It is more common in elderly persons, and in those with prostatic enlargement. The muscular coat appears to be sometimes alone involved.

The possibility of calculous cystitis must be borne in mind in these cases.

Urethra.—Urethritis of gouty origin is distinctly recognized and is apt to supervene at the end of an articular attack when the pain is passing away,[1] or may occur spontaneously, or be excited by pure connection. Fibroid thickening of the spongy and prostatic portions, leading to tough stricture, may be found. Thrombosis of the veins in the corpus cavernosum of the penis is not very rare, leading to painless nodules, hard and circumscribed, varying in size from a pea to that of a French bean. These disappear very slowly, sometimes not completely.

Indurations of the fibrous sheath of the corpus cavernosum, caused by fibroid tissue in bands or lumps, may occur on the sides, dorsum, or septum. These induce chordee on erection.

[1] *Essai sur l'Urethrite goutteuse. Thèse pour le Doctora'.* E. G. Turbure. Paris, 1887.

They are usually very chronic, and perhaps may not disappear entirely.

Prostate Gland.—Any *post-mortem* changes detectible here will be results of long-continued irritation from calculi or from enlargement of the gland. Paroxysmal prostatic gout is sometimes met with, and temporary hardness and great tenderness of the part are found on examination by the rectum.[1] Sacculation of the bladder may be caused as the result of the more permanent enlargement, and chronic cystitis, with hypertrophy of the muscular and congestion of the mucous coats, may be met with.

Testis.—Although gouty orchitis occurs, nothing is known of its morbid anatomy. Chronic induration, generally of the body of the organ and not of the epididymis, may, however, result from acute gouty orchitis. The testis is less nodular and hard than in other forms of this disorder. Dr. Debout D'Estrées has recorded cases.[2] In one of these the body and epididymis were both involved, the testis being thrice its natural size. No effusion occurred into the tunica vaginalis. The latter may, however, be met with. The left testis is that commonly affected. Ebstein has recorded a case of hydrocele and orchitis on the left side in a bed-ridden gouty man.

Uterus and Ovaries.—Cases of uterine gout have been described. Sir James Simpson reported several.[3] The uterus may be affected metastatically from articular gout, and has been found large and fixed as by perimetritis. Tumefactions may occur in the broad ligaments. The mucous membrane has not been found inflamed, nor the cervix, in these cases. Menorrhagia and dysmenorrhœa are certainly sometimes due to gouty influences. As against these assertions, I may quote the opinion of my colleague, Dr. Matthews Duncan, who tells me he knows of no facts, either during life or *post-mortem*, that warrant the term "gouty" affections of either womb or ovaries, and that he knows of no gynæcologist who now asserts such an opinion. He remarks: "That gout may affect every organ and tissue I do not deny. I remember well having the same opinion, or knowledge, when I saw Simpson's so-called cases in Edinburgh, and when I was more credulous than now."

It is possible, however, that the influences of gouty heredity are not recognized in many cases of diseases of women, simply because no overt gouty symptoms present themselves.

[1] "I have certainly seen gout in the urethra and prostate gland."—*Sir H. Halford*, *op. cit.*, p. 108.

[2] *Loc. jam cit.*, p. 220.

[3] James Begbie, *op. cit.*, p. 19.

The disorders of the uterus and ovaries which depend on gouty influence have no recognized morbid anatomy, because they do not destroy life. Their true character is, therefore, only to be determined clinically, and, for this purpose, regard must be had to the general, and not merely to the local, conditions present in any given case.

I shall return to this subject in a future chapter.

CHAPTER V.

HÆMATOLOGY OF GOUT.

In discussing the humoral pathogeny of gout, I have already entered at some length into the question of the morbid state of the blood. Repeated research has confirmed most of what has been set forth by Sir Alfred Garrod, and, in truth, there is little to add to the fruits of his labours. Not many physicians are so qualified as he is to throw light on this subject, which demands the knowledge and practical skill of a physiological chemist. He has shown that the blood in gout is invariably rich in uric acid, and is specially charged with it prior to and during attacks. During recovery from acute attacks, diminution of uric acid occurs. In the intervals between earlier attacks no appreciable increase is detectible; but in chronic gout, at all times, even in inter-paroxysmal periods, excess of uric acid exists.

The amount of uric acid in healthy blood is so small as practically to elude detection. In gout, Garrod has found as much as 0.175 parts in 10.000. His "thread-test" has been repeatedly practised with the blood of the gouty, and, when carried out with exactness, seldom fails to indicate presence of uric acid (as uratic salts) in the blood. Better results are obtained from blood-serum procured from blood directly than from blister-serum, and the probable explanation of this is, as Garrod suggests, that the inflammatory process induced by vesication is apt to destroy the excess of uric acid in the part thus influenced. It is best to withdraw about one or two ounces of blood from a brachial vein in order to practise this method, and, as pointed out by Garrod, care must be taken to employ acetic acid of proper strength, that of the British Pharmacopœia, with sp. gr. of 1.044, of which 100 parts by weight contain 33 parts of real acetic acid, being most suitable.[1] Failure to find evidence of uric acid may arise

[1] *Vide* Garrod's method, described at length in his book, 2nd edit., p. 86.

from decomposition of the serum, due most frequently to high temperature, in which case the uric acid is changed into oxalic and carbonic acids.

If blister-serum is used, it should not be taken from a goutily inflamed part.

My own observations confirm Garrod's facts. It may be confidently affirmed that in the blood of most gouty persons there is excess of uric acid. This fact has been noted in the blood in both acute and chronic gout, but more markedly in the former, and not only is this true during active phases of the disease, but also in the intervals between acute attacks when the general health is good.

Urichæmia, however, is not peculiar to the gouty state alone, neither is it always demonstrable in cases of unequivocal uratic arthritis. It is present in leuchæmia, chlorosis, lead-poisoning, and other morbid states, which have no marked alliance with gout. Hence, mere urichæmia is not an absolute sign of gout, and does not by itself entail a paroxysm of it, though it is most often present, and it may determine incomplete phases of gout.

Diminished alkalinity of the blood is assumed as a pathogenic factor, depending on presence of uratic salts. Garrod affirms that the reaction of the blood in chronic gout is more nearly neutral than in any cases but the stage of cholera-collapse and some forms of albuminuria.

In gouty subjects uric acid is believed to be often retained in the liver and spleen. Dr. Haig's researches indicate that after five grains have been thus stored it is difficult to cause, by drug- or other interference, any further retention.

Blood-Plasma—Corpuscles.—Corpuscular richness is not affected in gout, the red globules being in full number. The leucocytes are not increased.[1] Impoverishment of blood is not usual after paroxysmal gout, at all events in the earlier attacks, and in this respect there is a marked difference from rheumatic fever.[2] Anæmia occurs as part of the cachexia of gout, and may be considerable after hæmorrhages by epistaxis and from hæmorrhoids. It is, however, rapidly recovered from in the gouty, as in the subjects of hæmophilia, if the flux be arrested. In two cases of chronic gout under my care, Dr. Tylden found the hæmoglobin in about normal, or in very slightly lessened, proportion. Where the kidneys are already affected with chronic nephritis, there is

[1] Dr. Tylden made several examinations for me.

[2] The red globules are diminished in acute gout, according to Quinquand. *Vide* Coupland's Gulstonian Lectures, Lancet, March 26, 1881.

diminution of red corpuscles and some increase of leucocytes. This is in relation to the co-existence of albuminuria, and may hardly be reckoned as a specifically gouty change. In granular kidney-disease, the red globules may be reduced to nearly one half the normal, and the leucocytes may be increased to thrice the average number, as pointed out by Dickinson. In saturnine gout, there is commonly marked anæmia or spanæmia, but in this case there is direct influence of a special blood-poison on the red globules. This degree of anæmia is rarely reached, even in cases of gouty cachexia, unless there has been poor living or alcoholic intemperance.

Fibrin.—Hyperinosis may fairly be assumed in certain phases of gouty habit, and is manifested especially by tendency to venous thrombosis, which is commonly associated with phlebitis.[1] This is now recognized as an incomplete form of gout. No exact researches have, however, been made. Blood drawn from a gouty patient appears to differ in no way from that taken from the subjects of indifferent inflammatory processes, and the amount of fibrin is probably in relation to the degree of inflammation present in each case, being increased in acute inflammation.

Albumen.—Little is known respecting the albumen in the blood of the gouty. It is stated to be in normal amount, even where there is renal degeneration. But little loss of albumen occurs in gouty nephritis. Garrod never found increase of albumen in blood from a gouty person, but believes that the specific gravity of the serum of the blood in gout is lower than in other diseases, with the exception of albuminuria and scorbutus.

Urea.—It has not been shown that excess of urea occurs in the blood in the earlier stages of gout. The research is, however, beset with difficulties. In all cases of granular kidney-disease, there is probably increase of urea in the blood. The amount is doubtless in relation to the degree of renal inadequacy, however ætiologically induced.

Oxalic Acid.—Oxalic acid has been frequently found by Garrod in the blood of the gouty, and is believed by him to occur chiefly in the paroxysmal stages, being derived by oxydation from uric acid.

In blood drawn from a gouty patient, Ebstein found that xanthin and hypoxanthin were formed on exposing it in a warm chamber, while minute quantities of uric acid disappeared.

[1] According to Sir W. Gull, fibrin is increased in painful affections.

CHAPTER VI.

UROLOGY OF GOUT.

In discussing the pathogeny of gout, reference was made to the fact that, both in the truly gouty and in those goutily disposed, there might be, occasionally, uratic deposits. The latter, though not alone significant of gouty habit, were shown to be more frequent and persistent in that condition.

A study of the whole question of the changes observable in this secretion in gout is necessarily a very large and complex one, and relates not only to the important point respecting uric acid and its salts, but to every constituent of this fluid. It is complementary to a study of the associated changes in the blood, and a similar inquiry should be addressed to the secretions of the skin, if we would obtain a deeper insight into the humoral relations of gouty pathogeny. I propose to discuss the conditions of the urine: (1.) in the præ-paroxysmal stages of gout; (2.) in acute paroxysmal gout; (3.) during the intervals of such attacks; (4.) in chronic or cachectic gout; (5.) in incomplete gout; and lastly (6.), in that known as gouty glycosuria. The points to be noted in any case are: the quantity passed, density, acidity, amounts of urea, uric acid, fixed salts, and the organic constituents.

1. Urine in Præ-Paroxysmal Stage.—It has occasionally been observed that free emission of a pale and watery urine has occurred before an acute articular attack. Scudamore noted this, and remarked that it was only seen in persons whose constitutions were much weakened by gout. I have inquired for this symptom in many cases, and have occasionally met with it.[1]

[1] It was marked in the case of an army-surgeon, aged forty-eight, under my care, who inherited gout from his father and grandfather, had lived freely, and had many attacks of gout, the first at the age of twenty-nine. He noted that his urine was very copious and pale for from five to seven days before the attacks, and on one occasion it had been so for three weeks before a paroxysm. He was much broken down, had suffered from shingles, and from attacks of eczema.

There is commonly a diminution of the principal ingredients, in particular of the uric and phosphoric acids and the pigments. Urea has been found deficient before an acute attack. Traces of albumen may occur.

A steady increase in the excretion of uric acid and urea in persons goutily disposed has led M. Lecorché to prognosticate the onset of primary acute attacks.

2. Urine in Acute Paroxysmal Gout.—In acute gout the urine commonly presents ordinary febrile characters. The quantity is found to vary, but is, as a rule, somewhat diminished. The fact is that, in acute gout, as I shall show later, there is commonly present only a moderate degree of pyrexia.

Hence, the water may not be much reduced in many cases. In acute attacks supervening in the subjects of chronic gout, the urine is less "febrile," and may be passed in full amount.

Density.—The density varies in relation to the excretion of water, and especially of the several constituents in solution. With respect to all examinations of urine made during a paroxysm, regard must be had to the diet employed, which materially affects the results.

Taking the mean density in ten cases of acute gout, I find 1.020, or the average of health, as the figure, the highest being 1.028, and the lowest 1.015. Other observers mention 1.007–1.025. A common mean is 1.014. The higher densities represent concentration of the urine to some extent, and the colour is also similarly influenced. A high density has been observed to prevail in the urine of many members of families who inherit gouty proclivity.

In acute gout the reaction is uniformly acid, especially for the first few days. This is attributed by Lecorché to its concentration, since at that time there is diminished excretion of uric and phosphoric acids. The acidity falls towards the end of the attack.

Acidity.—The acidity in health varies according to the state of digestion, and also in relation to the time of repose. Urine is alkaline after a meal, and most acid during fasting and after sleep, at which periods there is least excretion of carbonic acid from the lungs. The acidity is due to acid phosphate of sodium. It may occasionally be due to excess of acid urates, or to hippuric acid, the latter especially after a vegetable or fruity diet. (Garrod is of opinion that the presence of uric acid has no influence on the acidity.) The reaction of health, however, is in relation to that of the phosphates, whether they be acid, neutral, or basic.

As Bence Jones showed, when the urine is very acid, there may be but little uric acid present in it, and when the latter is largely present, the urine is sometimes neutral in reaction.

Urea.—As already mentioned, urea has been found deficient before an acute attack of gout. Garrod found in the case of a man, æt. fifty-seven, that the mean excretion was 320 grains, which, considering his age, was a fair amount. But few analyses of urea-excretion have been made in acute gout. Such as are recorded indicate that no material variation from the normal is to be expected, and it appears to be proved that such variation as exists is in no relation to the amount of uric acid excreted at the same time. Diminution during the paroxysm is attributed to loss of appetite, and to the simple diet employed during the attack. The relation of urea to uric acid in health in the adult is given by Lecanu[1] and Dr. Haig as 1 to 33, and the latter found in his researches on "uric acid headache" that the excretion of urea in these cases was practically not interfered with, while that of uric acid fluctuated much, and specifically, in relation to headaches.

It would be remarkable if urea were not increased during the febrile period of gout, since it is increased in all acute diseases, and especially during pyrexia.

In a number of gouty cases examinations of the urine were made by Dr. Mortimer Granville.[2] He found that there was no increased proportion of urea in most of them.

Uric Acid.—Persistent excess of uric acid in the urine is always significant of some important constitutional state, and indicates increase of tissue-metabolism in some particular organ or organs, or even throughout the whole body.

Non-elimination of uric acid has been proved by Garrod to be a marked and constant feature of paroxysmal gout. In seven such cases he found the mean out-put to be less by about five grains than the normal,—3.62 grains as against 8.569 grains. With this defective elimination by the kidneys there is simultaneous retention of it in the blood, or, at all events, within the body. Dr. Haig has recently studied these phases of uric acid retention, and believes that the acid may be stored in the liver and spleen. He has shown that gouty manifestations are apt to occur with excess of uric acid in the blood, and can be checked by means which cause its retention in the system—liver and spleen. In health, the excretion of it is greatest during the alka-

[1] Lecanu, *Journ. de Pharmacie*, t. xxv. p. 261.

[2] Med. Press and Circular, March 9 and 23, 1887.

line tide of digestion, as shown by Roberts, and Dr. Haig regards this as a washing out of the uric acid accumulated in the liver and spleen during the acid tide of sleep, and not entirely due to increased formation during digestion. In gout there is probably no defective formation of uric acid, but the reverse; and retention, stasis, and defective elimination of it constitute malign faults attendant on a paroxysm.

Hence, Garrod's theory as to deficient excreting power in the kidneys in respect of uric acid during a paroxysm of gout, and the very fair argument in favour of this afforded by the free elimination of it by the kidneys in other states, such as in leuchæmia and hepatic disease, where uric acid formation is also largely increased. It is probable that the excretion of uric acid in gout, could it be watched from hour to hour, would be found to vary considerably. Garrod showed that in the early stages of an attack the out-put was small, and gradually increased above the normal excretion till the paroxysm subsided, when it again diminished. The antagonism is therefore between retention in the system and elimination by the kidneys, and there probably is not, as has been supposed, "a rupture of equilibrium between production of the acid and its elimination" (*Rendu*).

In 1000 grains of morning urine in a case of acute gout, Sansom found .830 grains of uric acid; in one of chronic gout, .120 grains—a comparative experiment in a healthy person yielding .250 grains.[1]

M. Lecorché's researches on the elimination of uric acid by the urine in gout are the most exact I have met with. He has shown that the out-put is diminished before an acute attack, and is low for several days—two to four—during the paroxysm, increasing much above the normal the third and following two days, and again falling to normal towards the close of the attack. Hence, according to Lecorché, the greatest elimination occurs at the height of the paroxysm, and not at the end of it, as found by Garrod.

Phosphates.—It has been asserted that, along with uric acid, there is also retention of phosphates in acute gout. Bence Jones, Parkes, Böcker, and Stockvis have demonstrated this fact. The latter found in a case of gout that the phosphoric acid in combination with earths was diminished in comparison with other phosphates, not only during the paroxysms, but in the intervals. One-third of the phosphoric acid excreted daily is combined with the earthy oxydes—calcium and magnesium. Parkes conceived

[1] Quoted by Beale in "Urine, Urinary Deposits," &c., 2nd edit., p. 162, 1864.

that, as phosphate of lime is a constituent of tophi, it was probable that retention of this product would commonly be found to occur.

Teissier noted that the amount of phosphoric acid excreted was increased in the gouty. Lecorché found that it varied exactly as did the uric acid. It appears probable that these contradictory results arise from examinations made under very different conditions. As with uric acid and urea, so with phosphoric acid, the amount excreted is certain to vary according to the state of the patient's nutritive powers and his capacity for taking and digesting food. If we accept the view that there is retention of phosphoric acid in paroxysmal gout, we can hardly attribute any of the specific phenomena of the disorder to it alone, though we have here a factor which helps to explain the acknowledged diminution of the alkalinity of the blood in these cases, and the tendency to uratic precipitation in the tissues. W. Gairdner noted very free discharge of phosphates after paroxysms of gout.

Hippuric Acid.—This never forms a deposit. W. Budd found in some specimens of urine from cases of gout a flocculent precipitate consisting of benzoic acid, which probably resulted from decomposition of hippuric acid.[1]

Sulphuric Acid.—Parkes found this in normal amount, and assumed that there was no change as to retention or metabolism of sulphur-holding tissues.

Pigments.—The urine is commonly of full amber colour in the gouty. The pigments are frequently increased, the urine sometimes being red. The great affinity of urates for urinary pigments is well-known, and thus, in acute gout, deposits of the former are apt to be highly charged with the latter, and so to express the exact tint in each specimen. They are derived from urobilin and from hæmatin. Sometimes pale urine, deficient in pigments, is passed before and during a paroxysm (*vide* p. 188).

As often happens, with deficiency of ordinary pigments, there is in gout increase of uroxanthin. This occurs in acid urines, sometimes with deposit of uric acid, and may be demonstrated by heating with strong acids which cause red, blue, and green coloration. In one instance Parkes found a large amount of indigo on adding hydrochloric acid.

Albumen.—In cases of paroxysmal gout, certainly during the earlier attacks, there is usually no passage of albumen. With increasing frequency of attacks, the kidneys becoming gradually

[1] Quoted by Beale, *op. cit.*, 1864, p. 165.

involved, a trace of albumen is apt to be found. Its presence is therefore in relation to the degree of renal inadequacy, and the ability of the kidneys to withstand the additional stress thrown on them by the metabolic changes proper to the pyrexia and to the specific characters of the attack. Amongst the latter may be reckoned irritation of tubular epithelium by excess of uric acid eliminated.

Some degree of tubal catarrh may thus be set up in kidneys which are only slowly undergoing cirrhosing change, and which may be expected to pass off with remission of the acute attack.

The albuminuria of acute gout is always fleeting, not lasting more than two or three days.

According to Lecorché, it appears to be in relation to the excretion of uric acid being present about the third and fourth days of the attack, and diminishing along with the uric acid.

Glucose.—A small quantity of glucose may sometimes be found in the urine during a gouty paroxysm. This, like the albuminuria in such cases, is but fugitive, and almost certainly owns a hepatic origin.

The urine in acute gout has a strong "urinous" odour. It undergoes little change on standing after uratic sediments are precipitated, and from its marked acidity is little prone to fermentative process.

Sediments, and Microscopical Characters thereof.—Mucous corpuscles and epithelium from various parts of the urinary channels may be found in the sediment, together with deposits of amorphous pigmented urates and uric acid.

It is probable that the unduly acid condition of the urine leads to such irritation of the urinary passages as may cause some proliferative formation and shedding of the epithelial linings of their coats. Mucous clouds may be suspended in the urine, sometimes containing small crystals of urates.

3. Condition of the Urine in the Intervals between Gouty Paroxysms.—It is obvious that great variations must exist in the condition of the urine in the intervals between gouty paroxysms, these being determined according to the length of the interparoxysmal periods, the age and general nutritive state of the patient, and, more especially, by the presence of gouty cachexia. In younger persons of sound constitution, if care be exercised in maintaining health and in avoidance of dietetic errors—whereby long intervals may be predicted between acute attacks—no marked abnormal features prevail. In cases of chronic gout or

of gouty cachexia, other characters obtain which will be presently described.

Phosphates.—Stokvis found diminution of phosphoric acid in combination with earths in the intervals of gouty paroxysms as well as during acute attacks. The condition of the bones should be considered in relation to excretion of phosphates.

Ebstein quotes Bramson and Marchand as authorities for the fact that, in the apparently healthy bones of two gouty individuals, there was a diminution of the earthy phosphates and carbonates.

4. Urine in Chronic or Cachectic Gout—Uric Acid.—The result of analyses in these cases indicates that the excretion of uric acid is rather below the normal amount.

Bartel found in one case, in an infirm person, diminished excretion of uric acid during attacks of gout, and in a case of chronic gout he found an excretion of 0.225 gramme per diem.[1]

Urea.—Urea appears in normal quantity, or very nearly so, unless there is renal inadequacy due to some degree of interstitial nephritis. With defective excretion of uric acid there is tendency to retention in the blood or in the system, and, so far, a constant liability to paroxysmal gouty recurrence. Garrod believes that the kidneys lose to some extent their function of excreting uric acid in chronic gout. We are justified by study of the morbid anatomy of chronic gout—the only form of its anatomy of which we have knowledge—in believing that some measure of inadequacy is present in all cases coming under this category, and, hence, may believe that there are sufficient structural defects to account for insufficient elimination.

The amount of urea excreted is almost certainly in relation to the particular diet employed in each case, and to the extent of metabolic force available in the liver.

Phosphates.—The same must be said in respect of the excretion of phosphoric acid. Further researches are, however, much needed to complete our knowledge. Such as have been made are contradictory, and the probability is that they have been conducted under very different conditions in patients variously affected with gout, and without regard to the special diet employed, or the capacity in each case for its due assimilation.

The urine in chronic gout often presents the characters of that excreted by patients the subject of chronic interstitial nephritis,

[1] *Deut. Archiv. f. klin. Med.*, Bd. i. p. 30, 1866; *Nierenkrankheiten, v. Ziemssen's spez. Path. u. Therap.*, Bd. x. p. 375. Leipsic, 1875. (Quoted by Ebstein.)

which condition, indeed, is just what is present in so many of these cases.

Polyuria Common.—The urine may be copious in amount, from three to four pints daily, pale bright, often frothy when passed, of low range of specific gravity, 1.005 to 1.015, containing sometimes a trace of albumen, or for long periods none, throwing down a slight mucous deposit, and occasionally a few hyaline or granular casts, some renal epithelium, seldom fatty. Several micturitions are common during the night. In such cases, associated cardio-vascular and retinal changes may be looked for.

It often happens that the patient is satisfied with this condition of his urine, believing the secretion to be in a more healthy state than when dark, more scanty, and loaded. The practitioner will form a different opinion on reviewing the whole features of the case. Hence, it is proper to examine the urine at intervals in all cases of chronic gout, and to meet any indications as they arise.

Albumen.—Some degree of albuminuria is common in chronic gout.[1] The albumen is not present in large amount, and the urine has the ordinary characters of that passed by the subjects of chronic sclerosing or interstitial nephritis. Thus, albumen may be present in small quantities, and fugitive, sometimes disappearing for long periods, and, hence, the presence of this abnormal constituent is not necessary in order to justify diagnosis of progressively granulating kidneys. Other characters, such as polyuria and low specific gravity, due to deficient urea and salts, afford sufficient presumption of renal change.

Albuminuria is sometimes the leading symptom of visceral gout, falling mainly upon the kidneys.

5. The Condition of the Urine in Incomplete Gout.—By incomplete gout is signified imperfectly developed gout. Many cases may fairly be placed in this category which, while they manifest many of the phases of well-recognized goutiness, yet present no classical or paroxysmal features of the disorder. Many terms have been used to indicate this condition, thus "latent," "masked," "lurking" gout have been described. It is desirable to employ none of these terms, and it is wiser to recognize the real underlying habit of body which exists in such cases.

[1] In sixty-one cases of gout occurring in hospital practice examined by Pye-Smith,* there was albuminuria in seventeen instances; in another case, where it was absent, the kidneys were found after death to be granular and contracted. In two instances there was eclampsia, apparently of renal origin, so that this affection probably existed in at least a third of the cases.

* Guy's Hosp. Reports, 1873.

Many of the symptoms thus described are due to visceral gout, and these naturally vary much according to the organs involved. Many of the vague pains and aches to which gouty persons are obnoxious are due to incomplete outcome of the disorder, and are commonly considered and treated as "rheumatic," often without success. Both sexes suffer, women, perhaps, more often than men. I am only here concerned with the condition of the urine in cases which may be considered typical of the state referred to, and it is, in truth, much the same as that already described in respect of urine in the præ-paroxysmal stage of gout. Thus, the urine is apt to be loaded and to deposit lithates. It is high-coloured, very acid, and strongly urinous in odour. The *urina sanguinis* is unduly acid, the *urina cibi* often alkaline, apt to be turbid and to throw down phosphates. Micturition is more frequent than usual. Oxalates with mucus, as a hummocky cloud, may sometimes occur as a deposit. Albumen and glucose are absent as a rule, but there may be fleeting glycosuria to slight extent. Many cases of lithiasis fall under this category. Dietetic errors, and excess in wines, fruit, or rich food, will speedily aggravate the conditions just described, and often induce vague pains both in the liver, the head, and various joints.

Some sufferers so far recognize for themselves the gouty nature of their troubles, thus induced, that they purposely indulge further in gout-provoking diet with a view to induce a regular attack, and so render an incomplete attack complete. This is not always achieved, however, and paroxysms are not readily to be induced in some persons, and rarely in the aged, or those already broken down in health. There is often an atonic condition, or an absence of all the elements necessary to induce complete gout in the subjects of the incomplete form of the disorder. In particular, there is insufficient nervous activity, and nutritive metabolism is possibly insufficiently vigorous for its production.

In some cases, where articular manifestations are in abeyance or only slightly manifested, various other troubles, such as eczema, phlebitis, hepatalgia, dysæsthesiæ, palpitations, headache, or neuralgia, often occipital or cervico-brachial, supervene, and nothing short of anti-gouty medication will afford relief.

6. Gouty Glycosuria.—The occurrence of fugitive traces of glucose in the urine passed during paroxysmal gout has been already referred to. Saccharine urine may occur at intervals in cases of incomplete gout, and may alternate with deposits of uric acid. Confirmed gouty glycosuria may pass into chronic diabetes, and a parallel condition is thus established with albuminuria, the

presence of glucose being the indication of a variety of visceral gout effecting the liver.

Dr. Bence Jones was amongst the first to direct attention to gouty or, as he termed it, intermittent glycosuria. In France, diabetic patients have long been placed in the two categories of lean and fat. Amongst the latter are many of the class under consideration.

Garrod observed the onset of glycosuria in gouty patients who thereafter became free of most of their gouty symptoms, and he surmised that, the solids of the urine being carried off by the polyuria, uric acid was no longer retained in the system. He found the intervals between the attacks lengthened, or the paroxysmal tendency prevented in cases with free flow of urine, while in those cases in which no polyuria occurred, although much glucose was passed, acute attacks still occurred. In such cases he believes the uric acid to be incompletely removed from the system.

In pronounced cases, well-marked exacerbations occur, and sometimes with more or less distinct gouty symptoms, articular or visceral. The quantity of urine is increased, and may be double the normal amount. In some cases the quantity is below the normal out-put. The colour may vary from bright amber to that of pale straw. The specimen strongly refracts light, is markedly acid, and unusually void of deposits. The amount of glucose may vary greatly. Sometimes the *urina sanguinis* is more impregnated than the *urina cibi* in the same case. From three to fifteen per cent., or more, may be present; according to Lecorché, thirty to forty grammes per litre. Lithates are, as a rule, never precipitated in urine of this class, but, occasionally, uric acid sand may occur as a deposit, and alternate with glucose. In persons of gouty heritage, in youth and in early adult life, a small amount of glucose may be present together with an increased excretion both of uratic and phosphatic salts, not generally exceeding one or two per cent. This condition is commonly amenable to suitable treatment, but demands recognition as early as possible.

CHAPTER VII.

HEREDITARY AND ACQUIRED GOUT. ATAVISM IN GOUT.

Gout is commonly described as a hereditary and also as an acquired disease. The history of the greater number of cases affords illustration of the influence of heredity in varying degree, and amongst the upper classes of society this factor is not often far to seek.[1] Where ancestral history is obtainable with any degree of accuracy, a disorder like gout, at all events in its regular form, is not likely to be forgotten or mistaken, and any record of the disease in its "chalky" or tophaceous form cannot be wrongly interpreted. It is, however, often difficult to discriminate in the accounts given by patients whether they, their ancestors, or relations have suffered from ailments truly gouty or truly rheumatic; and this is especially hard to determine if gout has occurred in an irregular or incomplete form.

History of both gout and rheumatism is common in the families of gouty sufferers, and in such cases it is proper, I believe, to have regard to the presence of an arthritic diathesis as implanted in the stock, with potentiality for evolution in either direction, according to the special environments of each individual thus originally impressed.

It is certain that this diathesis is widely spread, and that by intermixture many and varied phases of its presence are witnessed. This is true of all diathetic states. So much is this the case, that it naturally becomes very difficult, if not actually impossible, to affirm with certainty that in any given instance a disorder is actually acquired *de novo*. A study of life-histories over long periods, conducted by the light of modern views as to evolution,

[1] In a pedigree extant at the Heralds' College it is gravely stated that our common ancestor Adam died of the "Gowte"! (Compiled by a monk, probably in the thirteenth or fourteenth century.)

points very conclusively to the fact that tendencies in families and individuals may lie long dormant, may even be repressed in certain lives, and yet reappear in later members of the same stock under certain provocatives. This up-cropping of tendencies is so plainly seen in many instances, that it is only fair to recognize the possibility of it in others where no history is available or readily forthcoming to explain it.

By tendency, in the case under consideration, I mean tissue-peculiarity or potentiality, whereby certain textures are so impressed as to undergo, in course of time, definite trophic changes in response to definite exciting causes which lead to the evolution of gout or gouty manifestations.

It is seldom possible, I believe, in any case of gout occurring in an individual whose ancestors have been long settled in this country, to affirm with certainty that the disease is absolutely newly acquired. Many points in the case may favour this belief; but, I repeat, no certainty is possible. It is less difficult to assume that the disorder is acquired when the individual comes from a stock, and also from a country, where gout is not known to have prevailed. In the cases of many Irish people of the lower orders who come to London and grow gouty, we may be fairly sure that their progenitors were not gouty, inasmuch as gout is, and probably always has been, unknown in the peasantry of Ireland. But even here we find that they come of a stock which may be very markedly arthritic, since chronic rheumatic arthritis is a common disorder there.

Facts, however, go to prove that, with few exceptions, certain habits of life and peculiarities of diet will induce gout in any people and in any country. Over-eating and intemperate drinking of certain alcoholic liquors, together with indolent habits, prevail too commonly to induce gout in all but a few climates. Hence, the fact of acquired gout must be accepted.

As pointed out by Dr. Harry Campbell in his very suggestive work on the "Causation of Disease,"[1] the power of acquiring may be inherited, but the inheritance of the acquisition may not be entirely implanted in any case; for, if an individual were placed under an environment incapable of effecting structural change or acquisition, it would never appear in him. Hence, many persons may be potentially capable of developing gout, but owing to fortunate circumstances never have the chance of becoming gouty. This argument is applicable to any diathetic condition. *Per contra*, many persons are potentially incapable of developing

[1] Page 140, 1889.

gout or rheumatism, or any manifestation of the arthritic habit of body.

Statistics culled from English, French, and German practice go to prove the powerful factor of heredity in gout. Amongst the upper classes, in whom more gout prevails, and whose family histories are more trustworthy and complete, the influence is strongly shown, and occurs in from fifty to seventy-five per cent. of the cases. More complete knowledge of ancestral peculiarities would, I conceive, allow of a much larger percentage in this relation, perhaps even ninety per cent. of all cases. For the remainder, acquirement may be fairly acknowledged.

Without doubt, heredity is one of the most powerful factors in gout. As I have pointed out already, the disease does not always "breed true." Owing, probably, to intermixture of diatheses, to reversion to former conditions of trophic habit, other forms of arthritism may be developed, and many notable strange products along with them. To take one somewhat common example: the daughters of gouty men are not infrequently the subjects of chronic rheumatic arthritis. This may be merely a phase of the arthritic habit, or a reversion to a type of it long dormant in the line of a long ancestry; or, again, it may be a compound result of blending with a strain of strumous proclivity introduced at some period into the family line. The problem is so complex that it cannot at present be unravelled. We may guess, and we should do so, at the causes of the result, and in time we may find the component factors and put them into their true relations. Meantime, the features and evolutional potentialities of each arthritic case must be closely studied with the trained eye, mind, and acumen of the naturalist. This is the scientific method in pathogenic inquiry, and it is best begun by a humble confession of our ignorance up to the present time.

The study of hereditary tendency is of the utmost value in clinical work, for, by knowledge of it, we have power to avert, not seldom, from individuals the malign evolutions of inherent potentiality. By variation of surroundings we may accomplish much while the organism is still young and pliant. When habits are formed by definite environment, it is very difficult to secure any marked variations from the type assumed.

The causes of fresh acquirement of a gouty habit may be defined as relating especially to ease-loving and luxurious habits, over-indulgence in good living and alcoholic drinks, deficient bodily exercise, and exhaustion of the great nerve-centres.

Facts prove that heredity leads to early establishment of the

disorder, while acquirement seldom entails gouty symptoms before the middle or end of the fourth decade of life.

The prognosis in the two conditions varies, and is, on the whole, better for the victim of acquired habit, especially if he is a prudent and sensible person with a strong will, who can control his appetites.

Graves noted as a distinction between hereditary and acquired gout, that in the former case arthritic attacks were apt to come on suddenly without the slightest precursory derangement of health, or the operation of any assignable cause, whereas he had seen no instance of a similar kind in acquired gout.

Atavism.—It has been affirmed of gout, as of other constitutional diseases, that it is apt to skip a generation, and reappear in the grandchildren of those who suffered from the disorder. I must confess that I have seen reason in many instances to question the truth of this. Several fallacies beset the inquiry. It is now well-ascertained in biology that certain characteristics often lie dormant in successive generations, and that such latent qualities are apt to crop up from time to time in response to favouring conditions. This common belief in regard to gout has probably had reference alone to instances of overt and articular attacks of the disease. The minor degrees of the dyscrasia, including the multiform phases of it now recognized as goutiness, or as forms of irregular or incomplete gout, have been much, if not altogether, disregarded or unappreciated, and it is certain that many of these occur in the children of the gouty by direct descent, without classical and paroxysmal phenomena. These characteristics may be so far suppressed as to disappear for one or more generations, but may reappear under due provocation, and develop into intense manifestations. When all the circumstances and clinical histories of gouty families are critically reviewed, the facts do not lend support to a doctrine of pure atavism in gout. Cross-breeding and blending of diathetic states may do much to modify and check the inherited proclivity, and careful selection in alliances might, conceivably, be potent to suppress all gouty taint. But the facts in many cases do not lend support to this proposition, and much labour and study are still required to produce materials whereon to found exact and definite laws in respect of inherited taints and implanted proclivities. In the meantime, it is the duty of the physician to recognize the specific characteristics, physiognomical, textural, trophical, and evolutional, which are distinctly proper to the manifestations of the arthritic diathesis.

CHAPTER VIII.

ON CONDITIONS ALLIED TO GOUT IN THE LOWER ANIMALS.

THE results of some careful studies of this subject have not hitherto thrown much light on it, nor been fruitful for humanity. It may be fairly stated that no disease bearing close resemblance to gout during life has been met with in other animals than man. Concretions of uric acid have been found in some reptiles and birds which have been kept in confinement and under unnatural conditions. These occurred in the kidneys and other viscera of the former, and about the joints of the feet in the latter. Guanin-gout is met with occasionally in swine. After ligation of the ureters in birds and removal of the kidneys in snakes, masses of urates have been found in the viscera and on serous membranes, also in joints. It would be a straining of terms to call this gout. The latest contribution to the subject is that of Dr. Mendelson of New York,[1] who describes in detail his research into the nature of guanin-gout in the hog. Guanin has the chemical composition $C_5H_5N_5O$, and was first obtained from guano. According to Foster, small amounts are found in the pancreas, liver, and muscle-extract. It unites with alkalies, acids, and salts, and forms crystallizable compounds. By oxydation it yields, amongst other substances, small quantities of urea, xanthin, and oxalic acid. Uric acid may be converted by sodium-amalgam into xanthin and hypoxanthin. Guanin is thus an ally of uric acid.

Dr. Mendelson found in his case of guanin-gout in the hog, that guanin was first deposited in the bone without signs of adjacent inflammatory action, the cartilage being simply pushed before it. In the cartilages the deposit was primarily interstitial (in the ground-substance), but also met with inside the cor-

[1] Amer. Journal of the Medical Sciences, February, 1888.

puscles. The needles of guanin in the cells were not parallel to those in the ground-substance. Deposits were found in the medullary spaces of bone spreading towards the cartilage, and they were met with in the veins, in tendinous sheaths, and muscles. He agrees with Ebstein as to molecular necrosis at the site of deposits, but believes that in the hog the latter are primary and the necrosis secondary. The guanin-crystals were fine and hair-like, and doubly refractive under the polariscope, transmitting light when the Nicol prisms were crossed.

This disorder is rare in swine, though it might be expected to be common under the conditions of their lives as directed for the market. It must be borne in mind, however, that they are slaughtered at an early period, and seldom reach advanced age.

Although these researches are helpful in determining part of the morbid anatomy and the pathogeny of gouty deposits, they afford little insight into the more abstruse and multiform features of gouty disease as met with in human beings.

CHAPTER IX.

RELATION OF GOUT TO OTHER MORBID STATES, AND ITS INFLUENCE ON THESE. COMMINGLING OF GOUT.

MANY of the acknowledged difficulties relating to the whole subject of gout are due to the peculiar and unquestionable influence exerted by the gouty habit of body upon other diathetic predispositions and tendencies.

Hitherto, we have been concerned exclusively with the changes wrought by pure and uncomplicated gout. To describe the disease as met with in daily practice, demands at this point a larger consideration of the whole subject. Although classical examples, both of articular and visceral gout, abound, many instances are met with in which other conditions are modified variously by gouty influence.

I propose, therefore, to treat of the relations which exist (1.) between gout and rheumatism, discussing the latter in its widest sense; (2.) between gout and lead-impregnation; (3.) between gout, struma, and tuberculosis; (4.) between gout and cancer; (5.) between gout and syphilis; (6.) between gout, diabetes, and glycosuria; (7.) between gout and obesity; (8.) between gout and oxaluria; (9.) between gout and splenic leuchæmia; (10.) between gout and purpura; (11.) between gout and hæmophilia; (12.) between gout and traumatism; (13.) between gout and osteitis deformans. I shall also discuss (14.) the influence of the gouty habit on some specific febrile and acute diseases, (15.) its influence on painful affections, and (16.) pyæmic arthritis and gout.

1.—The Relation between Gout and Rheumatism.

I have already discussed this subject in part under the head of the Pathogeny of Gout, and I take my stand with those who

regard the latter as an offshoot from the parent arthritic stem. Those who have most studied both diseases will best appreciate the difficulties which beset the truth-seeking inquirer in establishing an exact diagnosis in certain cases. So pressing are these difficulties, that even the most practised observers will sometimes hesitate in pronouncing for one or the other morbid condition, and some have arrived at the conclusion that discrimination is often impossible.

My own observations have led me to be dogmatic in most instances, and only doubtful for a time in respect of others. An exact diagnosis is not always possible on the instant, but a few days' observation usually suffices to clear up doubtful points.

For a clear comprehension of the difficulties, an intimate study, both clinical and pathological, of the features of each morbid state is essential. In this country there is no lack of material in either category, and if this vexed question can be settled anywhere, it should assuredly be solved in the British Isles.

Some of the errors which still prevail on the subject may be traced to dogmatic teaching from centres where one or other of these diseases—but not both—happens to be common. It is even now maintained by some in this country that, so much are gout and rheumatism blended and intermixed through successive generations, that it is not possible to unravel the problem, and pronounce with certainty that this is pure rheumatism and that pure gout. I am prepared to deny such an affirmation. My opinion is that each "breeds true," but that, as will be shown, comminglings sometimes occur.

I discuss, first, the relations between gout and acute articular rheumatism or rheumatic fever. Difficulty seldom occurs in establishing the diagnosis here. Excluding pyæmic arthritis and gonorrhœal arthritis, we have to distinguish between rheumatic fever and those rare instances in which polyarthritis uratica occurs, that is, gout set up acutely in several joints at the same time, or between rheumatic fever and acute attacks, or exacerbations, of chronic rheumatic arthritis.

The difficulties here are clinical. Diagnosis is for the living, and not a puzzle to be solved by necropsy. Not to enter fully into the differential characters of the disorders now under consideration, which, indeed, are known by any tyro in medicine, it may suffice to indicate that rheumatic fever is a disease chiefly of adolescence, and gout in its paroxysmal forms one of middle or advancing life.

The antecedents of each are markedly different, so that while an attack of rheumatic fever cannot be predicted, a paroxysm of gout may not infrequently be foretold days in advance. The pyrexia of the former is severe, and may be dangerously so, while that of the latter is commonly mild, and hyperpyrexia is unknown in connection with it. The acute cardiac troubles and profuse sweatings of acute rheumatism have no place in gouty paroxysms. It is hardly possible to speak of any paroxysmal tendency in acute rheumatism in the sense in which such term is applied in gout. The anæmia induced by rheumatic fever does not follow acute gout. With respect to the joints specially affected and their appearance in the cases now under consideration, there is a certain resemblance. In both rheumatic fever and acute gouty polyarthritis, large and small joints may be attacked simultaneously or in succession, and the degree of effusion will by itself hardly aid in diagnosis. The difficulty of diagnosis is enhanced in any instance if there is, as there may be, history of previous rheumatic fever in early life, and possibly signs of old cardiac valvulitis, since it is certain that the subjects of true rheumatism may sometimes grow up gouty.

The essential distinction is to be made by examination of the blood and the urine. In acute rheumatism, there are no noteworthy changes in the blood in respect of uric acid, while the urine presents the characters of that secretion in ordinary febrile states, there being no excess of uric acid excreted.

In gout, on the other hand, there is plain evidence of urichæmia, while there is the characteristic diminution of uric acid at the outset of the paroxysmal stage, and the equally significant excessive discharge for several subsequent days.

Therapeutical measures are also a touch-stone here, since sodium salicylate is almost specific, and rapidly so, for the one, and colchicum inoperative; the converse being equally true, certainly for a few days, in the case of gouty polyarthritis.

It may, therefore, be affirmed that there is but little relation between acute polyarthritic gout and acute rheumatism, and that little indirect. No one could venture to draw an analogy between rheumatic fever and acute gouty monarthritis. The only common ground for such morbid states is that of the arthritic habit of body in which there is inherent predisposition, determined by nervous influences affecting the great motor centres, for errors of nutrition in the tissues of joints; and in virtue of this neuro-trophic instability, the specific peccant matters of each disorder in question work out their mischief. The gouty and

rheumatic branches of the parent arthritic stem do not run parallel, but diverge.

It must, however, be allowed that doubt will remain occasionally for a time in cases of the acute form of rheumatoid arthritis. When these occur in middle life, or when an exacerbation takes place and several joints are affected at once in this disorder, the case presents many of the features of generalized gout. It is not the fact that, as stated by some authors, gout most often attacks the robust and full-blooded.

There is a poor and atonic true gout, the appanage of the feeble and lowly vitalized, and in such persons there is much resemblance under the stress of unequivocal gouty polyarthritis to attacks of acute rheumatoid arthritis. Such cases are often for a long time obnoxious to any treatment, and, hence, the therapeutic tests are hardly of avail to clear up diagnostic difficulties. Family history and antecedents, however, commonly help here, and urichæmia is the abiding factor even in these cases.[1]

The greatest difficulty, however, is sometimes experienced in making a differential diagnosis between the chronic forms of gouty and rheumatic arthritis. Some of the most honest and careful observers decline in many instances to be dogmatic in respect of these. The particular joint affected may be a guide to diagnosis; thus, if the shoulder-joint be alone affected, the disorder is unlikely to be gouty, since true gout in this part is of extreme rarity. Omagra is almost always rheumatic.

The relationship of gout to chronic rheumatic or rheumatoid arthritis is one of such large importance that I am compelled in considering this question to discuss the nosological position of the latter at some length.[2]

At the outset, I think I may affirm that London, as a sphere of observation, affords, perhaps, the largest opportunities for study of all forms of articular trouble. We see the joint-troubles of all nationalities; we have a large field for observation amongst the Irish, who form a considerable part of our hospital and workhouse inmates and attendants. We see, without any doubt, the largest number of gouty cases anywhere met with on the habitable globe. We have scrofulous cases in abundance, and no lack of

[1] In an analysis of five hundred cases of acute rheumatism made by Dr. Syers of the Westminster Hospital, he obtained a family history of gout in 7.6 per cent. of the number. Lancet, June 30, 1888, p. 1292.

[2] I have treated this subject in an address on the Nosological Relations of Chronic Rheumatic Arthritis, delivered in Belfast, Brit. Med. Journal, August 9, 1884, and in the Art. "Chronic Rheumatic Arthritis," C. Heath's Dict. of Practical Surgery, 1886.

unequivocal rheumatism. We see more than this in the comminglings and coalescence of all these forms of disease. We see, what is very interesting and important—the influence of London life upon persons who come from Ireland, Scotland, and the provincial districts, from the colonies and elsewhere, and who bring with them their peculiar heritage and morbid tendencies, to be not seldom modified, more or less, by the peculiarities of life in our vast metropolis. An honest observer, seeking the truth in London, may rest assured that he has a large and fair field for his study of arthritis in all its forms.

In approaching the study of rheumatoid arthritis, it is necessary, in the first place, to have a clear idea of what is sought, and we must start with well-defined conceptions as to the significance of certain symptoms and physical signs. Some might here join issue with me at once, and, possibly, reject such definitions as I propose. Nevertheless, I may confess myself familiar with rheumatoid arthritis as it is recognized in its purest form in Scotland and Ireland. I have seen it in the hospitals of Edinburgh and Dublin, and studied its morbid anatomy on the shelves of the museums there.

I have good reason for taking as typical such a case as would be at once recognized in Scotland or Ireland as characteristic, and my reason is, that in these countries there are probably fewer influences at work than in England to modify the natural course of the disorder; and, in particular, there is practically an entire absence of gout amongst the classes who furnish the commonest subjects of rheumatoid arthritis. It may be partly for these reasons that some of the best studies respecting the disease have been conducted in Ireland; and I think we cannot seek a purer source of information, unless, indeed, it be the exact contributions of the Parisian school to this subject, especially those of Cruveilhier, Trousseau, and Charcot.

It may be certainly affirmed that in France there is less gout than in England, and in America gout is as yet hardly recognized. So, too, in Holland, in Germany, and in Russia, true gout is amongst the rarest of diseases, and rheumatoid arthritis in all these countries is a common malady. It is not unimportant to note this fact, because it is certain that the descriptions of the disease given by foreign writers better agree among themselves, and also with those of the Irish and French schools, than they do, for the most part, with some of those of the English school, particularly as represented by London.

It may, I think, be fairly said, that but for English ob-

servations we should never have had the term "rheumatic gout."[1]

By rheumatoid arthritis I mean an essentially chronic form of joint-disease, affecting both small and large, one or many, articulations.[2] It may begin insidiously, with pain and swelling gradually increasing, or it may begin by more acute local symptoms. The tissues of the joint are affected by a chronic and often progressive inflammation, beginning first in the synovial membrane, affecting next the articular cartilage, and this, perhaps, in most cases more severely than any other texture; then the ligamentous structures; and, lastly, the ends of the bones.

The morbid anatomy varies according to the intensity and duration of the disease, the simplest expression of it being Heberden's nodes or "end-joint rheumatism," in which there is no more found after death than an enlargement of the natural phalangeal tubercles, slight synovial thickening, and expansion of the articular cartilage. In its gravest form there may be profound change in all the structures of the joint, with effusion into it, ulceration of cartilage, eburnation of bone, bony and cartilaginous outgrowths.

Rheumatoid arthritis implies more or less deformity and crippling. The term, as I understand it, covers all cases known as arthritis deformans, osteo-arthritis, monarthritis (*e.g.*, malum coxæ senile), and nodular rheumatism.

But the type of the malady varies as it is more or less acute, more or less general, and, somewhat, according to the age and sex of the patients; and its forms have been clinically well-described as acute, chronic, and irregular. Both sexes are affected, but females in larger proportion. The acute and general form is more common in young persons, is met with even in childhood, and especially in women, and thus assumes the characters of a severe constitutional disease in more marked form. At the menopause, too, the disease is apt to be acute and rapidly progressive. The smaller joints, especially of the hands, suffer more in this form. The chronic form, of more insidious origin, is met with more often after middle life and in the male sex, affecting more especially larger and often single joints. This is often excited

[1] "A name which seems to have been invented to cover the difficulty of nicer discrimination."—*Letter on Rheumatism and Gout, addressed to Sir George Baker, Bart., P.R.C.P., by John Latham, M.D., Physician to St. Bartholomew's Hospital*, p. 69. London, 1796. Haygarth alluded to nodosity of the joints as commonly called "Rheumatick Gout." See Clinical History, p. 188, 1805. John Hunter, believing that no two diathetic conditions could co-exist, was strongly opposed to this appellation.

[2] The term "rheumatoid arthritis" was first introduced by Garrod in 1858.

by local injuries. Many exceptional cases, however, are met with. In by far the great majority of cases, there are no associated visceral lesions. In the exceptional instances, these may often be traced to antecedent true rheumatism. I forbear to describe the more minute changes set up by the disease. Enough has been stated to differentiate the malady from other forms of arthritis. Many negative symptoms also avail to distinguish it. I have given the typical characters, and with these we are in a position to prosecute further study. At this stage of the inquiry it is nothing to the point to affirm that such a malady is in relation to other forms of arthritis. It may be, and I believe is, in such a relation, but we must have a recognized type for comparison at the outset. And so far most authorities are agreed. It will not be contested that the only possible methods for study of any disorder must proceed on the lines, first, of accurate clinical observation, and, secondly, on those of morbid anatomy.

We get but incomplete knowledge by the pursuit of either method alone, and it may be taken as an axiom in pathology that coincidence of structural changes, as found in the dead-house, by no means implies identity of process in leading up to them. I would say that some of the difficulty attending this subject has come from a forgetfulness of this. Although both lines of study, clinical and pathological, have been followed in respect of rheumatoid arthritis, neither the precise nosological position nor the exact clinical relations of the disorder have as yet been accurately determined. The whole question is a very difficult one. The following are the opinions that have been entertained as to its pathological origin :—

1. That it is chronic rheumatism.

2. That it has no direct relation to rheumatism, but only a likeness to it ; and that it may exist with or without a rheumatic or gouty tendency, and is in no way antagonistic to either, partaking rather of the nature of a senile change, induced by wear and tear.

3. That it is closely allied to rheumatism, yet presenting some features of gout, is neither rheumatic nor gouty, but " intermediate between the two," presenting some characters of both, and therefore well-named rheumatic gout.

4. That there is an arthritic diathesis, or peculiar condition of tissue-health, involving tendency to inflammation of joints and fibrous structures ; and that upon this as a foundation may be built up, under the influence of special causes, a tendency to gout, rheumatism, or any one of their various modifications and combinations.

That rheumatoid arthritis is to be included under the term rheumatism. That rheumatism is an almost universally spread tendency to arthritis in connection with catarrhal nerve-disturbances, and gout is a tendency to arthritis in connection with blood-disorder. That these two classes of causal influence are not antagonistic, but are, on the contrary, often met with mixed together, and that thus the term "rheumatic gout" is completely justified. Further, that most cases of this disease seen in practice represent, not a simple mixture of these two causal influences, but the modified result of such in former, perhaps in many, preceding generations. Hence, the inseparable blending of the two often witnessed.

5. That the disease is of nervous origin, and due to irritation of nerve-centres.

6. That it is neither of rheumatic, gouty, scrofulous, nor in any way of specific origin, but "is a lesion common to several kinds of ailment," and "not justly separable as a disease of independent character." It is, according to this view, always symptomatic.[1]

I now proceed to a critical review of the several opinions which I have enumerated, and address myself to each as concisely as possible.

1. Is it, or is it not, the case that rheumatoid arthritis is nothing else than a form of chronic rheumatism? I confess to some dislike of the latter term. It has been, and still is, a much-abused one, too often a cloak for ignorance, and used without exactitude for many different conditions. It is too vague. It implies at once an origin of unequivocal rheumatic nature, and fairly presupposes an acute attack as a necessary antecedent in the case. In this sense alone I would retain the term, and only apply it to cases, not perhaps very common, in which the joints have not recovered after rheumatic fever, and show a tendency to enlargement and thickening. It is probable, I believe, that some of these cases pass on to become indistinguishable from rheumatoid arthritis, and, thus, the difference is reduced to a question of terms for this minority. There should be no insuperable difficulty in ascertaining whether rheumatic fever is an antecedent of rheumatoid arthritis; but a practical inquiry on this point I find very difficult. It is easy to elicit a history that will suffice to satisfy superficial inquiry, but the answer thus obtained is far removed from the position of a medical fact. Thus, it is common to get a history of an acute attack which lasted

[1] The miasmatic theory of rheumatism has not been applied to this disease.

weeks or months, which happened either decades of years previously, or only just before the obvious symptoms of rheumatoid arthritis began. Stress is laid upon the occurrence of migratory pain and sweating as leading and confirmatory symptoms, but these are not really trustworthy in most cases. Of far greater importance is the fact, admitting of no doubt, of cardiac damage in any case. This may fairly be taken as unequivocal evidence of previous rheumatic fever, and it is met with in a small percentage of cases. This being so, we may confidently affirm that these patients are of rheumatic habit. But how is the argument for the proposition that "rheumatoid arthritis is evolved out of acute rheumatism" affected by the results of careful inquiry into this point? Most materially, as I think, and in a sense contrary to that view. It is not often that the joints are examined in the bodies of those who have suffered from rheumatic fever. For some years past, however, my colleague, Dr. Norman Moore, our Lecturer on Morbid Anatomy, has habitually made this examination, and he informs me that he can find no evidence of any kind to indicate the previous influence of rheumatic fever on the joints. The rule is, that the process subsides entirely, leaving all the articular structures in a natural condition. The cardiac damage, if any, remains; the joint-mischief is, as a rule, temporary, and passes off completely.

As Dr. Moore examines the joints in nearly every *post-mortem* investigation which he makes, his evidence is obviously of the greatest value. Judging by the light of these facts—namely, the extreme difficulty of securing trustworthy history of past acute rheumatism, and the absence of joint-mischief in cases that have suffered from it unequivocally—we are in a position to affirm that the opinions of those who believe rheumatoid arthritis to be an evolution of rheumatic fever in many instances is erroneous. The most that can rightly be said is that, in a small proportion of the cases, there is evidence of past rheumatic fever. As I shall show later on, this evidence is not without value, and aids us in a proper conception of the malady in question. It is probable that the illness so often described as rheumatic fever is an acute beginning of the disease now well-advanced. I could array a list of eminent authorities in support of this first proposition, and of others who regard it, with myself, as only occasionally true.

If the foregoing view be held, on the understanding that rheumatic fever, as a rule, is but a rare antecedent in the disease, I have otherwise not much objection to offer to it. I consider, however, that the term chronic rheumatism insufficiently expresses

all the peculiarities of this disease, and I would rather call it *a form* of chronic rheumatism, since the true nosological relation of the disorder is thus expressed. Still I think a better definitive term may be found.

2. The *second* view is, that rheumatoid arthritis has no direct relation to rheumatism, but merely resembles it in some points; that it has no relation to gout, may exist with or without any rheumatic or gouty tendency, and is not antagonistic to either, partaking rather of the nature of a senile change, and induced by wear and tear of joints. I cannot accept this doctrine. I have already adduced some evidence of a relation to true rheumatism, though, it is true, this relation is not proved to be direct, save in a few instances. The resemblance, then, can only be in respect of the arthritic affection; but I have shown that, beyond the fact of inflammation, in the one case acute and temporary, and in the other chronic and persistent for the most part, with acute phases, there is nothing in common between the two conditions. Hence, I admit this view, which has been very ably set forth by so careful a thinker and observer as Dr. Pye-Smith, only in respect of its recognition of this disorder as a distinct one, and not in direct relation to either rheumatism or gout.

That it exists without any rheumatic or gouty predisposition or tendency, I dispute. If by this is signified that the ordinary characters of acute rheumatism, or of true gout, are not present in the ordinary forms of the disease, I assent; as I do, also, to the further statement that rheumatoid arthritis is not antagonistic to the occurrence of true rheumatism or of true gout. That the specific changes induced by the disease have the characters of senile degenerations in joints is a fair proposition, but a proper conception of the whole disorder includes the articular disorganizations known as *malum senile* and single-joint rheumatism; and many premature textural degenerations in the body may be termed senile—to wit, atheroma in a child's artery. From this point of view we get no light in the inquiry. We must make larger and deeper generalizations.

3. We come next to the *third* view, that the disease is neither rheumatic nor gouty, but occupies an intermediate place between the two. I quote now the late Dr. Fuller's well-known opinion. Dr. Fuller employed the term "hybrid" in the first edition of his book, but omitted it subsequently. It was incumbent on him to do so, for a malady could not be a *hybrid* which, according to him, was "not a compound of the two diseases" in question.[1]

[1] Fuller, On Rheumatism, &c., 3rd edit., 1860, p. 331.

For Dr. Fuller, rheumatoid arthritis was something "essentially distinct from both," "closely allied to rheumatism while presenting some of the features of gout." Although denying that the disease had "any sort of connection with rheumatism," Dr. Fuller declared that he "had repeatedly known patients crippled by rheumatic gout, which commenced, in the first instance, as a sequel of acute rheumatism." Surely, this was a contradiction. The experience of most observers coincides with that of Dr. Fuller, but their deduction is different from his. Sir Alfred Garrod allows that this sequel occasionally happens, but guards himself by adding, that error may arise from mistaking the acute stage of the disease for true rheumatic fever. So far as I know, only two authorities give their experience from the other side. Thus, Sir Benjamin Brodie declared that in most of the cases occurring in the affluent classes the disease seemed to be of gouty origin; and Dr. Fuller believed that rheumatic gout might arise in persons who had been or might become truly gouty, but that there was no connection with either disease. To justify the term "rheumatic gout," I think proof is required that rheumatoid arthritis may be set up as well by gout as by rheumatism. I do not deny the possibility of this, but I can nowhere find proof of it. I believe, with Garrod, that most of Sir Benjamin Brodie's cases were truly gouty in their nature, and not unequivocal instances of rheumatoid arthritis. Dr. Fuller believed also in the existence of a specific poison as the agency of this disease, distinct from, though allied to, that of rheumatism and gout. He further described cases in which lithates of soda and lime were deposited on the cartilages, as instances of rheumatic gout in which the symptoms had been gouty during life.

To sum up: Dr. Fuller believed that rheumatoid arthritis was a specific disease, and that it might coalesce with rheumatism or with gout. He strongly insisted on the aptness of the term "rheumatic gout," remarking that "the titles of diseases are seldom used to indicate their pathology, but rather as a means of establishing their identity." We may hope for better things at the present day in this respect, and our effort is plainly to discover first what is true, and then to apply the best name we can find to express the knowledge we have acquired. The reason for my objection to this term will appear in discussing the next proposition.

4. The *fourth* view is that which has been set forth by Mr. Hutchinson, who indicates the use of the term "rheumatic gout" as exactly expresssing the nosological position of the disease in question. For him the whole matter is extremely simple. He

affirms that rheumatism and gout may mix in any proportions, as may spirits and water, and that the common malady, rheumatoid arthritis, is in most cases actually such a mixture. I cannot accept this teaching, and regret to join issue here with one for whose genius and clinical powers I have the deepest veneration. In combating this view, I take first as illustrations of the disease cases occurring in Ireland, Scotland, Holland, or indeed anywhere but in England proper, and I ask, Where is the gouty element or factor forthcoming in such patients? Gout is all but unknown in these countries, and the deduction necessarily follows, that here, at any rate, there can be no mixture of the two diseases. Mr. Hutchinson's reply to this objection is, I believe, that we have no right to affirm that uratic deposit is the only significant token of unequivocal gout. This is, however, the doctrine of the schools. When such deposits exist, we have unmistakable evidence of true gout. Now, in the majority of cases of pure gout this evidence is only forthcoming in the dead-house. The researches of Dr. Moore prove that they are often present when unsuspected, and that only very careful search will sometimes find them. No outward token of arthritis is present in many cases; certainly no indications of rheumatoid arthritis are found, as a rule, in such instances.

If it is declared that uratic deposit is no longer the criterion of true gouty disease, we are placed at once in a difficulty. For my part, I think we are not at present in a position to affirm more than this, that, in a given case of gout we may find uratic deposit in certain joints and parts, and evidence of more or less arthritis, such as erosion of cartilage and everted articular edges, in other joints.[1] I am quite convinced that uratic deposit is not the sole token of gouty arthritis, for I recognize ulceration of articular cartilage as almost equally significant in the same joint, or in others, without deposit. But I also maintain that gouty arthritis, which, be it remembered, is only one of the manifestations of gouty disease, never produces all the specific lesions of rheumatoid arthritis.[2]

It may sometimes closely simulate them during life, and render the diagnosis difficult, and at times impossible. Thus, there may be every external sign commonly recognized as

[1] Bristowe, Theory and Practice of Medicine, chapter, "Gout." W. Gairdner declared that he had seen many cases where urate of soda did not remain as enduring evidence of each attack of gout; but as no *post-mortem* evidence is given, no value attaches to this statement.

[2] Virchow has expressed the opinion that the one has no connection with the other. (On Nephritis Arthritica, *Berliner klinische Wochenschrift*, 1884. No. 1.)

peculiar to rheumatoid arthritis: knotty and dislocated joints, with and without effusion, crackling, eversion of fingers to ulnar side; and not only the signs, but the symptoms of rheumatoid arthritis. Such patients as have suffered both from gout and rheumatism can, however, usually tell of a difference in the respective pains and symptoms of each. I have met with such patients, and Scudamore gives particulars of one.[1] I believe firmly that cases of true gouty arthritis are frequently mistaken for rheumatoid arthritis, and Dr. Fuller's later experience led him to a similar opinion.[2]

The converse error is, perhaps, less frequently fallen into. Hence, for a correct determination in any case, that clinical acumen which Mr. Hutchinson so much deprecates, and which he thinks only leads us into error, must be applied. Until we have more knowledge, we must hold by certain symptoms and signs. Gout must signify the presence of uratic deposit somewhere in the body—I do not say necessarily or only in the joints,—or excess of uric acid in the blood; rheumatoid arthritis must signify destruction of synovial membrane and cartilage, ulceration of bone, eburnation, and osteitis; and acute rheumatism must be held to do no permanent damage to joints affected by it. I referred just now to Heberden's nodes as a form of rheumatoid arthritis. I have met with them in cases of pure gout. On dissection, I have found no uratic deposit in the joints, but only a small quantity in the investing ligaments. There was no appearance of tophi. The phalangeal tubercles were enlarged exactly as in cases of rheumatoid arthritis. The gouty nature, suspected only during life, was made certain after death, and hence I cannot accept Heberden's dictum that they are never gouty.[3] Clinical study of gouty and other forms of arthritis, carried on with an open mind, has convinced me that gouty disease will often simulate some forms and phases of rheumatoid arthritis, and I do not require to invoke any

[1] This case is very important, and worth quoting. A man, aged thirty, had two attacks of rheumatic inflammation of ankle-joints, with flying pains in different parts of the body. Two months afterwards, being in improved circumstances, and having lived indulgently, gout came in one great toe and instep, with shiny skin and swelling, deep red, then purple colour, and finally desquamation. No intermission in the pain was experienced in the rheumatic attack; but in the gout the worst pain was felt from twelve to three A.M., and about five he procured sleep. The character of the pain differed in the two disorders. The man's father was gouty; his only sister suffered severely from chronic rheumatism. *Op. cit.*, p. 168, 3rd edit., 1819.

[2] *Op. cit.*, p. 44.

[3] "Nihil certe illis commune est cum arthritide, quoniam in multis reperiuntur, quibus morbus ille est incognitus."—*Commentarii, De Nodis Digitorum.*

element of the latter malady to explain all the phenomena. I am aided in my diagnosis by other clinical considerations. The dead-house is not my only appeal, for it can only tell me a part of the whole story. In any given case, Mr. Hutchinson would say, All that is not obviously gouty is rheumatic. He remarks also that it is very difficult to say how far rheumatism pure can go. I would add, and equally difficult to say how far pure gout can go. This difficulty as to the gouty element has never much troubled clinical inquirers out of England. This is surely a significant fact, and I might add, further, that it has never much exercised clinical inquirers outside London. Now, London is the head-centre of gouty disease, and there is, probably, more gout and goutiness in London than in any other spot on the globe. Hence, our peculiar difficulties and perplexities in eliminating its influence in the arthritic affections we have to deal with. It affects our poor as much as our affluent classes. Scottish, Irish, and foreign immigrants come to London presumably innocent of all gouty taint, and grow gouty in the great metropolis. This is unquestionable, and in these persons we can show cases of unequivocal uratic gout, which we may firmly believe would never have developed in their original countries. London life and habits have to answer for this, be they what they may. The result of this has been to complicate cases of rheumatoid arthritis, and to lead many observers in England into errors which have not misled observers elsewhere.

Can there, then, be a mixture or coalescence of gout and rheumatic disease? I reply, Yes. It would be very remarkable if there were not. Such a mixture, a veritable hybrid, does occur. Clinical observation and *post-mortem* search prove this. Museum specimens, not many in number, however, attest the fact.[1] Gout may supervene on rheumatoid arthritis, and rheumatoid arthritis may come on in a gouty subject, and plain tokens of both will be manifested. There is no antagonism, as most good observers have remarked.

Mr. Hutchinson appeals to the experience gained in those stores of clinical information, the London Workhouse Infirmaries, an experience which has convinced him of the co-existence of rheumatic and gouty disease. I have also availed myself of this experience; but I read the story differently, and would venture the opinion that even in London practice it is possible to make exact diagnosis in the majority of these cases. I feel sure that the frequent occurrence of various forms of gouty arthritis in

[1] Dr. Adams records only one instance in his experience. The preparation is in the Museum of Trinity College, Dublin. *Op. cit.*, p. 309.

London has led to error, and to the inclusion of purely gouty cases in the category of rheumatoid arthritis. I do not agree with Mr. Hutchinson that it is proper to call all that is not plainly gouty, rheumatic.

Gouty arthritis will produce lips on the ends of bones, crackling, chronic synovitis, and other features commonly—but erroneously, as I believe—supposed to be alone significant of rheumatic disease. As I have already remarked, observers in Dublin and elsewhere have fewer of these difficulties. These peculiar perplexities are rather of English origin, and, without doubt, more cases of rheumatic and gouty coalescence are met with here.

I am entirely in accordance with Mr. Hutchinson's views as to the basic, so-called arthritic, diathesis on which these two maladies rest. And with respect to the modifications induced by the comminglings of these states, or of other diatheses, such as the strumous, all of which must be taken into consideration of a large question such as this, I am also in accord with Hutchinson, and think his view most suggestive and philosophical. The teaching of Laycock and of Paget on this point has never yet had its full recognition.

It must be remembered, however, that a trustworthy ancestral history of either rheumatism or gout is most difficult to secure, and often fallacious in all classes of patients, and especially in the lower orders. It seems certain, as Paget has shown, that latent tendencies often exist. Death may supervene before they are manifested, or they may only come out very late in life. The offspring will inherit the parental or ancestral tendency, and may develop it early. Or, again, far distant ancestral tendency may only come out in later generations.

Mr. Hutchinson affirms that so long as an arthritic person has sound digestion and healthy kidneys, his rheumatic manifestations will be free from gout; but once let him fail in these respects, and it is scarcely possible for him to have a rheumatic inflammation which is not modified and made gouty by the previously existing peculiarity of his blood. My belief is, that many of these cases continue free from gouty development in spite of failing renal and digestive organs, and that gout is not always, or often, waiting on rheumatism.

Heberden remarks, "It must be owned that there are cases in which the criteria of both are so blended together that it is not easy to determine whether the pains be gout or rheumatism;" and again, "These two distempers, though of the same family," &c. ("hos morbos, cognatos sane," *op. cit.*).

With respect to the humoral theory of rheumatoid arthritis, I think we have as yet no evidence of the hypothetical specific poison conceived by Dr. Todd and Dr. Fuller as the causative agent in this malady. So far as we have gone, we have established a place for rheumatoid arthritis outside rheumatic manifestations, and outside gouty disease. Is it possible to correlate the three diseases? I believe it is, and in doing so I must bring forward more of the teaching of Mr. Hutchinson and others respecting these disorders in general. Before proceeding to this, however, I must discuss the fifth view which is held.

5. The *fifth* view, that the disease is of nervous origin, is the latest that has been presented. Mr. Hutchinson's opinion that rheumatism is a catarrhal neurosis, or reflex nervous inflammation, leads up to the stronger expression of neurotic origin here laid down. The evidence for this is based upon the fact that joint-disease has been observed to follow upon lesions of the spinal chord. Thus effusions and painful arthritis have been noticed in cases of hemiplegia with descending degeneration in the chord, and a well-marked form of arthritis has been recognized in connection with tabes dorsalis, sometimes described as Charcot's disease.[1]

Sceptics in medicine, who, by the way, seldom advance any theories or suggestions of their own, may scoff, if they please, at the attempt to connect the two conditions last-mentioned, or decline to make up their minds while opinions oscillate between humoral and neuro-trophic theories of rheumatoid arthritis; but I think it is proved to the conviction of most impartial clinical observers, certainly amongst physicians, that there is a form of degenerative arthritis associated with tabes dorsalis. This is not the place to adduce all the arguments in favour of the specific connection, and I shall do no more than meet with a denial the assertion that the joint-disease of tabes dorsalis is commonly due to local injury. I am firmly convinced of the existence of the disease as a special form of arthritis, and I would ask whether it was in any degree likely that the man who did some of the best clinical work on the subject of rheumatic and gouty arthritis five-and-twenty years ago, would at a later period fall into error on a matter of observation forced upon him by larger and more special experience. It is noteworthy that surgeons have mostly refused credence to Charcot's views. This is perhaps not unnatural. Here, however, I am only concerned to note that the characters of spinal arthropathy are

[1] *Vide* Art. by myself on "Charcot's Disease," Heath's Dictionary of Practical Surgery, vol. i. p. 272.

clinically different from those of ordinary rheumatoid arthritis, and that the main differences consist in the sudden onset of the disease, the extreme effusion into the joint affected—a symptom which has been erroneously denied as common in rheumatoid arthritis—the rapid absorption of the ends of the joints, and the remarkable fragility of the bones.

The trophic changes run riot, so to speak, and such alterations as these seem to point plainly both to different ætiology and progress.

Opponents of the view that there is any relation between the spinal lesion and the joint-changes, conceive that there is nothing remarkable in the onset of ordinary rheumatoid disease in a certain proportion of cases of tabes dorsalis.

Those who adhere to the view that rheumatoid arthritis is a tropho-neurosis refer to its frequent onset after nervous shock, depression, and grief; and the possibility of direct injury to nerve-roots by the mechanical changes induced by spondylitis is also conceived. For my part, while fully recognizing tabetic arthropathy as a spinal lesion, I do not find evidence to warrant so large a deduction as that the disease which we know as rheumatoid arthritis owns thus directly, and always, a similar, or even kindred, lesion. Many features of the common form of the disease are perhaps best explained on a nervous basis, and these will be better taken note of in discussing the sixth and last view.

Not to do more than allude to the fact that in Charcot's disease it is commonly a large joint, such as the knee, shoulder, or elbow, which is affected, I may mention that in these cases we do not readily find the special diathetic characters proper to the rheumatic habit impressed upon these subjects, and for me this is a matter of much significance. I do not think there is any evidence to support the view that Charcot's disease is a new manifestation. I imagine that it had been previously overlooked, and that the specific relations of the disorder had not been recognized.

6. According to the *sixth* view, rheumatoid arthritis is neither of rheumatic, gouty, scrofulous, nor of any specific origin, and not justly separable as an independent disease, but is a lesion common to several kinds of ailment. This is the view of Dr. Ord, who has endeavoured to show that this disease is the result of a lesion of the spinal chord set up by peripheral irritation of various forms and degrees, and that a condition of exalted susceptibility and reflex activity of the chord must enter into our conception of

it.[1] Denying the humoral theory, he proposes a neurotic one, and contends that we meet with the disease as a result of primary lesion in the chord, and as also resulting from such varied irritations as gonorrhœa, simple urethritis, ovario-uterine troubles, traumatism, acute rheumatism, chronic gout, and foreign growths in joints. He proposes to discard the term rheumatoid, and to apply in each case such an one as will express the form of the exciting cause, such as traumatic, blenorrhagic, urethral, rheumatic, hystero- and myelo-arthritis. He believes, further, that the disease may spread from joint to joint symmetrically by reflex nervous influence. This view is very philosophical, and constitutes a distinct advance in our conception of the disease. It appears sufficiently comprehensive, and it is not easy to controvert. My chief objection to it lies in this, that too little regard is paid to the inherent, and often latent, tendency to some form of arthritic disturbance in certain persons. If there is not something special about the individual, these alleged sources of peripheral irritation, which are sufficiently common, should surely affect indifferently all persons exposed to them; but this is not the case. The specific changes of rheumatoid arthritis are only induced in the diathetically-predisposed of the community.

There is an inherited something, or a superinduced something, which favours the particular evolution in the particular person. It is this specific vulnerability which, in my view of the matter, marks off the individual for an onset of arthritic trouble—just as much so, indeed, as does the special vulnerability of a person of strumous or tubercular tendency lay him open to the onset of active strumous or tuberculous trouble, when specially exposed. Hence, I join issue with Dr. Ord in discarding the term rheumatic, or rheumatoid, in this disease—holding, as I do, that these manifestations are implanted on persons with specially impressed vital tendency. I do not believe that the rheumatic diathesis is universal, as has been alleged. Some persons will never develop rheumatism in any form, however much exposed to exciting causes.

I can therefore conceive an imaginary person, of perfect health and constitution, upon whom no one of the indicated peripheral irritations shall reflexly set up such changes as we recognize in rheumatoid arthritis.

I think the evidence is strongly in favour of Dr. Ord's view, that extension of the disease and symmetrical implication of joints are often of a truly reflex character; and if this be a true explanation, it favours a neuro-trophic theory of the disease.

[1] British Medical Journal, January 31, 1880, p. 155.

Amongst less well-recognized causes leading to these changes are chronic dysentery, and arterio-capillary fibrosis with contracted kidney, the latter connection having been pointed out by Lancéreaux, but this has not yet been observed, perhaps because not looked for, in this country.[1]

And now to sum up.—To what view of the nosological relations of this disease are we led by a consideration of its characters? I find that some of the best thinkers confess themselves baffled for the present in the attempt to settle the question. Much as I could wish to lay down a theory that would command universal acceptance, I fear I can do no more than help to clear the way for further observation. I think we are only now in a position to advance with more rapid and certain steps than our predecessors. We have the advantage of an exact anatomy, we have fairly well-determined the characters special to definite forms of arthritis, and we have now to gather facts in family and clinical history which shall put us on the way to the goal we seek to reach. Collective investigation, properly conducted, will do much to help. Family life-histories, accurately recorded, will do more. But is this all? I think there yet remains the modern study of the modifications and transformations of disease, the effects of time, of locality, of habits of life. And this large question faces us here as we try to form a true conception of rheumatoid arthritis as we meet with it to-day. We may say that in a majority of cases we find the same changes induced now as were met with in ages past; but it may be that forms of arthritis occasionally come before us which differ from those observed by our predecessors, and that forms long-suppressed may again crop up, as it were, and be with difficulty relegated to their proper place. And, so, disease in a pure form may come sometimes to be modified, and give rise to varieties for which no place is readily found. It seems only too likely, for example, that in England the coalescence of rheumatic and gouty diseases has produced a mongrel type of malady in many instances—not a mere mixture, but a new type which may propagate itself, and that, thus, may arise some of our perplexities.[2] Such propagation is not likely to be enduring. If hybrids do not breed, mongrels certainly may. There are, however, facts to support

[1] Transactions of the International Medical Congress, London, 1881, vol. i. p. 384.

[2] Mr. Hutchinson has directed attention to various maladies affiliated with what he terms rheumatic gout and gout, but differing somewhat from both, and these include various eye-troubles, such as iritis, hæmorrhagic retinitis, and some forms of glaucoma, lumbago, sciatica, chronic rheumatoid arthritis, Heberden's nodes, and, possibly, hæmophilia.

the belief that while distinct types prove stable and enduring, mongrel progeny is not so; and hence we may fairly conceive that a strongly-marked and distinct disease such as rheumatoid arthritis, owning a much older ancestry than gout, will constantly tend, even amidst potent modifying conditions, to revert to its pure type. Still, we must be prepared to meet in practice with mongrel forms of disease. The immunity from chronic rheumatism enjoyed by light-haired men, if it be true, demands attention and close study.[1] Careful and prolonged clinical observation alone will help us here.

It is remarkable that we should still be ignorant of the measure of heredity of this disease. The difficulty, of course, is to get trustworthy family history, and to go back far enough for this evidence. My own experience has furnished in most cases a clear history of rheumatic ailments, or of arthritic disease, in the ancestry. I have observed, and Garrod and others inform me their experience is similar, that the daughters of gouty men not infrequently become the subject of rheumatoid arthritis. What is the proper explanation of this? Is this a transformation of gout in the female, or only a separate manifestation of the arthritic diathesis? The latter view alone commends itself to me, and, to explain the fact, it would be necessary to secure a family history extending over many generations. Until I can secure this, I prefer to accept the theory of a basic arthritic diathesis, which explains a relationship, though indirect, between the two disorders. The facts elicited by study of the heredity of rheumatoid arthritis, though somewhat perplexing in themselves, tend strongly, in my opinion, to support the view that the disease is a manifestation of the arthritic diathesis. Objection is made to the inclusion of gout as a branch or offshoot from the parent arthritic stem, because so many of the manifestations of it are non-articular. I am firmly convinced of this latter truth, but I venture to think that the arthritic phenomena of gout are so strongly impressed on the subjects of it, that objection to the view suggested cannot fairly be raised. The idea connected with an arthritic diathesis implies tendency to disturbance of motor structures and the nervous centres regulating them, and hence physiologists conceive the possible existence of a trophic nerve-centre, or centres, for joints. The differentiation of Charcot's joint-disease has lent support to this conception, and, so far, there is no evidence to disprove it. Minute examinations

[1] Statistics published by the American War Office in 1875, under the direction of Dr. Baxter, and quoted by Mr. Francis Galton.

of the spinal chord have yet to be made, in order to certify the existence of any definitely associated lesion in rheumatoid arthritis or other arthritic diseases.

As a rule, I think it may be affirmed that rheumatoid arthritis is commonly seen unassociated with other diseases. Dr. Sutton has called attention to cases met with in young persons where this disorder seems to be associated with phthisis and insanity in the family, and believes that there is some special relation between these conditions. He is, further, of opinion that there is relation, not as yet recognized sufficiently, between rheumatoid arthritis, rheumatic arthritis with heart-disease, also gouty arthritis and insanity. He thinks that these diseases have been too much regarded as definite entities, and that thus we have been blinded to their correspondence.

Charcot has noted the not infrequent association with scrofula and pulmonary phthisis in the family and collateral relatives of patients, also the frequent coexistence of Heberden's nodes with mammary and uterine cancer. In all these coincidences I see no more than the blendings and inevitable coalescences of diatheses.

The strumous condition may readily modify the arthritic, and the arthritic determinations of the former are sufficiently well-recognized. M. Charcot's and Dr. Sutton's cases may be explained, I think, by the occasional coalescence of other taints, such as struma or cancer, or of other inherited neuroses with the arthritic predisposition.

The association of diabetes is certainly rare. Garrod has recorded one case, and informs me that he has seen others in which glycosuria occurred, and he conceives that this may aid the development of the rheumatic affection. Dr. Ord and I[1] have recorded instances. Lancéreaux has observed the coincidence but rarely. Charcot has never met with it. The association of glycosuria in the gouty branch of the arthritic stock is, on the contrary, well-marked, and constitutes a determining symptom.

I have not observed a frequent occurrence of psoriasis with rheumatoid arthritis, as has been noted by Garrod, though the skin-disorder is certainly in itself a manifestation of arthritic disposition.

The views concerning rheumatoid arthritis which commend themselves to me may be set forth in the following series of propositions:—

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[1] See "Diabetes in Relation to Arthritism," St. Bartholomew's Hospital Reports, vol. xviii. 1882, p. 371.

That there is a basic arthritic stock, or diathesis, from which arise as branches two main classes of disorder, commonly recognized as rheumatism and gout.

That rheumatoid arthritis is one of several manifestations of this diathesis, and should be regarded as a rheumatic branch of this stock, and, therefore, a true rheumatism.

That this nosological position necessarily entails indirect relation with all forms of rheumatism and gout.

That although the disease has indirect relations with other branches of the arthritic family, its occurrence is not at any period antagonistic to the onset of other phases of rheumatic and gouty disorder. Albeit, the disease is commonly met with in a pure form, and uninfluenced by other arthritic manifestations.

That rheumatoid arthritis occurs specifically in more or less grave form, and may also be developed symptomatically by certain special agencies.

That arthritically disposed persons are peculiarly vulnerable and sensitive to changes of temperature, soil, and climate, and manifest this for the most part by certain trophic changes in the joints.

That gouty manifestations may supervene independently in the subjects of rheumatoid arthritis, or may coalesce with rheumatic conditions; and that, in the course of many generations, transitional modifications may occur, and give rise to unusual forms of arthritis whose place is not quite readily determined.

That some of these irregular forms may be due to coalescence with other inherited diathetic states.

That accurate family and clinical histories are essential for accurate diagnosis in any case.

That arthritic persons are more than others sensitive to urethral irritation, gonorrhœal, and, perhaps, some other specific poisons, and also liable to certain forms of inflammation of the eye; but that these troubles are distinctly more common in the gouty than in the rheumatic branch.

That heredity is a strongly marked feature of the arthritic diathesis, and that gouty or rheumatic affections may supervene in the descendants of either rheumatic or gouty persons.

That local forms of rheumatic disease plainly indicate the underlying rheumatic habit of body, which may have previously been latent.

That there are probably allied forms of rheumatism with various manifestations; for example, that state expressed merely by the occurrence of nodules in the skin, fasciæ, periosteum, sometimes with, and sometimes without, associated carditis.

That although there is an indirect relationship between rheumatism and gout, the two diseases are remarkably distinct from each other, as well as from rheumatoid arthritis.

That the nervous system is markedly implicated in the arthritic diathesis, and that many of the features both of rheumatic and gouty diseases point to the probability of there being a trophic centre for the joints situated in the spinal chord; and that a morbid or unstable condition of this centre may result in a definite neurosis, which may be either inherited, acquired, or modified.

That it is not necessary to conceive of the perverted chemical conditions, so far as they are discoverable, in either rheumatism or gout, as other than epiphenomenal, and constituting but a part of the dynamic state induced by these maladies.

That the term "rheumatic gout" should be expunged from our nomenclature, and "chronic rheumatic arthritis," the term proposed by Robert Adams, be employed in its stead.

2.—The Relation between Gout and Lead-Impregnation or Saturnism.

The connection between lead-poisoning and gout is well-established, although the nature of that connection is still but little understood. It is not too much to assert that the facts adduced on this subject by Sir Alfred Garrod, first in 1854, and subsequently added to by him, have been fully confirmed by other observers, and the merit of fully setting forth the connection between the two disorders rests with him, although previous indications of it were made known more than a century ago.[1] Garrod states that "at least one in four of the gouty patients who had come under his care in hospital had at some period of their lives been affected with lead, and for the most part followed the occupation of plumbers or painters."[2] In 1870,[3] he stated that 33 per cent. of people who suffered from gout had

[1] Dr. William Musgrave is believed to be the first writer who directed attention to *arthritis* in connection with *colic*. He did not, however, attribute the latter to the influence of lead, but thought that cider-drinking induced it. A perusal of his chapter *De Arthritide ex Colicâ* in his *Dissertatio de Arthritide Symptomatica* makes this clear. This was published in Exeter in 1703. It was left for the acumen of Sir George Baker to discover, sixty-three years later, that lead-impregnation of cider was the real cause of Devonshire colic, a fact which he disclosed in an essay read in the College of Physicians on June 29, 1767.

[2] Clin. Lect. on Lead-Poisoning, Lancet, 1870, vol. ii. p. 781, and Reynolds' Syst. of Med., vol. i. p. 841, Lancet, 1872, vol. i. p. 1.

[3] Gout and Rheumatic Gout, 3rd edit., p. 237.

been poisoned with lead. These facts are very remarkable, and are probably insufficiently realized. Such an experience is not readily procurable, and London practice affords perhaps the only field in which such a study is possible on a similar scale.[1]

The connection between lead-influence and gout, whatever it may be, is naturally to be studied almost exclusively amongst the artizan classes, and therefore in hospital practice. Lead-impregnation is now, happily, very rare amongst the upper classes, owing to proper care in the storage and supply of potable water. Yet even here this influence should never be lost sight of in any case. Amongst the artizan population of London it is common to meet with cases of lead-poisoning, but the worst cases are seen in the workers in lead-mills. These persons are generally very poor, and only resort to this occupation when other means fail them. I find that they are often Irish, and that many women are amongst them, and it is not without importance to note these facts.

My own experience is taken from a series of 136 cases of unequivocal gout in both sexes, which came under my care some years ago amongst the out-patients at the Hospital. Twenty-five of these patients, or 18 per cent., presented signs of lead-impregnation, and followed the occupation of painters, plumbers, compositors, or workers in lead-mills. They were all males. The age of the youngest was twenty-five, of the oldest sixty-two, the mean being about forty-three years. In seventeen of the cases there were either present, or there were histories of, blue line on the gums, colic, and wrist-drop. In at least one-half of these patients, there was history of intemperance, commonly in both malt liquors and spirits. In at least one-half, the urine was slightly albuminous, of low specific gravity, and there were histories of cramps in the legs and of nocturnal micturitions—all symptoms of chronic interstitial nephritis.

These cases are taken from my note-books, and under the pressure of hospital work facts of lesser importance have been sometimes omitted. They were recorded for no special object, and simply to illustrate the varied phenomena of gouty disease. The percentage of saturnine gout is large and remarkable, but it is considerably under that recorded by Garrod, viz., 18 against 33 per cent. of all cases of true gout.

[1] *Vide* Cases in the Acute Rheumatism and the Gout, by Thos. Dawson, M.D., late Physician to the Middlesex and the London Hospitals, Lond. 1774, p. 83. Case of a glazier and painter, gouty from nine years of age. Dropsy and asthma followed. Dr. Dawson suspected the deleterious quality of the lead as laying the foundation of, or at least aggravating, the complaints.

Garrod sought to ascertain how far his views were borne out by experience obtained in other places, and he quotes the evidence of Sir Robert Christison, which showed that both lead-poisoning and gout were practically unknown in the Edinburgh Infirmary.

I have endeavoured to gather some new facts in reference to this matter, and now communicate the experiences of several eminently competent observers in various cities and manufacturing centres.

In Edinburgh, Professor Grainger Stewart finds that the same immunity both from lead-poisoning and from gout still prevails, and he thus confirms Sir Robert Christison's evidence in reply to Garrod's inquiries in 1859.

He remarks, "Although I see a great deal of gout in my consulting-room here, I do not find it increasing among the Infirmary patients—indeed, I scarcely ever get a case. I may, however, say that during the years I have been in practice, I have gradually gleaned evidence enough to satisfy me of the correctness of the view which was believed in by Warburton Begbie and others here."

It is interesting to point out that the views of Sir Robert Christison, as expressed to Garrod in 1859, did not meet the approval of the late Dr. Warburton Begbie, for, three years subsequently, he denied the great infrequency both of lead-poisoning and of gout in the same sphere of observation—to wit, the Edinburgh Royal Infirmary. He published the particulars of two cases fully illustrating the connection, and declared that he had met with about twelve of the kind in the course of seven years. In both of his published cases there was history of intemperance in spirits as well as in malt liquors.[1] He believed that lead-impregnation, together with the employment of fermented liquors, gave strong predisposition to gout.

Professor Gairdner, of Glasgow, writes that his experience is entirely negative. He says, "I never saw a case of lead-poisoning in association with gout having its *genesis* in Scotland. I will not say that my experience in this matter is to be taken as absolute; only, as lead-poisoning and gout are *each* rather rare in the working-classes here, the combination is, of course, still more uncommon. I have no doubt of the London facts, also little doubt that beer is a large factor."

Supposing that lead-impregnation must be common at Newcastle-on-Tyne, I addressed my friend Dr. Drummond, who is

[1] Edin. Med. Jour., August 1862, p. 125.

physician and pathologist to the Infirmary there, with reference to any gouty prevalence. He replied as follows:—"I think I may state very positively that in Newcastle and district, where we meet with a very large number of cases of lead-impregnation, we never see gout associated with that condition. Such is my own experience, and I have given a great deal of attention to lead-poisoning, having ample material in the Infirmary to draw from. It is also the experience of Dr. Embleton, our consulting physician, and for a long time medical officer in charge of nearly all the lead-factories in the neighbourhood. We see lead-kidney (granular), lead-encephalopathy, fits, optic neuritis, optic atrophy, lead-palsies of upper and lower extremities, lead-colic, and lastly, lead-arthralgia; but I have never seen anything like gout in a lead case. The arthralgia has always appeared to me to be more of a myalgia than a joint-affection proper. Some of the cases are allied to subacute rheumatism without effusion into the joints, but unlike gout. We do not often meet with gout in Newcastle, and it is very rare amongst the lower orders." As to the liquors consumed by the labouring classes, Dr. Drummond states that a great deal of whisky is drunk as well as beer. "The chemical labourers drink whisky to 'kill the gases,' as they say, but the pitmen drink both ale and whisky. On the whole, I may say that 'halves of whisky' is the favourite drink."

Sir Walter Foster informs me that lead-poisoning is not common in Birmingham, and that very little gout is seen amongst the lower orders.

Dr. Wynne Foot, senior physician to the Meath Hospital in Dublin, states that he is "quite familiar with articular symptoms in painters, plumbers, and others exposed to lead-intoxication." He terms the affection plumbic arthritis, and has come to regard it as a form of spinal arthropathy due to poisoning of the nerve-centres. He has not had any *post-mortem* examination of these cases.

Professor Cuming, of Belfast, reports that his experience is decidedly against the connection, for which he has often looked, and always in vain.

The evidence here amounts to this, that lead is a factor in the production of arthritis, the nature of which is not exactly known. It may be presumed that no manifest gouty characters prevailed, or they would certainly have been noted. Gout is rare in Dublin, although it has been stated to have become more frequent since the lower orders have taken to drinking porter instead of whisky.

In Liverpool, both gout and lead-poisoning appear to be very rarely met with. I have before me the experience of three of the physicians to the largest hospitals in the city, Dr. Cameron, Dr. Waters, and Dr. Davidson, and it furnishes no facts in support of the relationship.

With respect to Paris, we find the evidence of Charcot in 1868 to the effect that "Il existe parmi les *saturnins* quelques goutteux, chez qui l'empoisonnement par le plomb est la seule cause qu'on puisse invoquer." He believes that gout may be developed under this influence alone, but that such cases are rare. He has published one example illustrating this. Lancéreaux has contributed some important facts from his experience at La Pitié. He communicated to the International Medical Congress a series of twenty-four cases of saturnine nephritis, and from this list I find that in over one-third of the cases there was distinct history of gout, or of uratic infiltration of joints. I think, too, that had the joints been examined in all his cases, a still larger proportion of gouty evidence would have been forthcoming. In respect of the dietetic habits of these patients, Lancéreaux informs me that many of them had drunk brandy and absinthe to excess, and were also wine-drinkers. In no case was there history of excessive beer-drinking. These cases plainly illustrate the combination of lead-impregnation and alcoholic excess as factors in the production of gout in a community and country where that malady but seldom occurs. Lancéreaux agrees with those observers who believe that the lesions of gout and, so-called, saturnine gout are identical in all the organs of the body, save that in pure gout there may perhaps be present more uratic deposit; but he does not believe that intemperate habits count for much in the production of urate of soda and of gout, for the reason that his hospital practice yearly furnishes him with hundreds of cases of alcoholic excess amongst which gout is most rarely seen, and when met with, is regarded merely as a coincidence.

It is to be noted that the kidneys were severely implicated in the majority of Lancéreaux's cases.

Dr. Pye-Smith, in a series of sixty-one cases of gout at Guy's Hospital, met with evidence of plumbism in only two instances. He does not find that plumbers and painters admit the common opinion that men in these trades drink more freely than others.[1] (My own observation in London would not permit me to grant this admission.) In these cases there was history of inherited gout or of intemperance.

[1] Analysis of Cases of Rheumatism and Gout, Guy's Hospital Reports, 1873.

It is important to note the influences of lead in cases of gout where no inherited taint is discoverable, and also where no intemperance in strong drinks has prevailed to determine gout. Charcot [1] reports one such case—Todd's [2] was probably of this nature—and Dr. Wilks [3] has recorded three instances. In the great majority of cases there is found history either of predisposition to gout, or of distinct intemperance in malt liquors, or, indeed, of both causes; but none the less is the influence of saturnism very decided and noteworthy.

Dr. Begbie's [4] cases occurred in intemperate men, and the late Dr. Falconer [5] reported another. Dr. Fagge [6] also recorded one. M. Bricheteau [7] recorded a case in a painter whose father had followed the same occupation; and amongst my own cases are three where the fathers were either painters or compositors. In these instances we must regard it as almost certain that predisposition to arthritism existed, or was directly inherited. Great difficulty must always be met with in eliciting ancestral history of gout, especially in the cases of hospital patients; and another difficulty arises from the impossibility of finding certain evidence of lead-taint in some instances, since such may really be present without the manifestations of colic, wrist-drop, or even of the Burtonian blue line.[8] Lancéreaux's cases all occurred in intemperate men.

London experience certainly confirms Garrod's opinion that persons exposed to lead-influence are prone to develop gout, and that persons of gouty predisposition are specially liable to suffer quickly and severely from plumbism. It is remarkable that this experience should not be universal even in England. It is, however, noteworthy that in many cases where true gout is not developed in connection with lead-impregnation, rheumatoid pains and arthritis are apt to supervene, and this fact appears to me

[1] *Gazette Hebdom.*, 1863, No. xxvii. p. 433.
[2] On Gout and Rheumatism, 1843, p. 44.
[3] Guy's Hosp. Reports, 1870, p. 40.
[4] *Loc. cit.*
[5] Brit. Med. Journal, 1861, p. 464.
[6] Med. Chir. Trans., vol. lxiv. p. 221.
[7] *Gazette des Hôpitaux*, 1870, No. xxvi.
[8] My observations entirely confirm Dr. Hilton Fagge's respecting the blue (more correctly black) line. *Vide* Med. Chir. Trans., vol. lix. p. 327. Garrod has claimed the credit of its discovery for Tanquerel des Planches, who published his famous *Traité des Maladies de Plomb ou Saturnines* in 1839. Dr. Burton's communication was read to the Royal Medical and Chirurgical Society in January 1840, but he stated that he first discovered the blue line in 1834, and waited to confirm his observations. He also described articular pains "resembling rheumatism" in lead-impregnation. (Dr. Burton was physician to St. Thomas's Hospital.)

to justify the view held by Dr. Wynne Foot, Lancéreaux, and perhaps others, that the arthritis owns a neurotic origin. And this theory is specially acceptable to me, inasmuch as I am strongly convinced of the neurotic element in gouty disease in general. Lancéreaux believes the pathogeny of ordinary and of saturnine gout to be alike, and holds that they have their common origin in "a primordial trouble of nutritive innervation." He remarks, "Gout is certainly the result of such a disorder, and no one can doubt the obvious action of lead upon the nervous system."

In connection with arthritic changes induced by lead, attention may be directed to a series of cases which were very carefully described in Paris in 1868 by M. Gubler,[1] M. Nicaise,[2] and M. Bouchard.[3] A series of fourteen cases illustrated certain swellings which appeared in the extensor tendons and their sheaths in connection with muscular atrophy and wrist-drop. Sometimes the carpal and metacarpal bones were affected by bony outgrowths, and in several instances arthritis occurred in the metacarpo-phalangeal and phalangeal joints. Gout and uratic deposits appear to have been carefully excluded, save perhaps in one case. The extensor tendons and tarsal bones were also affected in some cases. These swellings, to which M. Gubler gave the name of "dorsal tumour of the hands," were found to occur commonly within two months of the onset of the paralysis. Sometimes they were formed within a few days, and in others not till six months had elapsed. After death, the tendons and their sheaths were found to be nodular and the synovia opaque, and bony outgrowths had occurred, but no uratic incrustation. These tumours entirely subsided in several of the cases, but a good deal of inflammatory disturbance and pain were met with at first. M. Nicaise in his papers showed that similar cases had been noted nearly three centuries ago by Plater, and by De Haen in 1745, also by Pariset in 1813, and by Tanquerel des Planches in 1839. M. Gubler recognized the same affection in one case of hemiplegia due to cerebral hæmorrhage; and in 1869 M. Tournié[4] contributed three cases, the tumours always occurring in the hand of the paralyzed side.

M. Gubler regarded these changes as due to enfeeblement of vaso-motor nerves, and such cases must be considered together with those which are distinctly gouty in their nature, all of them plainly illustrating neuro-trophic derangements. Erb[5] declares his belief that in lead-poisoning there is a primary lesion of the

[1] *L'Union Méd.*, 1868.
[2] *Gazette Méd.*, 1868.
[3] *Gaz. Hebdom.*, 1868.
[4] *L'Union Méd.*, 1869.
[5] Disease of Peripheral Cerebro-Spinal Nerves, Ziemssen's Cyclop., vol. xi. p. 548.

nervous system, mainly spinal, leading to motor-trophic disturbances, and he quotes observations by Remak showing that circumscribed alterations are met with in the anterior cornua of the chord.[1]

The effect of plumbism in inducing arthritis other than gouty, and articular pains, as well as the peculiar swellings in the tendons of the extremities and their sheaths, must be considered in relation to the production of true gout in many cases. These may be held to be of neuropathic nature, and akin to other forms of, so-called, spinal arthropathy.

Lead-taint superadded to already existing arthritic diathesis, or coalescing with ordinary excitants of gout, appears to promote and intensify the evolution of gout.

It has been stated in objection to the theory of any connection between plumbism and gout, that the cases should be more common than they are, and that women should present examples of it. But the cases illustrating the connection in males form a very remarkable percentage of all cases of true gout; and the fact that women are apparently exempt may admit of the explanation that they are seldom persistently exposed, as are men, to lead-impregnation. Women who suffer are commonly employed for short periods in lead-mills. They mostly take up the work in default of other and more wholesome employment, and leave it as soon as they can. Women, too, are less subject to gout during the period of generative activity than men, and they are certainly more temperate in liquors. Amongst my cases, most of the women affected by lead were Irish, and very destitute. Now, it may be affirmed that in such instances there is absence, for the most part, of both the factors of hereditary tendency to gout, and of intemperate habits. I see many cases of gout in Irishmen who have lived long in London, and who have almost certainly acquired the malady as the result of adoption of London habits of beer-drinking. Such men would probably never have become gouty in Ireland.

Women who acquire lead-cachexia manifest all the lesions producible by the metal, save unequivocal gout. They suffer the cardio-vascular and the renal changes very markedly, but the special uric acid perturbations are not found.

The facts relating to lead-impregnation and chronic interstitial nephritis admit of no question in either sex. Lancéreaux draws a distinction between the kidneys of plumbism and those resulting from arterio-capillary fibrosis unconnected with saturnine

[1] Vol. xiii. p. 715.

influence. In the latter, he finds the granulations coarser and more irregular, the arterioles more thickened, and the changes unequal in the two organs. Together with the granular kidneys associated with purely vascular change, he has observed, in a proportion of his cases, certain forms of arthritis quite distinct from gout, and more allied to osteo or rheumatoid arthritis, no uratic deposit being present.[1] The joints chiefly affected are the metacarpo-phalangeal of the thumb and the knees. Such cases have not, I believe, been hitherto differentiated in this country. Where the characters of saturnine nephritis prevail, Lancéreaux has, with one exception, found, when he has looked for it, the arthritic changes characteristic of gout, viz., uratic deposits in the structures of the joints.

In the large number of cases of lead-poisoning carefully recorded by Tanquerel des Planches, it is remarkable that there is no mention of gout.[2] The characters of lead-arthralgia are minutely described, and in frequency it is accorded the second place as a symptom, colic being nearly twice as often met with. Arthralgia was found to be most frequent during the summer season, and to occur more commonly in the fourth decade. The joints of the lower limbs suffered chiefly, while the upper extremities were affected by paralysis. It is specially mentioned that there were never observed heat, redness, or swelling, and that pressure relieved the pain.

The special susceptibility of the gouty to be affected by lead, as asserted by Garrod, appears to be unquestionable. In some cases, lead has induced the first obvious symptoms of gout, having, as it were, precipitated the specific morbid processes of gouty inflammation, and forming a sort of touch-stone for this taint.[3]

A consideration of the physiological action of lead upon the body shows that both the nervous and circulatory systems are profoundly affected. Lead has been found in most of the tissues after death, especially in the brain[4] and in the intestines.[5]

[1] Communication, with specimens of affected bones, to the Section of Medicine, International Medical Congress, London, August 1881.

[2] Intemperance has, however, increased greatly amongst the lower orders in Paris and the large French towns during the last forty years.

[3] My colleague, Dr. Lauder Brunton, kindly permits me to report the following case, which he observed a few years ago amongst the casualty patients:—A man, aged twenty-five to thirty, came under his care for chronic diarrhœa. He was treated with pil. plumbi c. opio. In less than ten days he returned with gout in one of his joints. He had never had a previous attack of gout.

[4] Troisier and Lagrange, *Gaz. Méd.*, 1874, 62.

[5] Fagge and Stevenson, *loc. cit.*, 1880.

Gaffky[1] believes that some change occurs in the vaso-motor nerves of the abdomen, especially in the sympathetic fibres of the splanchnic, by reason of which the renal mischief is induced. Kussmaul and Maier[2] record a careful *post-mortem* examination of a case of chronic lead-poisoning, in which, amongst many other changes, they found proliferation and sclerosis of the connective-tissue septa of the small ganglia of the sympathetic, especially the cœliac and cervical. These ganglia were hard, and the nerve-cells diminished. The smaller arteries were narrowed, and periarterial thickening was widely spread. It is not easy to follow the exact sequence or relation of these changes, but it is known that under the influence of lead the action of the heart becomes slow, and that the arterial tension is raised.[3] It is also now well ascertained that a persistent condition of high arterial pressure is in itself a certain source of cardiac hypertrophy and arterial thickening,[4] and it may well be that much of the mischief wrought by lead-impregnation is set up in this fashion, the particular form of kidney-affection met with in this cachexia being associated with it. The presence of retained matters, such as uric acid, in the blood is certainly often associated with a condition of arterio-capillary fibrosis, and this impure blood has been supposed to meet with resistance in the smaller vessels, and to provoke higher arterial tension in consequence. It is, however, conceivable that this chain of events may result from injury primarily inflicted upon the sympathetic system of nerves by the contaminated blood.

Garrod has demonstrated that lead distinctly diminishes the secreting powers of the kidneys for uric acid,[5] and Charcot[6] likens this inhibitory action of the metal to paralysis of the kidney. The uric acid is consequently retained in the body.[7] Due regard being had to these facts, it becomes easy to see a

[1] *Ueber den ursächlichen Zusammenhang zwischen chronischer Blei-intoxication und Nierenaffectionen*, Berlin, 1873.

[2] *Deutsch. Archiv*, ix. p. 233.

[3] The best and most recent research on lead-poisoning is that of Erich Harnack, published in the *Archiv für experimentelle Pathologie und Pharmakologie*, IXter Band, Leipzig, 1878, p. 152. His experiments go to support the view that the joint-affections, and the nervous symptoms generally, are due to irritation of different centres, those in the medulla oblongata and the brain usually supervening latest.

Naturally, we cannot look for much evidence as to the relation of lead-impregnation to gout from any experiments made in the physiological laboratory; such proof is only to be obtained from clinical studies.

[4] As demonstrated by Dr. Mahomed and other observers.

[5] On Gout, p. 240.

[6] *Leçons sur les Maladies des Vieillards*, &c., 1868, p. 124.

[7] Dr. Haig has recently confirmed this fact.

very close relation between lead-impregnation and the frequent occurrence of gouty manifestations. But it is not at once obvious why gout should not be more frequent in lead-cachexia than it really is. One or more factors in the causation are wanting. Dr. Pye-Smith[1] has never met with gout from plumbism without hereditary predisposition or intemperance, and enough has been already shown to justify this assertion. We may explain the fact that gout is not found to be associated with lead-impregnation to any noteworthy extent in the North of England, in Ireland, and in Scotland, by a consideration of the conditions of heredity and of the drinking habits of the people in these various countries. There is, and there has been for centuries, more gout in the English metropolis, and amongst the beer-drinking inhabitants of the southern counties of England, than there is or has been amongst the populations in the North, in Ireland, and in Scotland, where spirits are consumed; and, therefore, it is only to be expected that the Southerners should yield the largest return of saturnine gout. On the other hand, beer-drinking is not alone in inducing gouty disease, for this will occur in persons who have been habitual spirit-drinkers only, and also in those who, as in the case of Parisian artizans, drink,—immoderately, it is true,—both brandy and inferior qualities of French wine. Hence we may believe that the two main exciting factors, inherited taint and intemperance, act often together, and sometimes singly, in producing saturnine gout. In these cases there are certainly present two of the essential factors of gout, (*a*) altered innervation, and (*b*) retention of uric acid in the system.

The effect of lead in inducing gout may probably be attributed to the specific action of the poison upon the nerve-centres, this malign influence evoking such trophical changes in the entire vascular system, and in the kidneys, as are prone to be produced by the morbid condition which we recognize as gout in its most comprehensive aspect. The lines of degeneration in the two affections, saturnism and gout, run, as it were, parallel, and seem only to be modified by individual habit and diathetic tendency.[2]

Dr. Saundby, of Birmingham,[3] after a careful review of thir-

[1] *Op. cit.*

[2] According to Pouey, the liver is mainly at fault, being disturbed by the action of lead, with interruption to its due metabolic functions.

[3] Medical Times and Gazette, September 1881, pp. 385, 412. Dr. Saundby records one case in his practice of a male, aged thirty-nine, a file-cutter, with no hereditary gouty taint, who had had gout three years previously, with colic, and blue line on gums. He had been intemperate in beer-drinking. He presented all the signs of granular kidneys and cardio-vascular degeneration.

teen cases, most of which have just been considered, arrives at the conclusion that the "doctrine of saturnine gout rests rather on authority than on observation." A larger review of all the facts must, I conceive, lead to the belief that the connection between lead-impregnation and gout is both definite and unquestionable. The "authority" on which this doctrine rests is at least worthy of the highest respect, including as it does a large number of physicians who have been, and are, the keenest observers in the widest and best fields of study.

The late Professor Frerichs, of Berlin, was so good as to make for me the subjoined analysis of 163 cases of lead-poisoning. These cases were all observed in his clinic, and they have been examined with a view to discover whether any gouty association was noted in any of them.

It will be seen that the Berlin experience furnishes evidence of a negative character in relation to this matter, and goes, so far, to confirm the opinion expressed that the association of gout with lead-impregnation is most distinctly manifested where gout commonly prevails amongst the population.

Professor Frerich's Clinic.

"*Service of Dr. Ehrlich.*—An analysis of 122 cases of chronic lead-poisoning, of which only four occurred in women, and of which only two died (one patient having jumped from a window), resulted in the following:—

I. By *lead-colic* were attacked ninety-five men, three women. Of these, one had pulmonary phthisis, one croupous pneumonia, and one aortic insufficiency.

II. *Lead-palsy* attacked almost without exception the distribution of the radial nerve. It was noted in fourteen men and one woman.

- *a.* The affection was bilateral in twelve cases, and was here complicated six times with lead-colic and once with typhus.
- *β.* The affection was unilateral three times, and always limited to the right arm. One of these patients had colic.

III. *Lead arthralgia.* Six cases, three accompanied by colic.

IV. *Affections of the centres.*

- *a.* Cephalalgia saturnina.
- *β.* Encephalopathia saturnina colica.
- *γ.* Epilepsia saturnina colica.

δ. Epilepsia saturnina ĉ alucinationibus.
ε. Epilepsia saturnina, amaurosis fugax, colica.
ζ. Paralysis saturnina, ĉ poliomyeliti anteriore chronicâ.

V. *Varia.*

a. Two cases of circular gastric ulcer, of which one suffered perforation and proved fatal.
β. One case of lead-asthma (phthisis).

Palpable changes in the joints were not noted in any of these cases, nor were any cases of *nephritis vera* met with, although somewhat frequently, during the existence of the colic, albumen appeared temporarily in the urine.

Service of Dr. Litten.—In forty-one cases of lead-poisoning (colic, lead-palsy) were six in which joint-affection was present, generally appearing in a slight degree, and but temporarily. Only in two cases was much swelling of the joints of the feet.

However, in no instances were symptoms of true gout present.

Albumen was only found four times in these forty-one cases, and soon passed away.

Amongst these 163 cases of lead-poisoning, sometimes slight and sometimes severe, there was not one single case of true gout. Also in no case was *nephritis chronica* present.

Besides these, I have about 200 other cases which I have not been able to analyze on account of illness, but I am confident that in not one case amongst them was true gout present.

My experience does not agree with that of Lancéreaux respecting nephritis and saturnine arthritis.[1] Why this should be, I do not know, but the observations made in my clinic are so careful and exact, that I cannot conceive it possible for such complications of lead-poisoning to be overlooked. Perhaps these results do not agree with your observations. True gout is seldom seen here, and that may be the reason why it is not found in association with lead-poisoning. Alcoholism, however, is often combined with it."

Dr. Lorimer, of Buxton, in an excellent paper on saturnine gout, gives an analysis of 107 cases which came under his observation.[2] He found that this disorder usually appeared at an earlier age than non-saturnine gout. In 70 cases the first attack occurred prior to thirty-five.

[1] *Archives Générales de Méd.*, December 1881.
[2] Brit. Med. Jour., July 24, 1886.

Heredity was less marked than in non-saturnine gout, and anæmia proved a notable feature.

The type of arthritis was asthenic, the local and constitutional features being less intense. This he rightly attributed to the associated cachexia and to the presence of organic renal changes.

Albuminuria was present in 89 cases permanently, or with intermission. The specific gravity of the urine on an average was 1.012; the uric acid was diminished, and, in the last stages, absent.

Arterial sclerosis and atheroma were noted in 69 cases. Heightened arterial tension and cardiac hypertrophy were also observed. Pericarditis was once met with.

Cutaneous manifestations were seldom found, eczema occurring in one and psoriasis in another case. There is a marked contrast here with the non-saturnine cases, where eczema is met with in about 30 per cent.

Gouty eye-affections were infrequent. One case of iritis was noted. Neuro-retinitis, as a renal concomitant, was, however, found.

In 28 cases the joints of the feet were implicated; in 7 of these the great-toe only was affected. In 34 cases the joints of the hands and feet were both affected. In 20 cases the joints of the hands. In the remaining 25 the knees also were implicated, and in 4 the elbows suffered.

Tophi were found in the ears in 23 cases.

Gout with lead-impregnation, Dr. Lorimer believes, attacks the kidneys chiefly; with alcoholic excess the joints.

The following case well exemplified gout as occurring in a patient suffering from lead-poisoning:—

C. J. D., æt. 32, a single woman, engaged in trimming upholstery, admitted under my care, December 1886. Very sallow and cachectic; of small build. Had employed a cosmetic powder for her face for five years. This was analyzed and found to be carbonate of lead. Marked Burtonian line on gums and buccal membrane. Arms much wasted, double wrist-drop. Supinators unimpaired. Gubler's dorsal tumours of the hands well-marked (caused by over-flexion of carpus, deficient support of extensor tendons, and prominence of bones). Muscles of upper arms and scapulæ affected, the deltoids especially. Legs, muscles much wasted and flabby, tremors, no rigidity. Pains in muscles and bones, with much tenderness on pressure. Both knee-jerks increased. Slight ankle-clonus. Walks feebly. Faradic contractility completely lost in extensors of thumbs of both hands; much impaired, but not lost, in other extensors. Supinators react readily. Galvanic irritability lost in muscles of thumb, the others reacting fairly well, except extensor communis digitorum of right arm. Electro-sensibility unimpaired. Muscles of both legs react readily to both currents. (*Dr. Steavenson's Report.*)—History of attacks of colic. Urine of rather low sp. gr., with occasional traces of albumen and blood. Urea much diminished. Pulse frequent and of high tension. Attacks of acute gout in left great toe and

left wrist-joints. Appearance of small, shot-like, and movable subcutaneous nodules over the tibiæ.[1] No family history of gout obtainable.

Great improvement in the course of six months under treatment by good diet, warm baths, galvanism, and large doses of iodide of potassium.

3.—The Relationship between Gout, Struma, and Tuberculosis.

I think it may fairly be affirmed that gout and active tubercular disease are not often found associated. It is, of course, to be borne in mind that the former is, for the most part, a disorder of middle life, and the latter one manifesting itself chiefly in the earlier decades. Many who sink under tubercular disease might, if they were spared, eventually become gouty.

In the constant and inseparable blendings of diathetic states the gouty and tubercular must, and do, often coalesce.

It is not often that gout is well-marked in persons distinctly tuberculous. I have, however, met with examples, and seen others, in which struma and gout distinctly blend, and afford noteworthy manifestations at different periods of life.[2]

Some of the older writers described an arthritic or gouty form of phthisis, occurring chiefly in middle or late life in both sexes, characterized mainly by tendency to copious hæmoptysis, slight muco-purulent expectoration, and, especially, by its slow progress and tendency to cure. They also noted the occurrence of cases in which there was little cough but much dyspnœa, and of others which would now be recognized as examples of chronic bronchitis and emphysema. They laid stress upon the frequent presence of cretified masses in the lungs of such persons. These would now be regarded as evidence not of any specific gouty element, but merely as indications of obsolete and healed tubercular lesions.

So far, these signs afford proof of tendency to obsolescence in tubercular processes in the gouty.

Laycock described the "arthritic tubercular cachexia," and declared that, "with the taint of gout in the ancestral or collateral line, there are always present some of the leading characteristics of the arthritic diathesis, namely, regular features, well-set, sound teeth, and a pearly white or florid complexion.

[1] These presented the characters of the ephemeral nodules first described by Féréol, Troisier, and Brocq, and subsequently by Dr. Barlow, myself, and others, as occurring in rheumatic individuals.

[2] As pointed out by Paget, this co-existence may be found without any modifying influence of the one state upon the other.

But the lower jaw is usually contracted, the bones of the face small, the skin delicately thin or transparent; the neck elongated, the thorax narrowed, and the heart's action feeble and irregular."[1] He noted the tendency to hæmorrhage in such cases as a precursor of the tubercular deposit, due, as he thought, to fatty degeneration of the pulmonary vessels; also the absence of deposit in the lymphatic glands.

Gout and scrofula, according to Paget, are often, by inheritance, so intermixed that the resulting condition can hardly be analyzed. In early life strumous manifestations most usually prevail, and, at this period, little or no token of any gouty element may be apparent.[2]

It is, therefore, chiefly in later life that evidence is afforded of the coalescence of the two diathetic states. The observations of Paget have shown that while scrofula may often be outlived, yet manifestations of it may occur late in life, and materially modify ordinary gouty processes. He tells of a patient of gouty inheritance, who in middle life had inflammation of the tarsus resembling acute gout, but the pain, stiffness, and swelling did not subside as usual in due time. After several months there remained pulpy swelling about the tarsus, with dull aching, inutility and wasting of the leg, and other features quite characteristic of scrofula. Treatment was required for months with splints and other means inappropriate for gout. A daughter of this patient had scrofulous disease of the hip-joint.[3] In such a case the likeness to gout is manifest at the outset, but instead of a transient paroxysmal inflammation, there gradually supervene the characters of chronic scrofulous arthritis.

With the knowledge of specific bacilli as associated with tubercular processes, it has been sought to explain the acknowledged inhibitory effect of gouty influence upon scrofula as due to the direct action of uric acid in the blood upon these parasites. This is the teaching of Lecorché, who, with Dr. Pye-Smith, denies the existence of all diathetic habits of body. I am not prepared to

[1] *Op. cit.*, p. 102.

[2] The famous Dr. Samuel Johnson was scrofulous in early life, and had a scarred neck. He developed gout some years before his death, and died of gouty cachexia at the age of seventy-five. Dr. Norman Moore, in an introductury lecture on pathology, has published the notes of his *post-mortem* examination by Mr. Wilson, the anatomist. It shows that he had the anatomical conditions usually associated with gout, chronic interstitial nephritis, and emphysema of the lungs. His attack of hemiplegia, some time before his death, points to the degeneration of his cerebral arteries. He was never very robust. As Boswell remarked of him, he possessed "an inherent *vivida vis*, which is a powerful preservative of the human frame."

[3] *Op. cit.*, p. 437.

accept this view. I recognize an antagonistic influence of the gouty upon the tubercular habit, and agree with those who find tubercular processes checked, often for long periods, and rendered obsolete in virtue of gouty predisposition. In this country no one has more carefully studied this subject than Dr. James Edward Pollock.[1] He, however, considers gout and rheumatism so closely allied to each other in their pathological development, that they may be considered together in their influence on pulmonary tuberculosis, and I regret that he has not studied the two disorders apart in this relationship. He quotes the views of Noël Gueneau de Mussy as to the identity of the arthritic with the tubercular constitution. This author held that in many instances where gout is believed to "overleap" one generation and come out in the next, the intermediate one was not free from gouty attacks, which were exhibited in scrofulous and tubercular diseases; and that females manifested the inherited diathesis in various forms of tubercular disease. A distinction must, however, be made between the arthritic habit and the gouty branch of that stock.

There is evidence to show alliance between tuberculosis and the rheumatic branch of the arthritic stock,[2] but much less evidence to indicate a connection between tuberculosis and a truly gouty proclivity. Dr. Pollock's statistics afford proof of this, there being many more instances of associated rheumatic disease than of gout. He showed that the male sex was more affected in this association, the ages being from twenty to twenty-five years, while the gouty cases occurred, as might be expected, in older subjects, from forty to fifty years of age. In the case of rheumatism, the question of antagonism is also materially affected by the coexistence of valvular heart-disease, which is a recognized cause of retardation in tuberculosis.

Dr. Pollock showed that gout when developed in a phthisical subject possessed inhibitory power and checked the tubercular process, the pulmonary symptoms being relieved; and that, in such cases, the disease was protracted, and a prognosis for chronicity was warranted.

The cases I have seen, and those that have been recorded, have been mostly in men past middle life. Their lung-symptoms betokened a "quiet" form of phthisis, with progressive fibrosis and dependent clubbing of fingers and toes. I exclude

[1] Elements of Prognosis in Consumption. London, 1865, p. 270.

[2] *Vide* Dr. Sutton's views, noted under head of Relation of Gout to Chronic Rheumatic Arthritis, p. 154.

all cases with rheumatic disease and cardiac complications from this category, and allude to purely gouty cases. In some cases of associated gout and tuberculosis, regard must be had to the special influence of alcoholic excess, which exercises untoward effect on the latter.

Dr. Pye-Smith has recorded four examples of pulmonary phthisis occurring in truly gouty subjects, all males, aged thirty-nine, forty-eight, and sixty years respectively—the age of one not being mentioned.[1]

In the man, æt. sixty, there were tophi. There was history of gout in two brothers. The urine was albuminous, and there was hæmoptysis. In two others, attacks of gout occurred during the progress of the lung-mischief. In the youngest patient there were, with much uratic deposit, very "bad" arteries, granular kidneys, hypertrophied cardiac left ventricle, and gastro-enteritis, the latter causing death; tubercles in both pulmonary apices, with vomica in right lung and much cicatricial tissue.

In the autopsies of eighty cases of gout recorded by Dr. Norman Moore,[2] pulmonary tubercle was found six times in men whose ages varied from thirty-four to sixty. It occurred in all stages, from recent deposits to cavities and cretification. In no case was it the immediate cause of death, and in none had it given rise to prominent symptoms during life.

The noteworthy features of pulmonary tuberculosis as modified by gout are, that there is apparently more than ordinary tendency to free hæmoptysis at the outset, with tendency to occasional recurrence of it; that, with acute exacerbations of tubercular processes in the lungs, there is a marked tendency to limitation of the disease and to its subsidence, this being followed by the salutary processes of cicatrization; and that, as a result of this mode of tubercular evolution, inhibited by the gouty habit, such patients exhibit a marked tendency to recovery, or to endure for a long period. In such cases there is usually not far to seek ancestral history of tuberculosis, and the factor of gouty impress is seen, so far, to be of somewhat favourable import, since with each arrest of tubercular process is afforded means for improving the general health, and, sometimes, for promoting actual recovery. From whatever cause, the textures of the gouty appear to be less vulnerable than those of others in respect of tuberculosis, but the antagonism is far from complete, and hence the tuberculosis may prove a fatal association.

[1] Guy's Hosp. Reports, *loc. jam cit.*

[2] *Op. cit.*

The following case is illustrative of coalescence of saturnine gout and pulmonary phthisis.

F. P., æt. 39, organ-pipe-maker, came under my care in St. Bartholomew's Hospital, November 5, 1888. His father had suffered from gout. There was no phthisical history obtainable. Exposure to lead-influence for twenty-five years. No colic or paralysis. Blue line on gums. Winter cough five years. No history of alcoholic excesses. At age of twenty-three, first attack of gout in left great toe-joint. Many subsequent attacks in toes, insteps, knees, wrists, and elbows. Failing health and wasting for twelve months past. Cough worse last five weeks, with much frothy expectoration, and night-sweats. Twelve days ago hæmoptysis, "half a cupful;" to-day twice as much. Temperature 99°. Pulse 108, good volume and tension, arteries not markedly thickened. Respirations 32. Sputa in muco-purulent pellets, sour-smelling. Urine of sp. gr. 1010, void of albumen. Several nocturnal micturitions. On examination of the chest after a few days, the physical signs indicated consolidation and softening of upper lobes of each lung, with vomicæ more advanced on left side. Some general emphysema.

No indications of active gout, and no tophi detectible.

This man looked ten or more years older than his age. He improved a little after admission, but somewhat suddenly failed, lost strength, and died on November 17.

In this case I made a diagnosis of the supervention of pulmonary tuberculosis on chronic bronchitis and emphysema in a man the subject of chronic saturnine gout. I believed his kidneys to be in a condition of progressive (granulative) nephritis.

The autopsy showed that both pulmonary apices were involved with chronic interstitial pneumonia, leading to bronchiectasis. Numerous vomicæ, full of purulent matter, existed apart from the bronchial dilatations, which were fusiform and not expanded. Some scattered tubercles were seen in their neighbourhood. No tubercle in the bronchial glands. Dr. Wynne examined portions of the indurated lung and tubercular-looking matter, but found no evidence of true tubercle.

There was a good deal of general emphysema.

The heart was slightly hypertrophied in its left, and dilated and indurated in its right, ventricle.

The kidneys were of full size, very hard, containing cysts in places, and granulations in parts of the cortices. No uratic streaks in pyramids. The capsules stripped fairly well.

The articular cartilages of the right great toe-joint were encrusted with uratic deposit, and the same was found in the right knee-joint in streaks near the edges of the condyles, and along the margin of the patella. The aorta was atheromatous in places.

W. C., æt. 65, came under my care in June 1879 for hæmoptysis. He had had winter-cough for four or five years. The physical signs indicated consolidation and softening at both pulmonary apices, and there were symptoms of tubercular enteritis. Fifteen years previously, gout occurred in the right great toe-joint, and other attacks had followed. His maternal grandfather was said to be a "martyr to gout," and his

mother suffered from "chalky" gout. His father lived to be ninety. The urine was void of albumen. The result of the case is unknown to me.

S. J., æt. 51, a butcher, came under my care for hæmoptysis in June 1883. He had been a free drinker, chiefly of spirits. No gouty history known of in his family. First attack of gout in left great toe-joint eight or nine years previously. Many subsequent attacks. There were signs of consolidation and softening at each pulmonary apex, with indications also of fibrosis. The urine was free from albumen.

In attempting, as we ought, to determine the ultimate issue of each case of blended diatheses, we must discover how much of each prevails, since it is mainly the question of the intensity or predominance of one or the other state which must furnish the clue.[1] The onset of pulmonary phthisis in cases of chronic gout must be sometimes regarded as a mode of degeneration in cases where, by reason of alcoholic intemperance, the lungs become vulnerable and break down. The progress of the phthisis is retarded in such instances by the remaining degree of inherent vitality, and by the tendency to fibroid change, which always warrants a prognosis for chronicity.

4.—Relationship between Gout and Cancer.

The gouty are in no way protected from occurrence of cancer. They appear rather to be somewhat liable to it. In ten fatal cases of gout, Pye-Smith records cancer in two instances in men. In one, æt. forty-seven, there was cancer of the œsophagus opening into the lung; and in the other, æt. fifty-nine, there was cancer of the ribs, vertebræ, liver, &c. He speaks of its occurrence as unconnected with gout, save by its preference for the same period of life.

According to Paget, gout and cancer are often found together, each pursuing its separate course, "the cancer in one part, the gout in another." In treating of the succession of constitutional diseases, he declares it not to be rare to find a patient who has been scrofulous in early life, gouty in later life, and finally the subject of cancer. He relates the case of a gentleman of seventy-five years of age who had psoriasis for thirty years, and had taken calomel for it in grain-doses daily for twenty-five years, enjoying all the time excellent general health. At seventy-five, epithelial cancer appeared, and quickly increased on one little finger. After its amputation there followed his first attack of gout, a family disease, with which his brother, eighty years old, was at the time suffering. He died within a year with cancer in his axillary glands.

[1] According to M. Baumès, a gouty father and a tuberculous mother will beget an asthmatic child, the father furnishing a predisposing general cause, the mother a predisposing local cause.

Charcot noted at the Saltpêtrière Hospital that women with Heberden's nodes were rather apt to be the subject of cancer of the breast and womb. This is of interest in respect of gout, which is certainly the cause of some forms of these; and in one such case I met with cancer of the liver, and discovered uratic deposits in association with the digital nodes. I had a well-marked case of tophaceous gout under my care in a woman, æt. circ. fifty-five, who died of cancerous tubera in the liver.

William Budd recorded a case of cancer of the penis, with deposits in the liver and lungs, in a man of sixty-eight, who was the subject of true gout with tophi.[1]

Three examples of cancer of the stomach associated with gout are related by Lecorché, one in a man æt. fifty, one in a man æt. fifty-five, and the third also in a man æt. sixty-three.

In France, cancer has been thought to be especially frequent in persons of arthritic predisposition. Bazin, Cazalis, and Verneuil have maintained this; but the association has not been especially noted in this country, and my colleague, Mr. Butlin, tells me that he has not been struck with such a coincidence while studying on the broadest basis the whole subject of cancerous disease.

Respecting the influence of gouty habit on cancer, the opinion of Paget may be noted, to the effect that the latter is apt to be attended with more pain than is usual, severe paroxysmal pain, and that cancers in the gouty are liable to inflammations of their substance.

The occurrence of cancer of the gall-bladder may be noted in connection with the prolonged irritation of biliary calculi, which are not infrequent in persons of gouty inheritance and habit, and especially in women.

5.—Relationship between Gout and Syphilis.

With the more accurate knowledge of the manifestations of gout and of syphilis that has been attained of late years, has also come more certain knowledge of the mutual influences of these two disorders.

In this case, as in that of the relations between gout and struma, it must be noted that the early manifestations of syphilis occur earlier than those of gout; but even from the earliest periods a modifying influence may be observed in some cases.

And, first, it may be stated that there appears to be not

[1] Lancet, 1851, p. 482.

infrequently in the goutily disposed a special susceptibility to the poison of gonorrhœa, whereby they suffer more readily and intensely than others. It is in such persons that there is special tendency to arthritis, so-called gonorrhœal rheumatism, and to the eye-troubles often associated therewith. It would, perhaps, be more correct to affirm that persons of the arthritic diathesis thus readily suffer, for either rheumatic or gouty antecedents and peculiarities may be traced in the majority of cases of this nature.

It is still a vexed question whether conjunctival blenorrhœa is a result of direct inoculation or not. The weight of evidence is in favour of the non-contagious view. Such infectivity as is here conceivable can hardly be assigned as the cause of sclerotitis, which is not infrequently associated with gonorrhœa in the gouty. It hardly admits of doubt that cases of gonorrhœa followed by arthritis and sclerotitis occur most often in persons of the arthritic diathesis, and chiefly in the gouty line of it. There is evidence to show that urethritis may occur in the gouty after pure intercourse, where there may be hardly more than leucorrhœa as the excitant, and that such urethritis may again and again, when it occurs, induce articular and eye-symptoms of the type of so-called gonorrhœal rheumatism.

Such a sequence is very significant of gouty predisposition, exhibiting the special vulnerability just referred to, and may be met with in men who have had no regular gout.

With respect to the specific poison of lues, it is now—and I entertain myself no doubt on the matter—fairly well-recognized that its fruits will vary according to the tissue-soil on which it is implanted; so that varying manifestations may be looked for according as the patient is strumous or gouty, or degenerate by alcoholic and other excess.

In the gouty there is reason to believe that syphilis tends to evoke lesions of the skin akin to those which are more common in such persons, especially the squamous class, and renders such patches rather more itchy and irritable than they are in other subjects.

There is no evidence that the primary lesions are in any way influenced by gouty predisposition.

The tertiary symptoms, as occurring later in life, are more apt than any to be modified in the gouty.

Thus, we meet with the various forms of psoriasis of the tongue, or leucoma, and with chronic ostitis and synovitis rebellious to treatment. Persistent neuralgia and myalgia may occur in those

who are both gouty and syphilitic. M. Lecorché is inclined from his observations to believe that the gouty habit confers some immunity from the manifestations of syphilis, but his cases afford little support to such a doctrine.

Intractable gleet is sometimes dependent on gouty habit of body, and the same may be affirmed of certain urethral strictures, which, according to Paget, may be likened to the indurative changes met with in the corpus cavernosum or in the palmar fascia.

Mr. Hutchinson declares that he has never seen any reason to believe that gout-tendencies modify syphilis. "Syphilis," he states, "varies very remarkably in relation to the state of the patient, but it seems to me more a matter of inexplicable idiosyncrasy than of anything which can be assigned to complication with other diatheses. It is sometimes very difficult to determine between what is gout and what syphilis in cases where bones and joints suffer, and especially in cases of threatened ataxy after syphilis. Usually, I believe, gouty persons have syphilis just like others, and syphilitic patients have gout in the same way. I cannot see any reason to believe that the one effects any important modification of the other."[1]

6.—Relation of Gout to Diabetes and Glycosuria.

No fact in practical medicine is better established than the dependence of a variety of glycosuria on the gouty habit of body. The indications of gout are seldom far to seek in the cases now referred to. There may, or may not, be history of paroxysmal articular attacks. The family history is, as a rule, plainly indicative of the predominance of this diathesis. Thus, with gouty ancestry or parentage, there may occur in a family certain members who develop true gout, and others diabetes. Some may be the subject of megrim, of obesity, of biliary lithiasis, urinary gravel, eczema, asthma, or other forms of masked gout. These relationships have been dogmatically insisted on in the French school; my own experience amply confirms them, and a study of them is of the highest import and significance.

It has long been known that there is some connection between diabetes and the gouty diathesis.[2] A careful study of many cases

[1] Private letter.

[2] Stosch in 1828 and Naumann in 1829 are credited with the earliest mention of these cases in Germany.

It seems not unlikely that reference is made to some instances of diabetes of the class here discussed by Trotter, who remarks, "The majority of persons whom I have known subject to diabetes were lovers of the bottle. I suspect that many drunkards have this complaint upon them without taking notice of it, and that it comes and goes, without creating alarm, just as they happen to live regular or otherwise."—*An Essay, &c., on Drunkenness* (D. M. I., Edin. 1788), by Thomas Trotter, M.D. London, 1804.

Thomas Willis, in 1674, attributed diabetes to the "guzzling of strong wines, sadness, or long sorrow."

Rayer is alleged (by Charcot) to have noticed that gout changes into diabetes. I cannot find any proof of this in his *Traité des Maladies des Reins*, 1839.

of diabetes and of gout cannot fail to lead the clinical observer to believe in such a connection. It is remarkable, therefore, to find that but few authors, treating respectively of these disorders, allude to this relationship. Where the subject has been noted, there has been but little light thrown upon it, and indeed it is one of extreme abstruseness.

It may be well, in the first place, to relate some of the observations that have been already made, and it is interesting to note that the subject has been more often approached by writers from the diabetic than from the gouty side.

Prout [1] was amongst the first to note that glycosuria was common amongst dyspeptic and gouty individuals, and that hundreds passed years of their lives with this symptom more or less constantly present, who were quite unaware of it till the quantity of urine became increased.[2]

Prout's large field of experience led him to classify diabetic patients into two classes (and others have followed him in this), the spare and feeble type, and the robust and corpulent type, and upon this classification we shall find that we may best illustrate what is certainly known of this subject. The connection between the gouty habit and glycosuria is shown in two well-marked cases related by Prout, in one of which red gravel, and in the other renal calculus occurred. He also gives instances of glycosuria in corpulent women, and, so far as I know, was the

[1] On Stomach and Renal Diseases, 4th edit., p. 34, 1843.

[2] Opinions still differ as to the "unity" of diabetes. On the one hand, the milder and intermitting forms of the disease, and the temporary and slight degrees of saccharine impregnation in the urine, seem to favour the view that there is a separate disorder, or even a series of disorders, to which the term glycosuria is best adapted. On the other hand, the fact that these forms of glycosuria sometimes distinctly pass into true and essential diabetes appears to warrant the opinion of the unity of the disease, the glycosuric cases being merely regarded as mildly diabetic.

My own opinion inclines to the latter view. I cannot admit that any localized perverted chemical relations can habitually proceed in the body without the intervention of the directing influence of the central nervous system.

The fact that irritation of the liver by food of injurious quality sometimes induces glycosuria, is readily explained by reflex nervous action through the medulla oblongata, and may be termed, as it has been, hepatic glycosuria; but if clinical experience shows that such disorder eventuates in ordinary diabetes, with, probably, damaged nerve-centres, I think the unity of the disease fairly proven. There are, without doubt, varieties of diabetes in relation to causation, and of these are the symptomatic, or diathetic, cases. In many of them, if the primary cause can be removed, the disorder is arrested. Amongst these varieties is to be placed that in relation to arthritism. Such cases as those recorded by Lancéreaux and others,* where the disease was obviously due to destruction of the pancreas, constitute another, and certainly rare, variety of diabetes.

* *Bulletin de l'Académie de Médecine*, 1877, 2e série, tome vi. p. 1215.

first to show that pruritus vulvæ was often a symptom of diabetes in women.

Prout further insisted upon the fact that in favourable cases of diabetes the quantity of uric acid passed was very considerable, and he traced the earliest symptoms of the onset of glycosuria back to the time, in any given case, when the urine, which was formerly continuously turbid on cooling, began to be clear, or to a definite attack of gout or rheumatism. He stated that this change sometimes occurred abruptly, the diabetic symptoms gradually supervening. To such cases he applied the term "latent" diabetes.

About thirty years ago, Dr. Bence Jones wrote upon this subject under the title of "Intermitting Diabetes,"[1] relating seven cases in which alternations of glucose and excess of urates occurred.

In 1854, Dr. W. Gairdner wrote that he had long surmised that saccharine impregnation, not amounting to any diabetic tendency, was attendant on various phases of gout.[2]

Claude Bernard[3] referred to cases of alternating diabetes in which attacks of gout or rheumatism replaced the glycosuria, the urine being charged with uric acid:—"On voit quelquefois des malades goutteux dont les urines contiennent beaucoup d'acide urique, présenter tout à coup le symptôme des diabétiques, et les urines se charger de sucre, c'est-à-dire la goutte se changer en un accès de diabète."

Laycock[4] taught that the gouty diabetic patient did not waste nor become tuberculous.

Marchal (de Calvi) discussed this subject very fully in his excellent work on diabetes.[5] His belief was that gout and diabetes (in its most common form) are only different expressions of the same morbid state, or holopathy, sub-diatheses of the uric acid diathesis. He regarded uric or gouty diabetes as the common variety and the type of diabetes, and gave his reasons for this opinion at length. His theory was, that when the uric acid diathesis affected the solids, it gave rise to gout or rheumatism, and when it affected the blood itself, it set up diabetes; and that diabetes was nothing else than gout in the blood.

In reviewing Marchal's theory, Charcot states that his con-

[1] Medico-Chirurgical Trans., vol. xxxvi., 1853, p. 403.

[2] *Op. cit.*, p. 127.

[3] *Leçons de Physiologie Expérimentale.* Paris, 1855, p. 429.

[4] Lectures on Pract. of Physic. Edinburgh. 1862.

[5] *Recherches sur les Accidents diabètiques et Essai d'une Theorie generale du Diabète.* Paris, 1864.

clusion agrees with previous observations and with actual facts; but he believes that Marchal extended the influence of this form of diabetes, and that of the uric acid diathesis in general, too far, his views not being applicable to the favoured classes of society, at least in France. Lecorché[1] thus alludes to Marchal's theory:—"On aurait tort de vouloir donner à ce terme spécifique de diabète goutteux une comprehension trop grande, et surtout de regarder tous les diabètes, comme fatalement liés à la grande diathèse urique, ainsi que l'a fait Marchal."

The question of this relationship had much interest for Trousseau, who discussed it in his lectures both on gout and on diabetes. He fully recognized the alternation of a diabetic with a gouty state, but he differed from Prout in that he did not consider the glycosuria to constitute the disease known as saccharine diabetes. He described an intermittent diabetes occurring only after meals, sometimes becoming continuous, however, and a periodic form in which glycosuria existed at distinct periods and at long intervals; and believed that these were perhaps only different forms of true diabetes.

Garrod has made some interesting observations on the relation of gout and diabetes, showing that the supervention of the latter in any given case tends to check the expression of obvious gouty symptoms, the increased urinary water carrying off uric acid and other solids. In cases where the gout has continued, he has not found much increase of urinary secretion, although there may have been much glucose; hence he believes that the uric acid may not have been completely thrown out. He quotes one well-marked case in a male æt. sixty, who had gout yearly and half-yearly after his forty-eighth year. Diabetes suddenly supervened, and no gout appeared for four years. The diabetes was afterwards checked, the specific gravity of the urine falling from 1.041 to 1.021. Later, slight gout followed on an attack of bronchitis. Garrod has known of several similar cases, as also of instances where patients have lost all traces of gravel and calculi on the supervention of diabetes.

Charcot[2] has recognized the relationship, which, he states, is regulated by still unknown laws, and gives particulars of a case. He also shows in a tabular form how gout, scrofula, diabetes, and corpulence were found in many members of one family, and gives in another table the following particulars:—

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[1] *Traité du Diabète*, p. 273. Paris, 1877.

[2] *Leçons sur les Maladies des Vieillards et les Maladies chroniques*, p. 98. Paris, 1868.

Father gouty { First son had gravel. / Second son, diabetes. / Third son, gout, phthisis. / Daughter, gravel.

He believes that the frequency of this relationship varies according to the sphere of the observer.

Sir William Gull tells me that he has long observed the dependence of glycosuria upon a gouty state, and remarks that such cases are not uncommonly discovered, but that they do not discover themselves. He thinks they are not regular cases of diabetes, though they may drift into the confirmed malady. They occur mostly in men, and cardiac and renal changes may be associated with the condition.

Under the head of "Milder Types of Diabetes," Sir William Roberts describes a group of cases in which glycosuria is found in persons advanced in years, of full habit, where there is moderate conservation of flesh and strength, slight diuresis, small amount of sugar passed, abundance of uric acid deposit, together with the frequent occurrence of gout. The sugar, he states, is sometimes present for years, varying greatly in quantity, and sometimes intermitting.[1]

In his article on diabetes in Reynolds' "System of Medicine," Dr. Lauder Brunton remarks that the affection is often seen in those of gouty habit, and that in such patients the disorder may exist for a considerable time without producing much apparent effect upon the general health.

Dr. Dickinson[2] suggests that there is a form of glycosuria which is primarily hepatic. "It is," he remarks, "slight and transient, and without much diuresis. It occurs in full-fed, gouty, and plethoric people, whose urine is loaded with uric acid or lithates. In this form of glycosuria the constitutional symptoms of diabetes are mostly absent."

Lecorché[3] observes in his classical work on diabetes that "of all diathetic glycosurias, the gouty and rheumatic are, without doubt, the most important."

Of this variety of diabetes, he writes, that it is often preceded by intermittent glycosuria, which is in intimate relation to attacks of gout. Sometimes sciatica and gravel are the gouty indications. Diabetes, once declared, does not materially differ from the ordinary form of the malady. At first intermittent, it does not

[1] On Urinary and Renal Diseases, 2nd edit., p. 258. London, 1878.

[2] Diseases of the Kidney, Part i., Diabetes, p. 99. London, 1875.

[3] *Op. cit.*, p. 522.

become continuous till after a certain lapse of time, and even then exacerbations which are only explicable by the peculiar diathesis are apt to occur. The gouty symptoms may be little pronounced, but there is history of strong hereditary tendency, and there may be coincidence of neuralgia,—facial, sciatic, and lumbar,—and of neuroses, such as asthma and hemicrania. Dyspepsia, nephritic colic, pyelitis, and hæmorrhoids are sometimes associated. Lecorché further notes that there is but slight polyuria, and that the amount of glucose varies from 300 to 500 grains in the pint, red gravel being also common in the urine. The glycosuria may persist indefinitely without becoming transformed into diabetes.

Lancéreaux believes firmly in the relation between the arthritic diathesis and diabetes. "L'obésité, le diabète gras, la gravelle urique, et la goutte, forment une première série de processus morbides, qui se rencontrent successivement ou simultanément chez un même individu, dans une même famille,[1] se succèdent par hérédité, et procèdent d'une même condition pathologique, l'insuffisance des combustions. Un lien étroit de parenté réunit par conséquence ces états pathologiques, et les rend inséparables."[2]

M. Lasègue describes cases of imperfect gout becoming cases of incomplete diabetes in certain individuals, a single attack of bastard gout provoking a transitory diabetes.[3]

In a series of 600 cases of diabetes treated by Dr. R. Schmitz of Neuenahr,[4] he states that in forty-five subjects diabetes was directly attributable to gout, and its origin to the injurious influence exerted upon the nervous system by the fact of the blood being poisoned by uric acid. The gouty symptoms had existed in the most varied forms long before the appearance of the diabetes in all the cases.

With respect to obesity, he found it most conspicuous in thirty-five of the whole number, and increase of it occurred in some cases. In forty-six cases there was very little loss of corpulency. It is probable that many of these instances presented the gouty form of the disorder.

Dr. Ord[5] has called attention to cases of this nature. In an analysis of twenty-two instances of glycosuria occurring in

[1] "Deux frères que je soigne en ce moment sont, l'un obèse et diabétique, l'autre graveleux et goutteux. Les faits de ce genre sont relativement communs."

[2] *Traité de l'Herpétisme*, p. 282. Paris, 1883. [3] Private communication.

[4] Dr. Schmitz does not give the nationality of his patients in detail. Of his 600 cases 420 occurred in Germans and 180 in foreigners. In common with other observers, he met with a large number of cases amongst Jews, there being 93 instances. (Paper read before Medical Society of London, October 30, 1882, for the author by Dr. Sedgwick.) Lancet, November 4, 1882, p. 777.

[5] Brit. Med. Journal, November 25, 1882, p. 1041.

persons fifty years of age and upwards, where the disease in no case merited the term of diabetes, as commonly applied, he found the disorder, which he considered reduced to the rank of a symptom of other troubles, associated with four conditions of importance:—(1.) Nervous disorder, either as cause or as concomitant; (2.) Gout; (3.) Errors of diet, over-eating and over-drinking; and (4.) Albuminuria. In eight cases out of the twenty-two there was gout, and in one case rheumatoid arthritis of twelve years' duration. Albuminuria existed in ten cases, associated with gout in four. In the majority of the cases there was little or no emaciation.

Dr. Ord declares for the nervous origin of the glycosuria either as a central or a reflex disorder, and ingeniously offers an explanation for the intermittent form of the symptom in the gouty, comparing the disappearance of the sugar to that which occurs in diabetes during intercurrent inflammations, the glycosuria being, perhaps, "a phenomenon of the same class as gouty inflammation of joints, an active hyperæmia set going in part of the gouty process; set going in relation to irritation excited in the liver by dietary errors or other causes, just as inflammation of a joint is set up by a wrench or by over-exertion; that it may, in fact, be taken as meaning 'gout of the liver.'"

Having now quoted the opinions of various clinical authorities upon the existence of a relation between diabetes and the arthritic diathesis, I pass on to give what evidence I can upon the subject. It must be at once obvious that this is *a purely clinical question*, one, in the primary stages of the inquiry, at all events, upon which neither the physiologist nor the chemist can shed any light. Given sugar in the urine, the problem is to find its clinical significance, and its relation, if any, to some known diathetic state.

It is obvious that if the diagnosis of a diathetic diabetes be made, the line of treatment will vary accordingly in virtue of the special relationship established. Such treatment may possibly be only temporarily applicable with prospect of benefit, since cases of glycosuria, dependent originally upon diathetic states, tend sometimes, if neglected, to become simple instances of essential or confirmed diabetes.

It may be truly affirmed that the relationship now under discussion should be better studied in England than in any other country. If it be the case, as I firmly believe it is, that more gouty disease prevails in England than elsewhere, the cases illustrating the connection of glycosuria and gout call for exposition at the hands of English physicians.

The question has, however, received a good deal of attention both in France and Germany, partly, it may be, because the wealthier classes of sufferers from this country have sought relief at various French and German Spas, and, hence, a large mass of material has been placed in the hands of physicians who have not in their own countries many opportunities for this particular line of study amongst their compatriots.

It is important to note that it has long been held that there is a positive antagonism between diabetes and gout. Scudamore believed that diabetes was more frequently met with in Scotland than in England, and conceived that the dietetic habits of the two peoples explained the prevalence of gout in the southern, and of diabetes in the northern division of the kingdom.[1] The same opinion has also been expressed with respect to Ireland, where true gout is most rarely met with, and diabetes not infrequently.

It has also been observed that gouty symptoms in a given case vanish as diabetic symptoms supervene; hence, another reason why an antagonism has been assumed. Garrod has afforded an ingenious explanation of this clinical fact by supposing that the increased discharge of water from the system washes out the accumulated and superfluous solid matters from the blood.

The presence of sugar in the urine has now attached to it smaller importance than was formerly the case.[2] This arises from the systematic and careful examination of this secretion which is now made in every grave case of disease, and it has been found that in elderly people of both sexes a little glucose is often present, even when no noteworthy symptoms lead to suspicion of its presence, or of any serious injury to the health. It is found, too, that the sugar in such cases is intermittent in its appearance, being sometimes replaced by excess of urea, and of uric and oxalic acids. Thus, there is commonly an alternation in the respective presence of uric acid and glucose. Sometimes uric acid and urea are in excess together with sugar.

In many instances of glycosuria which may be relegated to this class, the symptom would appear, as Dr. Pavy points out, to be little more than a measure of the digestive incapacity for amylaceous and saccharine food. There is a limited power of assimilation, varying infinitely in different cases, and at different periods of the patient's life. The physiological capacity likewise varies in this direction at different hours of the day. This fact is noteworthy, and although forming a prominent feature in all cases

[1] *Op. cit.*, p. 74.

[2] Brucke and Bence Jones found $\frac{1}{50}$ grain per ounce normally present in urine.

of diabetes, is of especial interest in relation to the gouty cases, since other forms of digestive incapacity exist in gout, which forbid the use altogether, or in limited quantity, of certain articles of meat and drink. It will be shown subsequently that what is bad for gout is also bad for the diabetes associated with it.

It would be manifestly wrong to suppose that in all the cases of mild, latent, or intermittent diabetes a gouty taint is to be suspected. In many of the patients belonging to the class of fat diabetics, I have failed to find any history or indications of arthritism. In a certain proportion, however, evidence is not far to seek.

In looking over my notes of a large number of cases of well-marked gout, I find that glycosuria is of extreme infrequence; and I argue from this that the more accentuated and complete the gout, the less likelihood there is of undue saccharine formation. The type of case in which glycosuria is apt to occur is that of irregular or incomplete gout. This does not, however, hold good for cases of rheumatic type, since in these the joint-affection appears to be present in a severe degree together with the glycosuria.

It is not necessary to enumerate the various morbid conditions under which glycosuria, in whatever degree, has been met with. It may, however, be stated that in cases where there is a disposition for sugar to appear in the urine without any special or readily recognized cause, there is much risk of supervention ultimately of true diabetes, unless the morbid tendency be early recognized and averted. Thus the well-known indigestion of starchy matter leading to glycosuria may be, for a time, a trivial matter in the case of certain obese persons; but if a restricted diet in respect of saccharine and amylaceous food be not taken, there is an abiding risk of true diabetes being established, which may yield to no plan of treatment.

The cases which I seek to illustrate in this connection belong to the category of robust and corpulent diabetic patients. They have also been classified in the Parisian school, especially by M. Lancéreaux and M. Lasègue, as the fat diabetics, in distinction to the lean. Dickinson describes this type as "plump and rosy." In a considerable proportion of this class it is found, on careful inquiry, that there is a gouty history in the family or in the patient, and in some cases there are present arthritic changes, which cannot properly be called gouty, but have been called rheumatic. Of the latter I am constrained to state that the evidence forthcoming is but small. I have only met with one case,

which I shall describe later on. I think it not unlikely that some of the cases in which glycosuria has been found have really been examples of gouty disease, inasmuch as the diagnosis is often incorrect. Charcot remarks, "I do not believe that diabetes has ever been observed as a complication of chronic rheumatism,"[1] and he quotes Griesinger's statistics, which showed only two cases of acute rheumatism in 225 of diabetes.

Garrod,[2] however, has described a well-marked instance in a man, æt. twenty-six, who suffered from typical rheumatoid arthritis, and became diabetic five months after the disorder began. He also had cataract in one eye, and died within nineteen months of pulmonary phthisis.

Dr. Ord relates another example.[3]

It is important next to examine as far as possible in what proportion cases of diabetes of all degrees of gravity are connected with, or related to, gouty influence. Charcot quotes statistics given by Griesinger (who studied diabetes amongst all classes of patients), which yield only three gouty among 225; also, some by Seegen, who practises at Carlsbad, presumably amongst the wealthier classes, and who found three cases in thirty-one diabetics (seven in 140 cases, as quoted by Lecorché in his *Traité du Diabète*).

It is of interest to note the relative frequency of the occurrence of this variety of diabetes. In all forms of the disease as observed by Griesinger, gout was only recognized as the cause in .3 per cent.; in Seegen's cases, gout figured in 9.3 per cent.; in Schmitz' series, in 7.5 per cent.; while in Dr. Ord's cases of mild and intermittent diabetes, 36.3 per cent. were thus attributable.

The pathogenetic relations of glycosuria in the gouty are perhaps as obscure as those of the graver forms of persistent glycosuria, which are truly diabetic. I am not now concerned to argue for, or against, the unity of all glycosuric or diabetic states. I

[1] *Op. cit.*, note to p. 230.

[2] *Op. cit.*, p. 520, case given at length with necropsy. Sir Alfred Garrod informs me that he has met with other cases of rheumatoid arthritis in which glycosuria occurred, and he conceives that the development of the rheumatic affection may, in some cases, be aided by this untoward state.

[3] *Loc. cit.*

M. Lancéreaux, in reply to my request for his experience on this matter, writes as follows:—"Assez rarement j'ai rencontré le diabète dans le rhumatisme noueux; c'etait encore le diabète gras. Existait-il une relation entre les deux états pathologiques? Je ne le pense pas. J'ajouterai qu'il est parfois facile de confondre le rhumatisme avec la goutte."

Dr. Wynne Foot, whose experience of rheumatoid arthritis in Dublin is very large, states that he has not observed glycosuria in connection with it.

have already mentioned, what is unfortunately a clinical fact, that the milder form of gouty glycosuria may sometimes eventuate in chronic diabetes.

The view of the matter which best commends itself to my mind is, that glycosuria occurring in those of gouty heritage, or already gouty in some fashion, is to be regarded as a form of visceral gout, the organ mainly in fault here being the liver. As with gouty processes generally, so here, a neuro-humoral pathology is necessary for a due conception of the disorder. On the nervous side, regard must be had to the causes commonly prevailing in these cases, which are such as to entail strain and exhaustion of the great centres, thus predisposing to instability and a neurotic state. The lines of morbid action here are conceivably somewhat as follows :—As a result of irritation or exhaustion—possibly some definite (as yet undetected) lesion of the cerebro-spinal or sympathetic nervous system—a vaso-motor change occurs either in the direction of irritation or paralysis. The morbid impulses take the route of the cervical portion of the chord, pass through the inferior cervical sympathetic ganglion, and so, by the splanchnic branches, reach the cœliac plexus. The result is either a temporary irritation or a more permanent passive dilatation of the hepatic arterial system, a prime factor in glycosuria. Such is a hypothetical mechanism for the impulses of central origin. It is, however, conceivable that similar morbid impulses may proceed directly from the cœliac plexus, instigated by irritation arising in the digestive tract from forms of dyspepsia, perhaps especially from such as lead up to a gouty state. Uric acid, when retained in the system, is believed to be stored in the liver and spleen, and its presence in excess in the former organ may, as has been conceived by Ord, excite, under some conditions, a veritable gout of the liver. The prevailing vascular condition of the organ will, thus, be one of high tension with hyperæmia, one eminently favourable to glycogenesis. On the humoral side, the peccant matter is probably uric acid acting as a local visceral irritant.[1]

[1] The late Dr. Milner Fothergill remarked that "glycosuria is common in stout persons, whose digestion of starch is perfect, and in whom the liver only dehydrates enough into glycogen for the wants of the system, the surplusage running off by the kidney. If it were not for this 'waste-pipe,' the individuals would become inordinately fat. Such glycosuria is quite different from diabetes leading to wasting, where either (1.) the liver has lost the power of dehydrating the sugar brought to it by the portal vein—the more probable hypothesis—or (2.) the ferments in the liver hydrate the glycogen or animal starch into sugar again too swiftly for the wants of the body, and the 'fuel food' escapes unburnt. If food, which undergoes no saccharine transformation, can be taken in sufficient quantity and assimilated, the diabetic is preserved; if not, he perishes."—*Indigestion and Biliousness*, &c., p. 94, 1881.

Dr. Haig has shown that dyspeptic conditions in the gouty may be induced by hepatic congestion, and lead to a fall in the acidity of the blood with a corresponding excretion of uric acid. Thus may be explained the temporary glycosuria of the gouty, which not infrequently alternates with discharge of free uric acid in the urine. By repeated attacks of this peculiar metabolic disturbance in the liver, and possibly with slighter provocation, the vicious habit tends to become permanent, and we have to deal with a hepatic form of diabetes, due to vaso-motor paralysis, probably often in association with another form due to gastro-intestinal dyspepsia with over-production of glucose.

In confirmed cases of glycosuria in the gouty, there is not improbably established a central neurosis, which persistently dominates the whole course of the malady.

We are thus in the presence of a well-marked form of visceral gout, and the evidence of its gouty dependence is seldom far to seek in these cases. The family history and the personal proclivities of the patient strongly attest a gouty habit, and I feel as convinced of the fact that glycosuria is here the indication of the disturbance wrought locally in the liver by gouty influence, as I am of the corresponding mischief which is sometimes effected in the kidneys when the gouty process is established in those organs, leading to cirrhosis, with polyuria and occasional albuminuria. In neither case may any marked or classical articular troubles occur, but in each it is not uncommon to meet with articular gout.

The most obvious fact to be noted in most of the cases now under consideration is that the patients do not present the ordinary aspect or recognized symptoms of diabetes as commonly understood. There is often no diabetes whatever in the etymological sense, and the first indications are manifested either to the physician in the test-tube, or to the patient by the symptoms of some undue thirst, slight muscular weakness, loss of flesh, and more frequent micturition, and in women not unfrequently by troublesome vulvar itching. Such patients are commonly robust in appearance, in middle life, of large frame, and frequently corpulent, with much abdominal obesity.

The latter condition has been often observed to precede the occurrence of glycosuria, and in the course of the disorder a large reduction in its bulk may take place.

It is certain that the degree of glycosuria may vary largely in different cases and in the same individual at different periods, also, that the tolerance of the system for saccharine impregnation varies much in individuals.

In all diabetic persons, regard must be had not only to the amount of glucose produced and discharged, but also to the double effect of the impregnation itself on the various tissues, and the degree of general cachexia which gradually ensues as the result of the malady. Hence, it is often more important, if it be possible, to treat the cachexia than the leading symptom, and it is always necessary for the real welfare of the patient to treat *him* rather than his ailment. This is a point requiring special attention in the later stages, and it is sometimes quite overlooked.

Little is known as to the direct influence of an abiding saccharine impregnation upon the various textures of the body. It can hardly be supposed to be innocent, yet in many cases it appears to be so. It is certain that vascular degeneration is not common in diabetes of the gravest character, and where it is met with, it occurs in cases the nature of which is now under discussion, where the arthritic element prevails and leads to this particular change (arthritic cachexia—*Laycock*).

Wasting extends to fat and muscle, and the skin may wrinkle in consequence, although perspiratory function is not checked. Some enfeeblement, with intermission, of the heart's action has been observed in the depressed state of health met with after the glycosuria has run on for some time untreated.

This cardiac failure is sometimes very marked in advanced cases. I have notes of one instance where the greatest relief was always obtained when a quantity of sugar was added to the diet, the patient feeling, as he stated, "pulled together" by it.

Amongst the mental conditions, undue irritability of temper is to be noted. This is well-recognized amongst gouty patients; but this altered state is sometimes met with in ordinary diabetic patients, and cannot be considered peculiar to this form of the malady.

Severe intercostal neuralgia has been found associated with this diathetic diabetes. I shall subsequently append a short note of one case, and Sir William Roberts has reported another which presented many features in common with it.[1] Both occurred in elderly persons. Sometimes dyspnœa suddenly supervenes in obese glycosuric patients with præcordial distress and palpitation, constituting a pseudo-anginal attack. There may be dilatation of the cardiac walls in advanced cases after the obesity has begun to pass off.

Arterial sclerosis may gradually set in, and make progress in long-standing cases.

[1] *Op. cit.*, p. 262.

Examples of gouty diabetes, if they may so be termed, are found to occur more frequently in the male sex. This is in accordance with the greater prevalence of gout in that sex. The majority of patients thus affected have either been the subject of gouty attacks at some period, or a clear history of gout in their families is elicited. They sometimes prove to have been large eaters and free-livers, and to have much appetite for bread, potatoes, and sweets. In many of the cases a noteworthy history of mental anxiety, shock, worry, or of chagrin appears to have been the precise determining factor, and from the date of such trouble the diabetic symptoms are found to have arisen. Recrudescence may be excited by mental shock and anxiety. In other instances, exposure to cold and damp appears to have been an exciting cause, and recrudescence of the disorder has also been traced to the too bracing influences of east winds, especially at the seaside, and in connection with sea-bathing in unsuitable weather. It has been asserted[1] that sea-air is generally injurious to diabetic patients, and facts are not wanting in proof of the statement. Bilious symptoms and pneumonia have been found prominent, and patients have begun to fall back from the time seaside residence ensued. The late Dr. Camplin insisted strongly upon the inadvisability of making any important change in the diet or habits of a diabetic person during the prevalence of east and north-east winds.

The amount of glucose in the urine may vary much, as has been already stated. Sometimes the *urina sanguinis* is more charged than the *urina cibi* in the same case. This is commonly regarded as a grave sign in any instance of diabetes, as indicating a more confirmed vicious habit. The whole amount of urine passed may not exceed the normal quantity of health, or may even sometimes be below this.

In respect of the albuminuria accompanying the cachectic condition, with cardio-vascular changes, sometimes reached in those cases, Schmitz found that the glucose and albumen were often passed in inverse ratio. After exhaustion and severe efforts the albumen was increased; there was more in the night- than in the day-urine, and after food the amount of it diminished. The specific gravity may be of high range. I have a record of one case where it was 1.060 and over for some years.

With the removal of the sugar under dietetic treatment and other invigorating influences, uratic and free uric acid sediments may occur in the urine, gouty pains return in various joints, and itching eczematous eruptions appear on the limbs.

[1] Frederick Simms. Brit. Med. Journ., December 1881, p. 1006.

I suspect that it is in cases of this nature that anthrax and furuncular inflammation are especially apt to occur. It is not within my experience that grave and intractable cases of diabetes often present this symptom. Marchal called these boils "furoncles uriques," and regarded them as "gout of the cellular tissue," believing that they were vicarious of more obvious gouty inflammation in the joints, and that in the subjects of them the urine contained excess of uric acid.

It is, of course, possible that a single examination of the urine in some of these cases may reveal no glycosuria; there may be excess of uric acid, and the glucose may reappear subsequently. Regard must be had to the life-history and other characteristics of the patient in making the diagnosis. Anthrax is found to be more frequent in the male sex. Some indication of its occasional connection with a gouty habit may be gathered from the fact, now admitted by most surgeons, that it is best treated without stimulants, or with only a moderate amount of them, and by milk-diet.

In these cases it is certainly not usual to meet with the disorder which affects the teeth and gums in the graver forms of diabetes.

If the disease be unrecognized and untreated, and drift into an incurable state, so that the diabetic cachexia is induced, the conditions of alveolar catarrh, osteo-periostitis, and loosening supervene, all which have been well-described by M. Magitot of Paris.[1]

The same may be stated respecting the troubles of vision, such as asthenopia and cataract, which belong to the diabetic cachexia, and are, therefore, only met with in confirmed cases, which hardly yield to treatment.

The "sweet breath" of the severe form of diabetes is not usually observable in these milder cases. Dryness of the mouth may, however, occur, as well as thirst during acute exacerbations of the disorder.

The skin retains its softness, and perspiration is not reduced in amount. I have seldom met with any tophi in these cases; but in one instance there was a small crab's-eye cyst over a terminal phalangeal tubercle.

Gangrene of the extremities has been known to occur in cases of this form of diabetes. It is not by any means peculiar to them. The prognosis is not necessarily bad, and, with care, this alarming condition may issue favourably. The textures appear

[1] Paper read before the Academy of Medicine. Paris, 1881.

to become unduly vulnerable in all cases of long-continued diabetes, when the patient may be considered cachectic.

According to Marchal, cerebral apoplexy is common in the gouty and in the gouty diabetic.

The subjects of gouty glycosuria are usually mentally vigorous and active, "men of affairs," acting under pressure and responsibility. They often have large appetites, and so combine, not seldom, both high-living and high-thinking. The trouble is apt to come on in the fourth or fifth decade.

I have already alluded to the fugitive glycosuria met with in acute gout. This may be regarded as the simplest form of it, and it passes off, not to recur, in the majority of cases.

In other instances there may be temporary glycosuria of more importance, and it may alternate with actual gouty joint-troubles, or with active phases of goutiness elsewhere, the glucose disappearing, and uric acid sometimes appearing in place of it in the urine.

The urine in these cases is usually very bright, acid, and refracting, the only deposit, when it occurs, being uric acid in crystals; and hence it is very different from the loaded urines, which become turbid and throw down uratic salts. As Pavy has shown, when these cases are successfully brought under control, a copious deposit of urates occurs, and this is one of the best signs that can be witnessed. The natural acidity of the urine may be increased through the occurrence of lactic acid fermentation, and thus uric acid falls just as if an acid had been added to healthy urine.

Each case presenting symptoms of gouty glycosuria must be a special study to the physician. No two are alike in degree, or subject to uniform prognosis. The disorder may endure for many years, even when permanently established. Much will depend on the original vigour of the constitution, the strength of will to submit to adjusted (not necessarily restricted) dietary, and the available means to maintain the highest standard of bodily and mental health. I know of nothing more harmful for such patients than to pronounce them the subjects of diabetes, the idea of which commonly conveys very depressing and mischievous impressions, difficult to remove. If such cases be treated as those of the graver form of diabetes should be, they become worse, losing flesh and nervous tone.

The disorder may appear in young women, descendants from a gouty ancestry, and the approaches of it are sometimes insidious. Slight failure of power, with seeming health, ruddiness, and even

buxomness, should lead to examination of the urine for glucose, which may be present at first fugitively, or in small amount persistently. For purposes of proper treatment, it is well to be early aware of the tendency. If the urine is not copious, it is apt to be concentrated; hence, there is incomplete washing out of the tissues, and tendency for urates to remain in the system.

I have noted the occurrence of strange and indescribable sensations down the spine and in the limbs in patients thus affected. Fatigue and sudden emotion are apt to induce this.

They sometimes suffer from paroxysms of intense burning sensation in the hands and feet, a true causalgia, and have a constant air-hunger, being intolerant of hot rooms and aggregations of people. Hepatic pain is sometimes experienced.

The appetite is apt to be capricious, and there may be periods when there is actual loathing of all kinds of food.

The glucose may largely disappear and give way to uric acid or uratic sediments, the quantity of urine diminishing at the same time. With this phase there is general discomfort of the system, and aggravation of the various gouty symptoms common in these patients. Alternations of glycosuria with attacks of gout or urinary gravel may occur (diabetes alternans).

Diarrhœa may be almost a constant condition, and the supervention of constipation in such cases adds much to the malaise.

I have known copious sweating occur for long periods, but it has afforded no manner of relief to other symptoms, as might have been anticipated, rather the contrary. Shingles may sometimes be met with.

It has been already observed that with the onset of glycosuria or gouty diabetes (with polyuria) the tendency to paroxysmal joint-attacks ceases. I can confirm this experience as applying to the greater number of cases met with, so that the more diabetes there is present, the less gout there is. Minor and incomplete attacks, articular and abarticular, may, however, arise in these subjects, especially if there is no polyuria.

In Chapter x. p. 224, I have referred to a remarkable case in which acute arthritic gout occurred in the course of well-established glycosuria.

I have met with several cases in which deep-seated and severe dorsal and lumbar pains have been associated with this state, suggesting the onset of aortic aneurysm or of a new growth pressing on the spinal nerves, neither of which occurred; in these cases relief was only procurable by dosage with anodynes repeated over long periods. These pains were probably due to neuralgia.

The occurrence of glucose in the urine of patients suffering from rheumatic fever, and treated for this with sodium salicylate, is possibly of interest in relation to gouty glycosuria. Salicylate-glycosuria occurs when the toxic symptoms of the drug (such as tinnitus aurium and deafness) are manifested by the nervous system.

As in all varieties of diabetes, so in this, prognosis is largely influenced by the age of the patient. Every decade beyond forty is in favour of any given case, and considerable anxiety must attach to most instances below that age.

Glycosuria in connection with gout is not likely to be met with before the third decade, nor before such time as ordinary manifestations of gout occur. Diabetes in all varieties is most common between thirty and sixty years of age. Cases of ordinary diabetes, not necessarily presenting any arthritic features, are met with in the descendants of the gouty at an earlier period. Amongst my cases I find those of a young lady of eighteen and a man aged twenty-one. Such a history is not uncommon in gouty families, and is fully recognized in France.

7.—Relationship between Gout and Obesity.

Amongst evolutionary developments of the gouty habit is obesity in some members of families thus affected. This was recognized by Bouchard, Charcot, and others in France, and by Laycock, of Edinburgh. The latter, in his nosology, remarked that fatty constitutional diseases were allied to gouty diseases. In ninety-four cases of obesity collected by Bouchard, there were gouty antecedents in twenty-eight, and rheumatic in thirty-three. Allied gouty states also prevailed in a majority of the remainder, such as migraine, diabetes, lithiasis (renal and biliary), eczema, and neuralgia. This is very strong evidence in support of an arthritic habit in association with obese tendency in the offspring. There is sometimes coincidence of obesity and actual gout.

Cases of marked obesity, sometimes even developing before puberty, are found to occur in the families of the gouty. A single member in such a family may alone show this tendency, of which I have known several instances.

The occasional association of renal calculi with obesity has long been noted.

In proof of the affinity of obesity to the gouty habit may be cited the figures of Bouchard, who found in a hundred cases of

biliary lithiasis seventy-two examples of obesity amongst the personal and hereditary antecedents, and thirty-five cases amongst the direct parentage. Amongst the parentage were also thirty cases of gout.

Obesity is met with in association with glycosuria in the gouty, the variety of "fat diabetes" numbering many in this class. Sometimes cases of extreme polysarcia occur with this association. The obesity may long precede the onset of glycosuria, and in such instances glucose should be occasionally sought for in the urine, and, if found, be met by appropriate (not too rigid) treatment. Such diabetic patients may lose their fat gradually, and become "lean diabetics;" but this does not always happen. They are met with in both sexes.

There is some tendency in the gouty to formation of fatty tumours, which may occur singly or in multiform variety. These are best left alone, certainly after middle life, and treated, according to the dictum of a distinguished Irish surgeon, "with contempt."

8.—Relationship between Gout and Oxaluria.

The frequent occurrence of calcium oxalate in the urine under varied conditions in persons with and without symptoms indicating its presence is well-recognized.[1] Garrod, I believe, was the first to demonstrate that the blood in gout contains oxalic acid.[2] He relates that he has frequently found it, and believes that it chiefly occurs during the inflammatory stage, and is probably derived from uric acid by oxydation. He detected it also in the sweat of two gouty patients.

Prout remarked that the oxalic acid diathesis differed from diabetes in its non-liability to be excited by an attack of gout,[3] but that oxalic acid calculus occasionally followed such an attack. He noted that oxalic acid concretions sometimes replaced those of uric acid in the same case, and that persons of the oxalic acid diathesis subsequently became glycosuric. He found, further, that the subjects of oxalic acid diathesis sometimes began to secrete an excess of carbonate of lime, and as the quantity of lime increased, that of oxalic acid diminished, while the phosphoric acid increased

[1] "On Dyspepsia and Nervous Disorders in connection with the Oxalic Diathesis." Contrib. to Pract. Med., by James Begbie, Edin., p. 178, 1862.

Vide "Notes on Oxaluria." St. Barth. Hosp. Reports, vol. ii. p. 160, 1866. (Paper by myself.)

[2] Med. Chir. Trans., vol. xxxii., 1849.

[3] On Stomach and Renal Diseases, p. 70, 1843.

until nearly pure phosphate of lime was excreted. During the transition the urine frequently deposited triple phosphate, but less of this occurred than when uric acid deposits were transformed into phosphatic deposits. In children, the transition from oxalates to phosphates was found to be often accompanied by white urates, as well as by triple phosphate of magnesium and ammonium. Most practical physicians probably agree that in these several transitions there is nothing to be noted in the patients which in any way suggests a gouty habit of body.

The significance of oxalate of lime deposits is certainly varied, and may be stated as dependent mainly on the following conditions :—(*a.*) Direct ingestion of oxalic acid in certain articles of food, as rhubarb, sorrel, tomatoes, celery, watercress, &c. ; (*b.*) imperfect oxydation of saccharine, starchy, and oleaginous principles of food ; (*c.*) increased tissue-metabolism, whereby the fatty acids found in excess are incompletely reduced ; (*d.*) excess of lactic and butyric acids, formed in intestinal dyspepsia, insufficiently reduced ; (*e.*) excess of mucus in urinary channels, which tends to ferment and favour deposition of oxalates ; and (*f.*) ingestion of water rich in lime-salts.

Prout considered that the fact of oxalates appearing in the urine after partaking of food containing oxalic acid indicated feebleness of digestion, inasmuch as a healthy stomach should convert small quantities of this acid into more disposable matters, as carbonic acid. In graver cases, when the mal-assimilation resulted from imperfect transformation of ordinary food, there is usually present a form of catarrhal dyspepsia affecting the whole alimentary canal. The liver is disordered in such cases, the biliary discharges being varied in colour, the motions acid, and covered with mucus.

I have certainly met with discharges of oxalate of lime in persons of gouty habit, and suspect that the tendency to the forms of dyspepsia and mal-assimilation which lead to oxaluria is closely allied, if not quite akin, to that prevailing in the gouty. The articles of food which are bad for those with tendency to oxaluria are just those which are ill-borne by the gouty, and the dietetic conditions for the one are proper for the other. Urates and oxalates often co-exist in the urine of the gouty dyspeptic.

It was formerly believed that oxalates were derived from the subsequent decomposition of the uric acid in the urine passed. It is now known that oxalic acid results from free oxydation of uric acid, and that this active oxydation can proceed in the system in disorders attended by free metabolism and oxydation.

The amount of oxalates passed in some cases is, however, much larger than can possibly be accounted for by the amount of uric acid either in the body or in the urine, and hence must commonly own other sources, as has just been indicated.

A tendency to boils and carbuncles has been noted in the subjects of persistent oxaluria, and the same is found sometimes in those suffering from glycosuria.

The relation of oxaluria to gout may, therefore, be defined as indirect, and dependent on the degree of primary and secondary faults in the digestive processes. No directly gouty symptoms are referable to excess of oxalic acid, but its presence in undue amount may be associated with forms of dyspepsia and with mental depression, to which the gouty are obnoxious, and on which many of the manifestations of gout depend. Oxaluria may thus be a harbinger of more overt gouty symptoms, and, as such, may help as a guide in the treatment, by way of prevention, of future gouty troubles.

With respect to calculi of calcium oxalate, it may be stated, generally, that they are far less common than those of uric acid, and that the calculi met with in the gouty usually consist of the latter. Sometimes, the concretions consist of alternate layers of each.

9.—Relation between Gout and Splenic Leuchæmia.

In some cases of splenic enlargement uric acid has been observed to pass freely out of the body by way of the kidneys.[1] The spleen is now regarded by physiologists as a temporary storehouse for uric acid in cases of retention of this matter. The liver is also believed to retain much uric acid when there is defective excretion of it. Cases of splenic leuchæmia might, therefore, be expected to furnish examples of gout; but such an association has not been hitherto found with any frequency. Ebstein affirms that gout and leuchæmia never occur together.

The following cases are the only ones known to me:—

(1.) An adult male had become pallid, and felt weak for eighteen months or two years. The spleen and liver were much enlarged. The leucocytes numbered one to five red blood-globules. No history of ague. The urine contained a trace of albumen and urates, but no uric acid crystals were thrown down in it. No family history of gout, and no lead-impregnation. After a time an acute attack of gout supervened in the left great-toe at night. Under colchicum the arthritis passed off in a few days. A year previously a similar attack of gout occurred in the same toe.

[1] In some cases of splenic leuchæmia, the amount of uric acid excreted has been found to vary from twice to seven times the normal amount. Renal calculi of uric acid are sometimes formed and passed.

The following case came under my care in 1880 :—

(2.) W. F., æt. fifty-six, a printer, had gout in right great-toe fourteen years ago, a year afterwards in right elbow, and since in elbows, wrists, shoulders, neck, and right hip-joint. More attacks on left than on right side of body. No lead-taint recognizable. Arteries thickened. Tophi on knuckles and both ears. Skin smooth, eyelids puffy. Acute attacks always begin in the daytime. Present attack in right wrist and hand. Is the eldest son. Maternal grandfather had gout. Parents free. Has taken "all the pills famous for gout." Urine, trace of albumen, several nocturnal micturitions. Treated with iodide and bromide of potassium, and solution of veratrina (gr. x. ad f. ℥i.), painted over painful parts. Relieved in two days (sixth of paroxysm). Skin desquamating on ninth day over late seat of pain. In two weeks great improvement. Bark and nux vomica given with iodide of potassium, afterwards iodide of iron. This patient was in the hospital eighteen months previously with enlarged glands on both sides of the neck, and had an attack of gout. The enlargement subsided for twelve months, and then returned. Six months ago the glands were enlarged in the groins. Three weeks ago those in the neck and axillæ swelled, and the spleen was found much enlarged, measuring 6½ inches long, by the same in breadth. Increase of leucocytes found in blood under microscope. Six months later the spleen measured nine inches vertically. Recent attack of gout in both knees with much effusion. Three months subsequently, renewed attacks of gout and much splenic pain. Left axillary glands much enlarged. Urine 1.010 with trace of albumen. (Patient lost sight of afterwards.)

10.—Gout in Relation to Purpura.

Purpura may be associated with gout, as in the following case, which was under my care some years ago. It affords an illustration of hæmorrhagic tendency supervening in a man of strongly developed gouty diathesis under the influence of privation.

A Case of Purpura Hæmorrhagica in a Gouty Man.

Reported by Mr. Sydney Davies, B.A., M.B.

Frederick C., æt. forty-four, brushmaker, was admitted to St. Bartholomew's Hospital on the 16th of August. He presented the appearance of a well-nourished man of moderate size.

The patient gave the following history :—He had enjoyed very good health up to the last five years. About that time he became subject to gout, of which he has had since then, several attacks in the feet, knees, and left hand respectively ; the index, middle, and little finger had twice been the seat of the disease, the last occasion being only three weeks ago, and the pain in the index and little finger remains at the present time.

Contemporaneously with the last attack of gout (*i.e.*, three weeks ago) he was seized with pain in the neck, which was followed at the end of a fortnight by a swelling in the right posterior cervical triangle. This swelling had increased progressively till the present time. The day before admission he noticed that he was covered all over the body with purpuric spots, but chiefly on the legs. Since the morning of the day on which he was admitted, he has had severe epistaxis. At the same time that the tumour disappeared in the neck his voice became hoarse, and had remained so ever since. He had been unable to work for five weeks, and for the last three weeks had taken very little food. He had been an average drinker. The

family history had been one of gout on the mother's side, herself, her father, and two sons having been affected with it.

On admission the patient had a sallow complexion and a very hoarse voice. In the lower part of the posterior cervical triangle of the right side there was a firm, hard, immovable tumour, about the size of half an orange, and of a dark colour; the tumour was well-defined, and exhibited fluctuation and pulsation; it was painful, even when not handled. His body was covered with purpuric spots, which were most numerous on the legs. The lungs and heart presented no marked abnormality. The urine was acid, and contained a trace of phosphates, but no albumen. His evening temperature was 102°. Neither the liver nor spleen was found to be increased in size.

The following notes will best indicate the progress of the disease :—

August 17th.—No pulsation, and less pain in the cervical tumour; morning temp. 99°, evening temp. 100.9°. The patient less feverish. Did not sleep well. Tongue fairly clean.

18th.—A few purpuric spots have appeared on the tongue, those on the body are fading. The patient has a good deal of irritation about the larynx, which kept him awake during the night. Pain and tenderness on percussion at a spot about two inches below the left clavicle. Pulse intermittent. Temp. 98.8° and 99.4° respectively. Hæmaturia; gums spongy.

19th.—Patient does not feel so well; more feverish. Soreness of throat. Tongue thickly coated with brown fur; oozing from gums. The tumour has assumed a dusky greenish hue. Urine nearly black with thick sediment, giving blood-reaction; contains one-sixth albumen. Spots on legs fainter. Pulse very feeble. Morning temp. 98.8°, evening 101.2°.

20th.—Hæmoptysis. Tongue cleaner; the purpuric spot is ulcerating; gums bleeding. Tumour less tender, greenish-blue colour. Nausea, appetite worse, great thirst. Pain in testicles and bladder, more severe in the latter before and after making water. Hæmaturia increased. Pulse variable, very weak and frequently intermittent. Heart-sounds very feeble. Morning temp. 99.2°, evening 100.6°.

21st.—Feels very weak, appetite increased, voice clearer, less cough. Temp. morning 99.6°, evening 100°.

22nd.—Temp. morning 99.4°, evening 101.8°.

23rd.—No cough, voice improved. Hæmorrhage from gums less. Tongue clean. Sediment of urine seen under the microscope to contain red blood-corpuscles. Some blood from the finger was also examined by the microscope, and found to contain an excess of white-blood cells. Temp. morning 102°, evening 100.8°.

24th.—Patient very pallid and weak, feverish, and thirsty. No hæmoptysis, and less hæmorrhage from the gums. The swelling in neck has become more diffluent. Hæmaturia has diminished, and albumen has disappeared from the urine. Temp. morning 102.6°, evening 101.4°.

25th.—Pulse 124. Patient thirsty, and takes food well. Has passed eight pints of water in twenty-four hours. Temp. morning 100.6°, evening 101.6°.

26th.—Pulse 134, regular. Patient feels better. Tongue coated, rather tremulous. Bowels confined; flatulent distension of abdomen. Takes food fairly well. Very anæmic. Has passed seven pints of urine, of natural colour. A few large petechiæ on the abdomen. Temp. morning 99°, evening 100.6°.

27th.—Great dyspnœa; no cough; no pain in the chest. Tongue cleaner; bowels open twice. Abdomen distended, tympanitic. Vomiting. Pulse intermittent. Respirations 46. Morning temp. 99.6°.

The patient died in the middle of this day.

Treatment.—An ice-bag was applied to the blood-tumour in the neck, and appeared to stop its increase and cause its resolution. The general treatment included the use of fresh vegetables, of remedies such as ergot, ice, and other styptics, but no treatment appeared to materially influence the course of the disease.

A *post-mortem* examination was made, and gave the following results :—

There were small subcutaneous hæmorrhages on the front of the thighs and abdomen. Hæmorrhages, about the size of half-a-crown, were found on the under surface of the dura mater, to the right of the median line near the vertex. There was a patch of dark staining on the anterior surface of the heart, and some small hæmorrhages beneath the endocardium of the left ventricle. The intestines were much distended with gas, and contained greenish-black, pultaceous fæces. No spots of hæmorrhage were seen in the stomach or intestines. Spleen and liver were normal. The pelves of both kidneys were darkly stained, and a little blood could be scraped from the mucous membrane. The bladder exhibited one or two spots of extravasated blood near the neck. The cervical tumour was found to contain an accumulation of reddish fluid matter, probably the result of altered blood-extravasation.

Hæmatinuria.—In one case of paroxysmal hæmatinuria in a man under my care, an attack of gout occurred.

11.—Gout in Relation to Hæmophilia.

A connection between gout and this variety of the hæmorrhagic diathesis has been affirmed by various observers for sixty years past. Most modern writers on the subject of hæmophilia deny, or attach little importance to, such a connection. According to Legg,[1] true gout is extremely rare amongst those who thus suffer, and, as he points out, this is readily conceivable because of the youth of the majority of the patients.

The fact that the joints may suffer specifically in hæmophilia has, no doubt, been one reason for the belief that there is a gouty element in such cases.

It is, however, certain that history of true gout, and phases of incomplete gout, may be met with in the ancestors of some of these patients.

In analyzing the cases, seven in number,[2] reported by Legg, I find the following facts bearing on this point:—Case 1. The maternal grandmother was subject to gravel in the kidneys, and had passed several small stones.—Case 2. A brother of the patient, æt. twenty-five, is stated to have had "chalk-stones up the sides of his feet."[3] The father and his relations were gouty. One paternal uncle was gouty. Case 5. Father had chalk-stones in the hands, and all his family were subject to gout.

The disease is markedly hereditary, being handed down by the females to the males, who are the chief sufferers. Females suffer rarely, and only in mild degree from it, the joints not being, as a rule, the seat of effusion, but only of pains. Menorrhagia may be the only expression in a female bleeder. Amongst determinants

[1] A Treatise on Hæmophilia, 1872.

[2] One in Path. Soc. Trans., vol. xxxiii.; one *ibid.* vol. xxxvi.; and five in his monograph.

[3] Tophaceous gout sometimes occurs early in life.

of attacks of hæmorrhage in these cases, apart from traumatism of every degree, are exciting or depressing emotions, sudden variations of climatic condition, and exposure to cold and damp.

The attacks are preceded by euphoria, as is the case often in epilepsy and gout.

Articular attacks with effusion of blood into the joint induce, or are associated with, pyrexia, which may reach 104° or 105°. In 1829 Rieken (quoted by Legg) described hæmophilia as an anomalous variety of gout, and asserted that (1.) the tendency to extreme hæmorrhages has been of late observed only in those persons whose parents or grandparents have suffered from gout; (2.) in those members of "bleeder" families who have escaped the tendency to hæmorrhage, gouty paroxysms may often be observed; (3.) in bleeders themselves gouty paroxysms are nearly always seen, and sometimes an alternation of the joint-affection with the bleeding; (4.) gout is a disease which stands in a very close relation to the blood and blood-vessels, and often appears to be a direct cause of hæmorrhage.

Legg disputes each of these propositions, but I cannot agree with his reasoning. I do not regard it as probable that all cases of hæmophilia can be traced to ancestral gouty influence alone; but the occurrence of gouty history, so far as already proven, appears to me too important a factor in the ætiology of the disorder to be quite disregarded. The difficulty of securing trustworthy history of true gout in any ancestry is not slight, and is especially great in the case of patients of hospital rank. Rieken probably regarded the painful and tumid joints of active hæmophilia as examples of gouty arthritis. To dispute the fourth proposition, as does Legg, by affirming that in fifty cases of well-marked gout he found not more than three who had suffered from hæmorrhoids, and none who had had bleedings, appears to me unwarrantable, since the subjects of regular gout are seldom those who suffer from its incomplete manifestations, amongst which are hæmorrhoids and hæmorrhagic tendency. I should not expect to find a coalescence of regular gout with hæmophilia. Bleedings are not frequent in true gout, but are common enough as part of the general gouty habit, and more markedly so in females.

If regard be had to some of the leading features of gout and of hæmophilia, a conviction arises that there is an alliance or a degree of relationship between the two states. We may set out by way of parallel the following points relating to each disorder :—

Gout.	Hæmophilia.
Heredity strongly marked.	Heredity strongly marked, also, gouty heredity not seldom.
Females much less affected.	Females much less affected.
Females less liable to overt gout; bear gouty sons.	Females less liable, bear sons who bleed.
Attacks sudden, paroxysmal.	Attacks sudden, paroxysmal.
Attacks preceded by euphoria.	Euphoria preceding attack.
Determinants traumatic, climatic, psychical, dietetic.	Determinants traumatic, climatic, psychical.
Antecedent cumulative plethora.	Antecedent cumulative plethora.
Predilection for joints. Arthritic diathesis.	Predilection for joints, the larger more particularly; sometimes, great toe-joint affected. Arthritic predisposition.
Arthritis leading to degeneration of cartilage with specific deposits, ankylosis, synostosis.	Arthritis with degeneration of cartilage, fibrous ankylosis.
Chronic skin-diseases associated.	Chronic skin-diseases associated.
Hæmorrhagic tendency in incomplete gout, as met with in descendants of the gouty.	Hæmorrhagic tendency.
Tendency to recurrence of attacks, articular and other.	Tendency to recurrence of attacks, articular and other.
Alternation of articular attacks with other abarticular manifestations.	Alternation of articular effusion with free hæmorrhages, *e.g.*, hæmaturia or epistaxis.
Epilepsy occasionally associated.	Epilepsy occasionally associated.

By way of contrast we may set out the following points in the two disorders:—

Gout.	Hæmophilia.
Occurrence in middle life, as a rule.	Occurrence within first two years of life.
Largely dependent on dietetic causes.	Not directly dependent on dietetic causes.
Smaller joints affected more often than larger, *e.g.*, great toe, ankle, knees, fingers, elbow.	Larger joints affected chiefly, *e.g.*, knee, ankle, elbow, shoulder, hip; the digits rarely.
Pyrexia moderate in acute attacks.	Pyrexia severe when joints involved.

Dr. Barlow has related to me a very noteworthy case in which a young man who had hæmophilia with epistaxis, hæmaturia, and effusions into the joints, became the subject of uratic tophi on the ears.

In view of the predominant features of hæmophilia, it is, I believe, hardly possible to resist the conviction that there is a relationship between this disorder and gout, as understood in its widest sense. We are certain of gouty ancestry in a goodly proportion of the cases. A marked characteristic of hæmophilia is the tendency to recurrence. It would, thus, appear that the disorder is allied to certain recurrent illnesses which grow up from time to time till by accumulation they become manifest. Where

the bleeding tendency is very marked, no measure of precaution avails to avert attacks. This is so with gout. A safe equilibrium is then only maintained with difficulty, and very slight provocations suffice to determine outbursts in various forms. I regard severe hæmophilia as a gradually cumulative plethora, which must perforce discharge itself.

I therefore agree with Hutchinson that a possible explanation of this peculiar malady is to be found in peculiarities of vascular structure, developed originally by gout, which have become modified and specialized by transmissions through many generations. If this be the case, it is intelligible that, in the subjects of this new evolutionary disorder, we do not often find symptoms of overt gout. We must have regard to the type presented, and in this line of investigation we come to see a likeness in habit, and a predilection for tissue which recalls some of the recognized manifestations of the gouty diathesis. It is far from uncommon to meet with hæmorrhagic histories in the descendants of the gouty, epistaxis and menorrhagia in severe degrees being perhaps most frequently noted, while intracranial and retinal hæmorrhage are less so.

Cases of sporadic hæmorrhagic tendency I regard, with Hutchinson, as distinctly and closely allied to the graver form of true hæmophilia.[1] A study of sporadic cases of any disease is often strongly suggestive, and helpful to a better comprehension of its ætiology.

It would nowadays be little more than pedantry to deny the relationship of certain morbid states to the gouty habit, in its widest aspect, because one cannot place one's finger on a tophus, or demonstrate sodium urate in the blood of the affected individual. This is assuredly not the solitary touch-stone for all ailments owing dependence on an original gouty state. As I have already had occasion to remark, there are many perturbations in gout beyond those of uric acid, and many associated profound tissue-changes. It is not hard to conceive that some only of these variously impressed textural characters may be transmitted, and passed on, too, with variations, so that new evolutionary phases of disease come to be manifested in the remote descendants of those goutily disposed.

As with gout, so with hæmophilia, the nervous system is markedly involved; thus, the determination to the joints and the occasional paroxysmal features of the disorder afford, amongst other symptoms, indications of its specific influence.

[1] Pedigree of Disease, p. 25.

Instances are not wanting in which associated instability of the nervous system in hæmophilia has been declared by epilepsy.

12.—Relation between Gout and Traumatism.

I have already stated that gout may supervene in paroxysmal form in those goutily predisposed, as a result of shocks and injuries. Many instances are on record. Thus, a fall from a horse may determine an attack. Sprains of joints may evoke gout in them. The shock, mental and bodily, of even minor surgical operations may be provocative; thus, removal of tumours, of a tooth, ligature of piles, operation for cataract, and so trifling an irritant, according to Heberden, as a gnat-bite.[1] Vaccination has been known to induce a paroxysm in a man aged fifty. Paget has known a patient suffer a sharp attack after each of three operations which he had undergone. Operating surgeons, certainly in London, are familiar with such cases. The great toe-joint, being much exposed, is often the site of gout from injuries almost unnoticed at the time of infliction, and if the attack be a primary one, it is often attributed to the injury alone. Tight boots may be the cause. The part injured may not be the site of the attack, the gouty process fixing on some other—by preference a joint. Parts much used are especially liable to attack, as the ball of the thumb, wrist, knees and feet of riders, and the soles in painters and those working on ladders.

Parts once injured may long afterwards become the elective seat of gout. Phlebitis may occur in the saphenous vein and tributaries of the external popliteal vein from friction of stirrup-leathers, and renewed attacks may be experienced in the same veins at later periods without fresh provocation.

"Nothing," remarks Sir James Paget, "can show better than gout sometimes does how exactly health is, in some persons, just maintained; how nearly balanced in them are health and disease, comfort and misery. A person on whom I could rely assured me that within five minutes after breaking his forearm, while he was in what he thought good health, he had an attack of gout in his hand."[2]

Severe hæmorrhages, as hæmatemesis or epistaxis (traumatismes internes), sometimes lead by the shock which they occasion

[1] "Idem quoque interdum evenit, ubi membrum ab arthritide jam convalescens ictu aliquo, aut distortione, aut etiam culicis punctione, læsum fuerit."—*De Arthritide.*

[2] Clin. Lect. and Essays, p. 354.

to paroxysmal gouty attacks. A blow on a given part in a gouty person has been followed by the formation there of tophus. Tight boots have been blamed for determining gout in the great-toes of our immediate ancestors, but podagra existed classically when sandals were worn.

It is noteworthy that the same kind of vulnerability is met with in some persons of rheumatic predisposition. Monarthritis may follow injury to a joint, and determine, reflexly or otherwise, the spread of multiple rheumatic arthritis, illustrating the common basal arthritic diathesis in both cases.

It thus appears that goutily-disposed persons are often very vulnerable in their textures,[1] and this peculiar sensitiveness exists as a part of the specific nature of the malady when once established in the system. The original injury would appear to lower the tissue-vitality, and render it a specially susceptible part, a *locus minoris resistentiæ*.

Such trophic change is also well-known to be one of the determining factors in the localization of new growths, and, in particular, of malignant tumours.

It cannot be doubted that any prevailing habit of body exerts an influence on the repair of injuries and wounds in the individual affected. Hence, traumatic conditions are apt to be modified in the subjects of gout. Injuries to joints are thus recovered from tardily.

According to Paget, when, in a patient of middle or later age, an injured joint does not recover in due time, gout may be suspected. The reparative process in a wound or bony fracture may be temporarily arrested by an attack of gout in the part; on its subsidence, healing may proceed quite favourably.

The influence of gouty cachexia on traumatism is that which pertains to any cachectic state. The presence of anæmia or glychæmia, cirrhosed and inadequate kidneys, thickened arteries, and the low vital power thus entailed, will suffice to explain the facts that wounds in such subjects often heal slowly, perhaps bleed unduly, or are prone to low septical or erysipelatous inflammations.

The susceptibility of the skin to certain irritants, as arnica and iodine, has been specially noted in persons of gouty disposition, and must be considered in relation to treatment by such applications.

The influence of shock, either mental or bodily, in precipitating

[1] "Persons thus combustible are not rare. You may liken them to lucifer matches; gout explodes in them whenever they are roughly handled."—*Paget.*

a paroxysmal attack of gout, illustrates as well the unstable neurotic element present in gouty persons as the tissue-peculiarity. No mere humoral conception of the disorder suffices to explain some of its most marked features. The effects of injury or of operation tell both locally on the part and centrally on the nervous system, and the explosive result may be manifested either in the damaged texture, or at some distant part which may, or may not, be reflexly related.

13.—Gout and Osteitis Deformans.

The peculiar disease of the bony skeleton to which the term "osteitis deformans" was applied by Paget has been met with in, perhaps, the majority of instances in persons of gouty habit or inheritance. Paget declares that this disease "has appeared in no usual relation, whether by inheritance or coincidence, with any other disease except gout."[1]

By the kindness of Dr. Barlow, I am enabled to append the notes relating to a case of osteitis deformans in which gouty symptoms and uratic deposits occurred.

Rev. Mr. X., aged sixty. First seen by me August 11, 1885. Gave history of maternal grandfather having had gout. Patient himself had had several attacks of monarticular gout (big-toe). Gets pains in knees and feet if he walks much, but has felt necessity of exercise, and for several months has ridden a tricycle with advantage to general health. Two months before I saw him had suffered from some sharp pains on left side of chest, with short breath. Relieved by leeches, and able to be about in one week. Consulted me now on account of breath being short, and a little skin-eruption.

Condition, August 11, 1885.—Rather square-set, well-built man. Fair general nutrition. Left metatarso-phalangeal joint a little thickened; no tenderness now. Both legs, osteitis deformans; right presents general bowing outwards, and tibia is slightly but definitely thickened. No tenderness, and no separate node. The bowing and thickening quite different from old rickets. The left leg also bowed out slightly, but no definable thickening. Bone-ends not obviously altered; joint movements free.

No other osseous abnormality.

Slight chronic eczema on both legs, and a little over sacrum.

Lungs—a little wheezing at bases.

Heart-sounds natural. Pulse not hard. Brachial artery not tortuous.

Tongue nearly clean. False teeth.

No tophi.

Urine high-coloured. No albumen; no sugar.

I saw him next on March 30, 1886. There was then a very little puffiness under the eyes. He was complaining again of his bronchitis, though there was only a little rhonchus to be heard.

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[1] Med.-Chir. Trans., vol. lxv. p. 235, 1882.

Vide vol. lx. p. 37, 1877, for original account of the disease. Cases are related in both communications.

Next note is on March 7, 1887. He was then just recovering from a bad attack of gout, from which he had suffered for one month. Both feet had been affected and some of the finger-joints. There was a little œdema of the dorsum of each foot still present, though no heat or redness. The bronchitis and eczema had gone. His pulse was a little intermittent. Urine clear, dark-coloured, free from albumen. No tophi.

Next note, April 5, 1888. Complains of irritation, especially below the scrotum and in the perineum. There is a very little eczema there.

Tongue clean.

Urine dark-coloured. No deposit ; no albumen ; sp. gr. 1.015.

Now an undoubted tophus on edge of left helix, and (query) commencing small tophi on the edge of right lower eyelid.

My impression is that the bowing of the legs is slightly more marked than when he first presented himself, but the limb-condition is attended with very little discomfort. He can walk fairly on the level. General nutrition maintained.

14.—The Influence of the Gouty Habit on Specific Febrile and Acute Diseases.

There is little knowledge respecting the modification of specific febrile states or of acute diseases by gouty influence. In the young this habit is seldom detectible, or but rarely presents suggestions of its presence. Hence, it is not possible to gain trustworthy evidence of any peculiarities attaching to the offspring of the gouty while the subjects of the exanthemata or of acute disease.

My own experience fully accords with that of Murchison, who taught that persons of the "lithic acid dyscrasia," or lithæmic subjects, are more than others prone to ordinary febrile colds, and to unusually severe local inflammations. The gouty habit predisposes to local inflammations either by inherited tissue-peculiarities, or because of the altered blood-condition which may supervene from time to time.

Diphtheria.—Without doubt, such tolerance as is exhibited by the gouty in later life under the ordeal of acute disease will depend largely upon the structural condition and functional adequacy of the kidneys. In this connection the following case of diphtheria, recorded by Pye-Smith, is of interest. It was that of a man, aged forty-five, who died of uræmic eclampsia and coma, and whose kidneys together weighed only five ounces. While in Guy's Hospital he was attacked with diphtheria, and recovered, though he was before suffering from gout and albuminuria.

Typhus Fever.—In respect of typhus fever, the gouty habit is, according to Murchison, a very serious complication. He never knew a gouty person attacked with typhus recover. The risk is that of unsound kidneys, which always prevent recovery from this disease, and the fact is perhaps to be taken along with that relat-

ing to the age at which typhus kills, since persons over fifty years almost always die from it.

Pneumonia.—When pneumonia occurs in the gouty, it is not seldom itself a gouty manifestation, and is not so fatal as might be expected. Its onset and its disappearance may be somewhat sudden, and it may sometimes be plainly relieved by other gouty manifestations. The condition of the kidneys and other textures—no less than the age of the patient—determines the gravity in each case. If there be already present emphysema with chronic bronchitis in a gouty person attacked with pneumonia, the prognosis is rendered as grave as possible. Emphysematous lungs are commonly intolerant of the stress of acute lobar inflammation. An "arthritic" pneumonia is, however, less likely to be fatal than other forms.

Erysipelas.—Prout believed that erysipelas was unfavourably influenced by the gouty habit. He refers, however, to cases of gouty glycosuria occurring in middle life, where there is probably an enfeebled state of body. Dr. Gregory, of Edinburgh, observed, and I have noted the same, that the daughters of gouty men were particularly liable to attacks of erysipelas.[1] Scudamore [2] also noted this, and remarked that erysipelas appeared to represent, or come instead of, the expected fit. Dr. Copland [3] mentioned amongst predisposing causes the gouty diathesis. In the case of erysipelas, as in that of typhus fever, the gravity in any gouty patient is almost certainly in relation to the general state of nutrition and the adequacy of the kidneys. In low states of health the poison of erysipelas is certain to work in malignant fashion, and it is under such conditions that spontaneous gangrene sometimes occurs in the subjects of gouty cachexia with bad arteries, even when glycosuria is not a dominant feature.

It must be exceedingly rare for paroxysmal gout to occur at the same time with acute diseases. As the exanthemata are met with chiefly in the earlier decades of life, it is very unlikely that such a combination or coincidence should arise.

A touch-stone, as it were, for arthritic and other habits of body is sometimes forthcoming in the sequelæ of fevers and various acute illnesses. Thus, after enteric fever there may be subacute arthritis, also venous thrombosis. In such cases I have sometimes ascertained arthritic heritage or proclivity.

[1] Sir Robert Christison informed me of this.

[2] *Op. cit.*, p. 531. [3] Dict. of Medicine.

15.—Influence of Gouty Habit on Painful Affections.

It is certain that gout often aggravates the painfulness of painful processes. It may affix a paroxysmal character to them. The gouty have commonly undue sensitiveness, and suffer more than others from ordinary sources of pain.

One of the leading ideas about gout anywhere is its painfulness. Were gout nothing more than a mere inflammatory process, or goutiness but discomfort without pain, it would disturb its victims far less than is usually the case. But, in truth, most of the manifestations of gout are painful, and some exceedingly so. This is part of its specific character. A joint acutely involved by rheumatism is commonly but little painful unless it be moved or handled. A gouty arthritis is exquisitely painful when absolutely at rest. Those who have suffered both from rheumatism and gout, or who in the course of a single illness have attacks of each (in a truly commingled case), can clearly distinguish the respective pains of each. A notable instance of this kind was once under my care, and the man could tell at any time whether he was more gouty or more rheumatic.

The pain in gout is disproportionate to the apparent degree of arthritis. This fact, I conceive, tends to show that there is a special nervous erethism in the gouty. They all bear pain badly. Response to every source of irritation is heightened, not, I believe, by the manifestations of the disease, but by the essential nature of the malady. Such persons as are gouty would not be so if they did not possess, as part of their innate nervous disposition, a specially intensified susceptibility, and a tendency to explosive neurotic manifestations.

Paget tells of a pyæmial abscess which was very painful in a gouty man, and he believes that some cases of cancer are rendered specially painful by inflammations in goutily disposed persons.

Great painfulness attaches to even simple disturbances in the gouty. Not to mention here the agonising neuralgiæ due to gout, it may suffice to recall the special sensory disturbances attaching to the skin-diseases dependent on this habit, the pains of indolent furuncles, those deep-seated pains in the heel, sole, coccyx, muscles, tongue, teeth, and ensiform cartilage, and the incoercible cramps of the calves met with in the gouty. All these may be unduly severe, and some of them agonising.[1]

[1] Professor Ball, of Paris, has recorded the case of a gouty patient who never had a pain anywhere, however transient, without a tophus immediately forming there.

16.—Pyæmic Arthritis and Gout.

Pyæmia may, sometimes, supervene in gouty as in other persons, and the attendant phenomena may prove puzzling. It is easy to be wise after the event, but in some cases pyæmia may arise insidiously from a very small and latent purulent focus, and set up articular inflammations, which it is only too easy to consider as "rheumatic" or "gouty" in the subjects of arthritic proclivity. There may be severe pyrexia, which is greater than obtains in any form of true gout, but there may also be absence of any characteristic rigors, and of high flights of temperature. The diagnosis is not difficult if the latter should occur.

Cystitis and suppurative foci may arise insidiously in the subjects of chronic gout, and with some frequency in cases of chronic glycosuria with cachexia.

At the autopsy in such cases may sometimes be found, together with the presence of pus in the articulations, kidneys, &c., old changes in joints due to gout, to wit, erosion of cartilages and encrustation of urates with ostitis. There is commonly little to be done to save the patient in these malign cases, but it is at least proper that a correct diagnosis should be made during life.

The subjects discussed in this chapter respecting the influence of gout on various constitutions and diathetic states have attracted much attention at the hands of French physicians, and they have, accordingly, sought to classify gout under several varieties or types. These have not been commonly accepted by British authorities, at all events in modern times, with the exception, perhaps, of Laycock.

I have hesitated to adopt this teaching of the French school, and preferred to treat the subject, a confessedly difficult one, with less definition and dogmatism.

The classification of Durand-Fardel relates to gout as affecting those of sanguine, bilious, nervous, and lymphatic constitution. Lecorché describes five types founded on the predominant localization of the disorder, viz., articular, nephritic, muscular, neuropathic, and gastro-hepatic. These varieties have already been considered with respect to the several tissues and organs as affected by gout.

For clinical purposes it may be necessary to have regard to the predominant features in any given case of the disorder, but in all there is a basic unity of type. The important point is to recognize correctly the truly gouty element in any case.

In practice it is not always possible to fit the cases to the

particular types, and, indeed, several of these may be present in a single individual.

The possibility of new phases of gout, as of other diseases, arising in the course of time must be borne in mind. By variation in transmission, by coalescence with other states, and by altered modes of life and diet, it is at least conceivable that evolutionary changes may occur whereby some of the features of this disease, hitherto regarded as classical, may be less clearly marked, or even disappear, and thus new forms of gouty manifestation may come before future observers. Sir James Paget has directed attention to this large question, and in respect of gout has instanced the occurrence of phlebitis as a possible outcome of variation in transmission.

There may possibly be another such example in the case of subcutaneous nodules, which certainly were formerly unrecognized, and appear to be new manifestations of the arthritic diathesis.

In considering the varied possible comminglings of gout, it must also be borne in mind that this disorder may develop in persons owning arthritic heredity in very varying degree, or may grow up anew in persons of other diathetic habits. Hence, we find all varieties of goutiness in persons who present no obvious physiognomical traits of the disorder, as in purely nervous or spare subjects whose constitution is frail. Amongst these are examples of "poor," and many of "incomplete" gout, the latter including cases of visceral, and of what has been badly termed (as I think) "nervous gout." The disorder is perhaps only slightly indicated in some member of a gouty family, perhaps a female, while in a more robust brother it appears in more overt and vigorous form.

In these irregular or incomplete cases we have an implantation, or grafting, of the gouty on other diathetic habits. For the purpose of successful treatment of the various troubles thus arising it is important to recognize this coalescence.

Holding very strongly, as I do, the views already expressed in this and the preceding chapters respecting the wide relationship and multiform phases of gout, I must here express my complete dissent from the following passage, which occurs in the second edition (1888) of Fagge's "Principles and Practice of Medicine," edited by my esteemed and very able friend, Dr. Pye-Smith :—

"For some reason it has become common to ascribe bronchitis, dyspepsia, gastralgia, iritis, gravel, cystitis, and urethritis, phlebitis, eczema, and even psoriasis, to a gouty diathesis. But the evidence is very slight, and the 'gout' to which such evidence as there is applies is the distillation of morbid humours which belong to a bygone pathology, not deposit of urate of soda in the tissues."

I venture to hope that, in respect of gout, the pathology of the future, as elucidated by that which most especially concerns us as practical physicians—the clinical side of it—will help to enlarge our conceptions of the disease as a whole, and to bring into closer correlation the many and varied aspects of it.

CHAPTER X.

GOUT IN RELATION TO VARIOUS NEUROSES.

THE occurrence of gouty habit in the ancestors of persons exhibiting many neurotic disorders has not escaped the attention of the careful clinical observer. The same cannot be affirmed with respect to the scrofulous habit. It is of high importance to recognize the fact of special predisposition on the part of gouty inheritors to instability of the nervous system.

To the varied manifestations of the neuroses, and of their peculiar tendency to alternate in successive generations, I have already referred. Thus, we meet occasionally with forms of insanity, with epilepsy, asthma, angina pectoris, and cardiac neuroses (vascular), headache, hemicrania, neuralgia, vertigo, and the whole class of disorders included under the terms hypochondriasis and hysteria (neuromimesis). It is certain that in the families of many subjects of these disorders a distinct history of antecedent arthritic conditions may be obtained, and if such be found, it is possible that a clue to more efficient treatment may be gained thereby. The fact is of supreme importance in relation to the part played by the nervous system in gouty manifestations generally.[1]

I have, perhaps, already sufficiently insisted on this part of the pathogeny of gout, and directed attention to the peculiar instability of the nervous system in the gouty. I shall now briefly treat of the various neurotic ailments just mentioned, and endeavour to trace the various indications of arthritism presented by them. They are usually discussed by authors under the head of irregular gout.

Gout in Relation to Insanity.—Mania has been met with on the cessation of paroxysmal gout, and has yielded on the supervention of it.

[1] In Dr. Syers' 500 cases of acute rheumatism, already referred to, he found antecedent neurotic history in 16 per cent. of them. Lancet, June 30, 1888.

Dr. Rayner,[1] of Hanwell, supports the views of Berthier, which go to prove that every form of insanity may be produced by gout. He has recorded an instance in proof of the first allegation, and Garrod mentions others. In atonic articular gout with general debility, he noted two cases where hallucinations of sight and hearing, producing great suspicion and distrust, occurred, the patients recovering after an attack of gout. In cases of imperfectly developed gout, he mentions cases where there were delusions, at first exalted, then becoming melancholy, an attack of frank gout causing the disappearance of these symptoms. In saturnine gout he met with proptosis and an extreme darkness of complexion, especially in melancholic cases, both symptoms diminishing as the health improved. He concluded that—

1. Protracted gouty toxæmia, when not very intense, usually results in sensory hallucinations, or melancholia.

2. Sudden and intense toxæmia results in mania or epilepsy.

3. Intense and protracted toxæmia usually results in general paralysis.

4. If there is a tendency to vascular degeneration from plumbism, alcoholism, &c., varying degrees of dementia are produced.

In the discussion on this paper, Dr. Savage declared himself in agreement with Dr. Rayner. Sir J. Crichton-Browne was of opinion that insanity only occurred in gouty patients who were hereditarily predisposed to it, or to epilepsy. He believed that many cases of melancholia attonita in young girls with feeble circulation were connected with inherited gout.

Gout in Relation to Melancholia.—Gout may alternate with attacks of melancholia, and the latter may replace an attack of gout. Excess of uric acid in the blood is apparently the determining factor. Dr. Haig suggests that there may possibly be found every gradation of psychical abnormality, from mere depression of spirits and bad temper up to melancholia and suicidal, or other, forms of mania, produced by uric acid retention, and he remarks that the diet which is useful in headache and epilepsy—largely vegetarian—is of use in some forms of insanity.[2]

In such cases, as Dr. Haig points out, and as Dr. Broadbent has shown, there is often present high arterial tension, which is known to vary with the amount of uric acid in the blood, and also to be amenable to restricted diet without animal food.

[1] Trans. Internat. Med. Congress, vol. iii. p. 640, 1881.

[2] Practitioner, November 1888, Mental Depression and the Excretion of Uric Acid, p. 342.

Dr. Savage has directed attention to cases of this kind, and recorded instances in which attacks of gout were coincident with complete relief to mental depression and mania.[1]

I have knowledge of cases of grave suicidal tendency, and morbid apprehensions of giving way to it, having at once yielded either to anti-gouty medication or to outburst of acute gout. It is obviously very important to be aware of such facts, both for diagnostic and therapeutic purposes.

Gout in Relation to Epilepsy.—Certain cases of epilepsy appear to be connected with the gouty habit. Instances have been recorded in which the attacks ceased on the supervention of regular gout, and Garrod found a large amount of uric acid in the blood in one such case. From this category are, of course, excluded all cases of convulsions which occur in the subjects of gouty cachexia, where with granular kidneys the fits probably depend on uræmia. The most noteworthy cases are those met with in younger patients of neurotic inheritance, who may present modifications of that directly inherited.

Attention has been directed to this class by Dr. Haig, who presents some forcible arguments in favour of the view that certain epileptics owe their malady to the effects of uric acid irritation as a direct excitant. We may, therefore, take a neuro-humoral view of such cases, for the existence of epilepsy as a product of urichæmia alone cannot, of course, be admitted. There must, I hold, always be the "nervous" factor in any case, consisting of an inherited proclivity to instability in certain nerve-centres. With this, it is not difficult to understand that accumulation of uric acid within the body may sometimes determine and precipitate an explosive paroxysm.

Gout, Epilepsy, Injury to Back.

B. J., æt. forty-six, formerly in army, and in the Crimean war, was admitted under my care in Mark Ward in July 1882. A man of large frame, slightly anæmic. First attack of gout at twenty-eight in feet. Six months ago fell down-stairs and hurt his back. Three months ago had a fit, was unconscious and bit his tongue. Some doubt as to an aura. No history of syphilis. No gout since he was thirty-three. The heart-sounds were clear but feeble. Tarso-metatarsal joints enlarged. Optic discs natural. On August 4th an attack of gout, left great toe-joint. The urine was void of albumen and glucose. Several fits of epilepsy occurred at intervals while in hospital.

Epilepsy (? Uræmic Eclampsia) in a Gouty Man.

R. P., æt. fifty-two, son of very gouty father, and formerly intemperate, came under my care in January 1876. First had gout at age of thirty in great toe and

[1] Insanity and Allied Neuroses. Lond., 1888.

knee-joints. None for last two years. Been a total abstainer for nearly three years, but broke his pledge last Christmas. An attack of gout recently. Has had four epileptic fits in last three years, occurring half an hour after going to bed. Worry or overwork appears to determine these attacks. The urine was 1005, and free from albumen, and the patient had cramps. His kidneys were probably in process of contraction.

Van Swieten records the following :—"I had the care of a man who was seized at first with severe pains in his lower belly, delirium, and strong tremor over his whole body. He afterwards became epileptic, and having suffered, in the space of a month, three severe fits of that distemper, a sharp fit of the gout at last seized upon his great-toe, and from the time he became gouty he remained entirely free from the epilepsy, and was always sure of having a return of the gout regularly twice a year." He quotes Hippocrates for the opinion that "capital disorders, attended with an extreme degree of violence, are in a critical manner cured by the sciatica."—*Commentaries on Boerhaave's Aphorisms.*

Amongst occasional predisposing conditions of epilepsy, Dr. Copland mentioned the gouty diathesis.[1] The evidence adduced by Dr. Haig in favour of this view is that fluctuations occur in uric acid excretion in some cases of epilepsy, just as in cases of gout and uric acid headache. The value of vegetarian diet in this disease, and the benefit derived from alkalies given with bromides in many of the cases, are also adduced in favour of this view.

In many cases there is family history of gout or gouty ailments. Iron, which is harmful in this class of patients, and leads to retention of uric acid, is commonly injurious in epilepsy.

Chorea.—No evidence of any force has been adduced to prove any direct connection between gout and chorea. This is the more noteworthy because the relationship between rheumatic habit of body and chorea has been, certainly to my mind, very conclusively proved for the majority of all cases. The seat of chorea is without doubt in the nervous motor centres, and rheumatism is a disease especially affecting motor structures, in particular the heart and joints. I regard chorea as a motorial neurosis, and believe that a common kindred vulnerability, or susceptibility, in the great motor centres may predispose, under certain excitants, to one or other, or both, of the disturbances known as chorea and rheumatism.[2]

Amongst my notes I find the case of—

K. R., æt. seventeen, a machinist, who came suffering from a second attack of chorea. The first attack occurred two years previously, and lasted for three months. She was one of six children. The eldest had had "rheumatism," but never been bedridden, and one had "rheumatism" in an ankle. I saw her father, aged fifty-seven, and he gave a history of an attack of gout at the age of forty-one, which affected his toes, ankles, and knees. He was a free-drinker of "four ale," porter, and spirits.

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[1] Dictionary of Medicine. London, 1858.

[2] *Vide* An Address on Chorea, Brit. Med. Journ., January 3, 1885, in which I have urged this view.

In the following case, for which I am indebted to my colleague Dr. Gee, hemichorea occurred on the right side, and was probably due to hæmorrhage from arterial embolism in the left internal capsule. I was present at the autopsy.

Gout—Right Hemichorea—Death.

J. A., æt. fifty-four, painter all his life, was admitted to Luke Ward, November 7, 1881. Twice married, five children.

No symptoms of lead-poisoning; no blue line; no colic or paralysis. Gout seven years ago. Drank beer freely always; not much spirit. No syphilis. Bronchitis, occasionally, five years, last time two years ago. Went to work on November 2 apparently quite well. Right hand was noticed to tremble a good deal, and this soon extended to right leg (choreic movements ceasing during sleep), since when movements have been almost constant. Four months ago right hand was not very steady. No other affection of muscles of face or trunk. Sensibility perfect. Right hand dusky and congested. Special senses natural. Pulse 80, regular. Patellar tendon-reflex natural. P.S. in chest, wheezy respiration, prolonged expiration. Heart-sounds natural, feeble; dulness abolished. Liver depressed. Pulmonary emphysema.

November 10.—Became very restless in afternoon yesterday, and delirious in evening; occasionally very violent paroxysms. Leg more restless. At 10 P.M. removed to Casualty Ward, shouting. Got 2¼ gr. morphia subcutaneously. Takes food well.

November 11.—Restless, dyspnœa after paroxysms. Choral ℈ss. 2is horis. On ophthalmoscopic examination, discs natural.

November 16.—Quieter last few days. Right arm been getting sore, brawny, fluctuation; abscess opened.

November 19.—Excited by visitors yesterday. Passed urine under him in bed. Chloral and bromides given.

November 21.—Temperature rose to 104.6°. No rigors. Movements continue. Tongue dry and brown. No fresh P.S. in lungs. Urine no albumen. Very irascible; delirious. Temperature 101.2° on admission (on 10th). Nightly rises to 100°, 100.6°, 101.2°, and on 20th 104.6°. 21st same. 100.6° night of 20th. Last night 103.8°.

Died on 23rd (morning 1 A.M.).

Post-mortem examination (November 24).—Lungs emphysematous. Heart rather large, flabby; a calcareo-atheromatous ring over middle aortic cusp, sharp, and likely to cause onward murmur, and shedding of fibrinous fragments. Kidneys weighed together, just under eleven ounces. Commencing cirrhosis. No uratic streaks. Liver and spleen natural. Brain, a speck of hæmorrhage was found in the internal capsule on the left side, lying near the optic thalamus, but separated completely from it.

Gout in Relation to Asthma.—The relation of the gouty habit of body to asthma is marked and important. Certain cases of asthma appear to be plainly connected with gouty inheritance and constitution. Family and personal history often illustrate this connection. Paroxysmal tendency pertains to the gouty habit, doubtless in dependence on the inherent neurosal features of it. The subsidence of regular gouty attacks is followed sometimes by an asthmatic paroxysm, and the latter yields to onset of frank gout in some part.

The skin-affections common to the gouty may alternate with

attacks of asthma. The paroxysms may come on in the early morning hours, exactly as in the case of attacks of gouty arthritis. Bronchitis is common in the gouty, but may be void of asthmatic complication. In some cases both are well-marked. Amongst the varied metamorphoses of neurotic states, asthma takes its place in the list, and is found interchanging with epilepsy, neuralgia, chorea, migraine, and insanity.[1] It may be directly hereditary, or may appear as a transformation of another inherited neurosis. A gouty habit may be a basis for all these conditions.

Regarding asthma as a paroxysmal dyspnœa due to altered innervation of the bronchial tubes, its ætiology in each case has to be specially sought. Any pulmonary lesion by itself is insufficient to explain its occurrence. The relation of gout to this condition appears to depend not merely on the altered blood-state (humoral cause), but equally on the neurosal condition associated with this. The asthma of the gouty is, therefore, neuro-humoral, and due either to central or local irritation. Cases owning this dependence may arise at various ages. In the young, where no obvious gouty symptoms appear, the neurosal element is alone manifested for the most part. In persons in the fourth decade, overt gouty symptoms may appear, but arthritic inheritance may be equally strong in instances of either. Both sexes may suffer, but in women we are naturally less likely to find clinical evidence of the gouty taint than in men. It would appear, indeed, to be rather common to find the neurosal evolution of gouty inheritance transmitted to the female side with greater energy.

In elderly persons, thus affected, attention must always be paid to the efficiency of the kidneys, granular condition being so commonly associated with gout.

Uræmic asthma may be mistaken for the simpler and less grave form of alternating gouty asthma, but not seldom in the latter category may the urine afford evidence of progressing damage in the kidneys, some degree of albuminuria being present, and its amount possibly increased under the congestive influences of bronchitis, emphysema of the lungs, and distension of the right side of the heart.

A clinical distinction may sometimes be made between uræmic and bronchitic or other forms of asthma. In the former there may be no superadded respiratory sounds, the air-entry being clear, and even exaggerated. This fact has led to the belief that the obstruction in uræmic asthma is due rather to spasm of the pulmonary

[1] "Should gout seize upon the lungs, there succeeds a violent asthma that threatens suffocation, which is preceded by a dry, uneasy cough."—*van Swieten, op. cit.*

arterioles than to that of the finer bronchi, the cause being possibly some urinary poison acting on the blood-vessels.[1]

Hay-Asthma—Summer Catarrh.—Noël Gueneau de Mussy was of opinion that many cases of hay-fever were especially frequent in members of gouty families, and he regarded the changes in the mucous membrane of the nasal passages as akin to the eruptions which vex the skin of gouty subjects.[2] He recorded ten cases in illustration of this. I have sometimes noted the connection.

Gout in Relation to Angina Pectoris and Cardio-Vascular Neuroses.

A connection between gout and angina pectoris has often been affirmed by systematic writers, but not many cases in support of it are on record. Two prominent features of the neuroses pertain to attacks of angina pectoris, viz., paroxysmal tendency and extreme painfulness.

Peter Mere Latham was evidently sceptical of the direct connection between gout and *true* angina, and had no experience of alternation of one with the other. He remarked that he could conceive this to have happened in cases "where the angina has been an affection truly vital, and the heart has suffered pain and spasm, though perfectly sound of structure. That such an angina should germinate from the same root as gout is not unlikely."[3]

True angina pectoris with a fatal issue is, fortunately, a rare disease. It affects chiefly the male sex and persons in the upper ranks of life about the eighth climacteric period.

It may be affirmed that the grave forms of angina pectoris are hardly recognized apart from organic disease of the heart. Anginal attacks, sometimes termed pseudo-angina, may occur without overt cardiac disease, and be met with in young persons. Such angina as is clinically referable to gouty influence may be put into two categories: first and chiefly, pseudo-angina, a form occurring where the heart is presumably sound, in immediate connection with a recent or imminent articular attack; and, secondly, the severe form which is associated with arteritis, degenerative change in the cardiac walls and sclerosing valvular lesions, of which the commonest type is aortic, and especially that permitting of reflux.

Imprudent exposure during recovery from an attack of gout in

[1] This point was originated and well discussed by Dr. William Carter, of Liverpool, in the Bradshawe Lecture (Roy. Coll. of Physicians), 1888. Lancet, August 25, p. 359, 1888.

[2] *Gaz. Hebd.*, ix. 9, 1872.

[3] On the Diseases of the Heart, vol. ii., 1846, p. 419.

the feet has been known to excite pseudo-angina, as in a case related by Garrod, where several seizures occurred, the foot again becoming gouty.[1] There were no signs of cardiac disease in the patient. It appears probable that this was an instance of metastasis, akin to other forms of visceral gout, which may occur under similar circumstances.

Professor Gairdner, reviewing the evidences of connection between gout and angina, believes that it may be inferred that the, so-called, metastasis of gout to the heart is the result of gradual degenerative changes operating more or less throughout the organism, which, if not so distinctly related, as has sometimes been supposed, to the gouty paroxysm in its ordinary form, are at all events closely associated with the causes of gout, and, therefore, form part of its history as a disease of the constitution.[2]

Lecorché remarks, "Pour nous la maladie d'Heberden, chez les goutteux, est toujours due à une arterite goutteuse des coronaires."

It has already been shown in the chapter on morbid anatomy that the heart and arterial system suffer severely in the course of chronic gout. The morbid changes are precisely those which, when induced by other cachectic states, lead sometimes to associated angina; hence, it is impossible to resist the conclusion that the gross degenerations, however set up, are directly connected with the phenomena of the anginal attacks.

Direct evidence is not wanting to prove in some instances that the sclerosing changes in the aorta and coronary arteries directly involve and compress branches of the cardiac nervous plexus. The aorta and coronary arteries may be sufficiently diseased to induce degeneration of the cardiac walls without any very marked physical signs. In such cases, angina may supervene and the heart be deemed fairly sound, unless proof to the contrary is furnished by an autopsy. In this way may possibly be explained some of the cases of fatal angina in which the heart has been believed to be healthy.

It is, perhaps, more difficult to explain why angina should not be always present where advanced arterial atheroma has led to softening and dilatation of the cardiac walls, since it is certain that many cases of this kind occur and end fatally without a symptom of angina. I would suggest that, in such instances, there is an absence of the necessary neurosal element to determine the paroxysms.

[1] *Op. cit.*, p. 440.

[2] Art. "Angina Pectoris," Reynolds' Syst. of Med., vol. iv. p. 547.

The relationship of the gouty habit to angina may be thus expressed:—

(1.) Pseudo-angina-pectoris may occur as an attendant on chronic gout. This is certainly the more common form in which attacks of cardiac pain occur in the gouty. Its characters are, constrictive pain with paroxysms of palpitation, faintness, giddiness, and panting respiration. There is commonly gastric disturbance, indigestion, and flatulency. The patient may be under fifty years of age, at a time of life when true (grave) angina is uncommon, and is usually a male subject.

(2.) True angina pectoris may supervene in cases of chronic gout or of gouty cachexia, in which wide-spread arterial degeneration, aortic atheroma, and softening of the cardiac walls have occurred along with other signs of textural decay. Here, the gouty state is the factor which prepares the way for the onset of angina; but similar degenerations may be induced by other than gouty influences.

In this form the patient is usually over fifty years of age. The pain is tearing and violently constrictive, radiating to the back and often down the arms, but especially the left one, as far as the inner sides of the elbow. There is no sense of dyspnœa. The pulse is small, tense, irregular, and may be infrequent. The patient experiences a sense of imminent dissolution. There may be gastric flatulency. After the paroxysm a large flow of urine may occur.

It may be noted that the subjects of angina pectoris are not infrequently men of great ability and mental activity, just the class so often affected with gout.

Cases of angina pectoris have been described as diaphragmatic gout.

The heart is not found to present indications of organic disease in instances of pure cardialgia or pseudo-angina, and no marked change may be detectible in the arteries. The patients are, as a rule, too young to be thus affected, and are far from the stage of gouty cachexia. The attacks may be severe and well-pronounced, and may occur at long intervals. They are frequent in the night. Sometimes, patients present these symptoms without having experienced any regular fits of gout; but a marked family history of the disease is usually to be elicited in such a case. We may, therefore, agree with Trousseau, who regarded such paroxysms as "manifestations of the gouty diathesis."

The relationship of gout to anginal tendency cannot be dis-

missed without reference to the special neurosal quality attaching to each. In each there may be gross changes, or conditions favourable for attack; but the special determinant and predominating features come from the nervous side, whence the explosive paroxysm and the painfulness. It is impossible not to take note of an underlying specific state of the nerve-centres in the two cases, instability and proneness to discharge along certain nerve-tracts probably representing the perverse functional condition.

We may, thus, discover a radical (neurotic) relationship between the gouty habit and the occasional tendency to attacks, not only of angina pectoris, but of other painful nerve-states.

The influence of emotion as a determinant, and the tendency to paroxysms in early hours of the morning after the first sleep,[1] pertain to other spasmodic neuroses.

Graves' Disease, or exophthalmic goitre, has been noted in persons descended from gouty parents. I have, so far as I know, only met with one instance of this kind, in the case of a lady who was "nervous" and hysterical. Towards the menopause symptoms of Graves' disease came on. These passed off within a year, and much benefit was derived from subalpine residence in Switzerland. The father and several brothers had distinct gouty indications.

Tachycardia.—Cardiac palpitation is recognized as occurring in arthritically disposed persons. Cases of chronic rheumatic arthritis in its rapidly progressing form are sometimes characterized by tachycardia almost from the onset, the pulse being tense and rising to ninety or higher. I have met with one or two examples in women where the pulse remained persistently from 150 to 200 per minute; but I have never seen anything like this in gout.[2] No signs of gross cardiac disease are detectible in the rheumatic cases, and there is no associated pyrexia. This functional tachycardia may endure through quiet progress of the arthritis.[3]

According to Gerhardt, there are two forms of tachycardia, (*a*) lasting and (*b*) transitory. Most cases of the nervous form he

[1] "Nonnullos adoritur post primum somni tempus; quod in morbis ex distentione frequens est."—*Heberden.*

[2] Dr. Baillie related to Dr. Scudamore a case in which palpitation of the heart was experienced for six months without relief from medicine. A fit of gout suddenly and entirely removed it.

[3] Dr. Spender, of Bath, has described some cases of this nature. Brit. Med. Journal, April 14, 1888, p. 781; and Early Symptoms and Early Treatment of Osteo-Arthritis, London, 1889, p. 6.

attributes to paralysis of the vagus; those which have a pulse-rate of 200, to a combined stimulation of the vagus and sym-

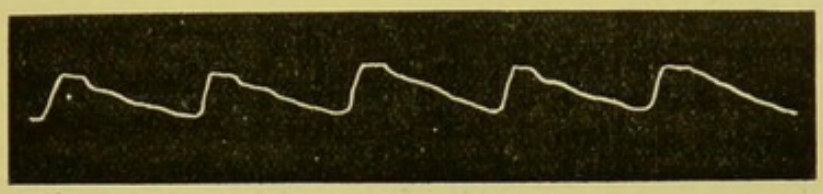


Fig. 17.—Sphygmogram from case of chronic gout with tophi and psoriasis. Urine, 1002; containing trace of albumen. Illustrating increased tension. (Probably interstitial nephritis.)



Fig. 18.—Sphygmogram from case of J. A., æt. 42. Heart feeble. No murmurs.



Fig. 19.—Under the finger this pulse did not indicate increased tension. A. J., æt. 50.

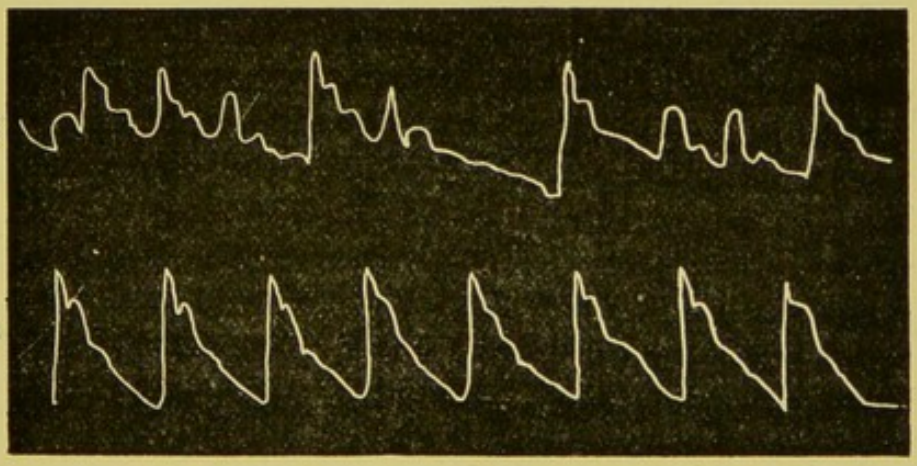


Fig. 20.—(a.) Illustrating irregular pulse in gout. The largest sweeps of the lever occurred during the respiratory pause, the smallest during and after inspiration. (b.) Pulse regular during an attack of gout (?aortic reflux). (From Prof. Burdon Sanderson's book on Sphygmograph, p. 77.)

pathetic. The higher forms he considers entirely due to the latter.[1]

[1] Volkmann's Collection of Clinical Lectures. New Sydenham Society Trans., 1881.

I have noted one instance in a case of severe gouty glycosuria in a man aged fifty-two, during recovery from an acute attack of arthritis, the temperature being but little raised, and the pulse over 130.

Undue pulsation of the abdominal aorta and of other large arteries is another occasional symptom in gouty persons, met with in both sexes, and sometimes associated with dyspepsia, tympanites, oxaluria, and hypochondriasis.[1] I have known many examples of this disorder mistaken for aneurysm, but have noted no changes of any importance in the vessels in such cases as have, from other causes, afforded an autopsy. An attack of articular gout may entirely remove the symptoms.

Irregularity and "true" intermission of pulsation have long been known as symptoms of the gouty habit. Although sometimes causing anxiety to the patient, they are void of serious import, and are not the expression of organic heart-disease. There is absence of cardiac systole as well as of dropped radial beat.

In patients of advanced age, with signs of arterial decay, there may be found with the arhythmia signs of valvular disease and fatty change in the heart-walls. In such instances there may be cardiac systole with dropped beat at the radials—"false" intermission.

Gout in Relation to Headache and Hemicrania.

It is probable that few bodily discomforts originate in so wide a field of causation as do the many varieties of headache. Much discrimination is necessary in referring any form of it to its exact source. Persons of gouty habit may certainly suffer from headaches which are independent of that habit.

The descriptions by the older writers of the cerebral symptoms of gout comprise cases of headache which would now be recognized as due to uræmia, rather than to direct influence of uric acid. These are not seldom dependent on chronic nephritis, the result of gout. The history of previous gouty attacks appeared formerly to justify the opinion that in such instances the disorder had fallen directly, or by metastasis, on the brain or its membranes.

An examination of the urine, heart, arteries, pulse, and retina

[1] First described by Dr. Matthew Baillie at the Royal College of Physicians in 1812. Med. Trans., published by the College in 1813, vol. iv.

should now prevent a diagnostic error of this kind. The subject of unequivocal gouty, or, as it is sometimes termed, uric acid headache, is free from any noteworthy signs of degeneration in any of the structures just mentioned. It may occur within the second decade of life. There are no special elective sites for it, and it may either be general, or localized in any part of the head. The pain varies in severity from that of the uncomfortable, or "muzzy," head to a degree of great intensity. In severe instances the surface of the scalp is apt to be hot and tender, so that even pulling of a single hair is painful. The pain may remit during an attack and become worse after an interval, thus presenting a paroxysmal character, which has sometimes led to the belief that the disorder was of a malarious origin. The headache, after lasting for some days, may suddenly yield to an attack of frank articular gout, or may replace such an attack.

Dr. Lauder Brunton declares his inability to distinguish a gouty headache from that of plethora or indigestion, and would suspect the gouty element only from the patient's family and personal history.[1]

Dr. Haig has directed attention to this form of headache, and has shown that it can be artificially induced at any time, in those subject to it, by indiscretions of diet, or, directly, by such agents as cause increased excretion of uric acid. The increased out-put of uric acid and the headache associated with this are believed by Dr. Haig to indicate a state of urichæmia, since, if there be no surplusage of uric acid in the blood or system, the exhibition of alkalies (the means whereby he induces headache in these cases) fails to produce either the increased excretion of uric acid or the headache.

It is possible in practice to distinguish between the gouty cephalalgia of most authors and varieties of hemicrania or migraine. The latter may occur in classical form with teichopsia, or "dazzles," (as one of my patients termed it), be strictly one-sided, and terminate with nausea or vomiting. Whether a true migraine or a severe and more general headache shall occur, depends probably on personal proclivities and peculiarities, and, not least, on the degree of gouty heredity. I believe there is one basic source for all forms in those who are gouty for the time being, or predisposed by inheritance or otherwise to gout.

Hemicrania owns other causation than a gouty basis, but is

[1] St. Barth. Hosp. Reports, vol. xix. p. 340, 1883.

perhaps more common in those of gouty inheritance.[1] It may occur in the place of regular gout, but more often appears in those who never develop the latter. In this way it constitutes one of the transformations of the gouty neurosis, or may be regarded as a form of incomplete gout. The violent headaches (gouty cephalalgia) which least correspond to hemicrania, and which are apt to persist for some days, prevail in those who are already the subjects of frank gout, or are likely soon to manifest it. These headaches are not so readily induced as hemicrania, and they do not begin early in life and recur periodically after the manner of pure megrim. The latter is much the more frequent form met with, but many persons suffer from non-classical varieties of it. The most regular migraine occurs as a paroxysmal neurosis, and many of gouty heritage thus suffer. Imperfectly developed attacks may also affect those who are goutily disposed, but in response to stronger provocation than is necessary to upset the nervous balance in the victim of the graver form. In the latter case it sometimes happens that no amount of care and prudence can avert occasional paroxysms. In the milder forms such measures are potent to avert an attack for long periods, as well as to mitigate the intensity of it.

As in other neurotic disorders, there is always a tendency for the disease to grow up or develop to a certain point before an outburst occurs. In the case of gouty ailments this development is commonly attributed to uric acid retention, and this view may be taken as generally correct. I have already tried to show that this humoral conception is not all-explanatory, and that the nervous factors in each case must be equally taken note of.

The provoking causes of hemicrania certainly often start from the nervous side; thus, exhaustion, mental or bodily, over-excitement, exposure to strong light, vivid colours, powerful odours, bad air, noise, anxiety, fright, &c., are no less determinants of an attack than are single or repeated indiscretions in diet.[2]

I agree with those who regard hemicrania as a paroxysmal nerve-storm induced by one or more of the causes I have just enumerated, and I am fully in accord with the views set forth by Dr. Edward Liveing in his masterly treatise on the whole subject of megrim. The characters of a well-marked attack are well-known.

[1] "Souvent c'est la seule expression de la prédisposition héréditaire chez des sujets nés de parents franchement goutteux."—*Trousseau.*

[2] The late Professor Rolleston suggested that after great mental emotion, worry, or brain-work, the worn-down nervous matter came to act as a poison in the system, especially affecting the sympathetic centres, leading to paralysis of them and a nerve-storm.

The onset is common in the early morning, the patient waking with a sense of discomfort in the head. It is often the case that there has been previously a sense of *bien-être*, the head having been clear and all bodily functions well-performed. The appetite may have been especially good. The attack continues during the day, probably increasing in intensity, with sensation of chilliness, cold feet, and general malaise. The pain is of throbbing character, shooting into the eye-balls, the conjunctivæ are injected, and may be a little icteric; pupils rather small. Exertion aggravates the pain, especially the effort of ascending stairs, which excites throbbing in the head. There may be slight nausea, or the appetite may be hardly affected. Teichopsia may, or may not, be present. The instinct is for absolute rest and tranquillity of mind in a darkened room, with warmth. Recumbency often aggravates the suffering. Pressure on the carotids, while maintained, relieves the pain. The pulse is infrequent, small, and tense, and subjects of these headaches commonly have abiding high arterial pressure. This is increased during a paroxysm. In my experience, the distribution of the headache is rather general than local, with sorer points in some parts, such as are animated by the great occipital or supra-orbital nerves. There is frequent desire to micturate, the urine being pale and watery. The pain may rage till the evening, sometimes increasing up to that time, when it usually subsides, or the attack may last over the second day.

Vomiting is occasionally coincident with the termination of the paroxysm. The motions have been found rather paler than natural at the time of the attack.

There is a tendency for the paroxysms to become less severe and less frequent after middle life, but they may occur up to the sixth decade. In well-established cases they may come on every third week, and it is seldom that an entire month passes without their occurrence.

There are more or less attendant prostration and depression, and to pursue the ordinary duties of life requires a strong will and a high moral purpose. Some sufferers are fairly overcome for the time being, and unfit to face their avocations.

The description just given relates to the worst form of megrim. There are many degrees of intensity in the attacks, even in the same patient, but in its most attenuated form the type of the disorder is always recognizable.

The measures which best afford relief to the paroxysm and prevent its recurrence throw light on the nature of the disorder, and especially when it is manifestly dependent on gouty habit.

Gout in Relation to Neuralgia.

The dependence of certain forms of neuralgia on the gouty habit is well-ascertained. Paroxysmal and periodic as in other forms, there is nothing peculiar about the painful nerve-states due to a gouty condition of the system. The diagnosis is founded on the ordinary concomitants of the basic disorder. Wherever painfulness is a feature in a gouty process, there is commonly a severe degree of it, and it is ill-borne by the sufferer. Almost any nerve-trunk may be affected, and it is characteristic, perhaps, of gouty neuralgia that it may appear in sites not usually involved by other forms of this trouble.[1]

Exposure to currents of cold damp air, especially to north-east wind, after being over-heated, exhaustion from any cause, bodily or mental, loss of sleep, undue or prolonged excitement, are the most frequent determining factors.

Gouty neuralgia may prove very rebellious to treatment, even to that intelligently directed towards the cause of it. This is especially the case in persons past middle life, whose textures indicate signs of decay.

The attacks may alternate with overt or articular gout, on the appearance of which the neuralgia passes off. They are apt to come on very suddenly, and to follow quickly on errors of diet or trivial excitants.

The most frequently affected nerves are the supra-orbital and occipital, especially the great occipital, and various branches of the brachial plexus. Some of the most severe forms of cervico-brachial neuralgia are connected with gouty taint. Intercostal neuralgia is sometimes thus dependent. In the lower extremities the great ischiatic nerve is most commonly involved, giving rise to a form of sciatica, and sometimes the anterior crural nerve is the seat of pain.

In some cases of, so-called, gouty neuralgia it is almost certain that a more accurate diagnosis would be that of neuritis. I suspect that this is especially the case in many examples of severe and rebellious sciatica. In such instances there are inflammatory changes in the nerve-sheath, leading to sclerosis.

There is sometimes difficulty in exactly determining whether the "pains" complained of are actually in nerve-area, or are due to uratic retention and localized gouty processes in lymph-spaces or in the aponeuroses of muscles. Such pains may be very

[1] "Gout affects the sensory much more than the motor elements of the nervous system."—*Paget, Clin. Lect.*, 2nd edit., p. 382.

severe and also fugitive. The regular paroxysmal tendency is wanting, as well as the peculiar benumbing character pertaining to true neuralgia after it has lasted for some time. The fulgurant pains of tabes dorsalis are hardly likely to be mistaken for ordinary neuralgia by any careful observer.

The causes that commonly excite neuralgia suffice to induce it in the gouty. The determining excitants may be seemingly trifling, and such as would fail to induce the disorder if the patient were not, so to say, in an explosive condition, that is, ripe for some gouty manifestation.

Sometimes, neuralgia is the exponent of gouty heritage in persons who have as yet developed no overt signs of ordinary gout, thus exhibiting one of the forms of transformation already alluded to. This is perhaps more frequently seen in women, and there may be found a low state of general health with such signs as betoken an incomplete, or asthenic, gouty state of body.

In more plethoric persons there will usually be found associated with the neuralgia the disturbed digestion, "muzzy" head, constipation, and hepatic fulness, with loaded urine, which so often precede ordinary gouty attacks.

It is not without interest to note incidentally that neuralgia is sometimes associated with chronic forms of saccharine diabetes. I have already recorded some cases of gouty glycosuria in which severe rachalgia, apparently neuralgic, was experienced, but I have no knowledge of instances of ordinary neuralgia in this class of patients. Bilateral neuralgia has been noted in diabetes.

Herpes Zoster, and other varieties of herpes, occur in the gouty, and the subsequent neuralgia, especially after zona, may in such persons be very intense, and long incoercible. I have known zona occur in this form together with equally rebellious gout in a great-toe and foot—a very formidable combination—lasting for many weeks.[1]

The most persistent and agonizing varieties of neuralgia which I have witnessed in the gouty have been those involving the great occipital nerve[2] and the cervico-brachial plexus, affecting chiefly males in middle life.

As noted by Paget, a gouty neuralgia may often be recognized by its being more fitful and sudden than others, and more quickly induced by errors of diet, indigestion, and other casual circum-

[1] Mr. W. E. Musson, of Clitheroe, informs me that his attention has been directed to the frequent occurrence of herpes, particularly in its facial forms, in gouty subjects. Dr. Symes Thompson has also noted the same.

[2] More often the right nerve is involved.

stances; and neuralgia in certain parts is especially significant of gout, as in the heel, external ear, tongue, palate, fingers, and breast. The same observer also noted many varieties of pain and dysæsthesiæ which torment the gouty, such as numbness, sensations of "pins and needles" in the toes and fingers, and "dead" fingers, which become cold and white, and subsequently flush and grow hot.

The following case of genito-crural herpes was recorded in the British Medical Journal of March 13, 1880:—

A patient, about sixty years of age, who had occasionally suffered from gout, experienced left lumbar pain extending to the iliac region, and causing retraction of the left testis. This came on after having got wet fishing, and while recovering from an attack of gout. The pain was constant, almost unbearable during exacerbations, and aggravated by movement. There was frequent micturition of clear and acid urine. Bowels regular. Pulse and temperature unaffected. The pain continued for two days. On the third day a patch of herpes appeared on the upper part of the left thigh, and on the fourth day the pain passed off. The patient's father suffered from vesical calculus, and had it crushed at the age of seventy-three.

Sudden twitches of intense pain, lasting only a few seconds or minutes, or for some hours, are apt to seize goutily disposed persons, and without obvious cause. The suffering is sometimes very severe, and would be intolerable if long-continued. The legs, feet, and toes are the commonest sites of such fugitive pain. Sometimes, the ear is affected with sudden pain, which lasts only a few hours. Graves was a sufferer from this on one occasion for an hour, and the ailment ceased on the occurrence of gouty pain in his fingers. He believed that sudden congestions of the parts so affected took place. It is probable that temporary local stasis of acid sodium urate is the cause.

Dr. Anstie noted six situations in which gouty pains, or pains of latent gout, are apt to occur simulating neuralgia.[1] He did not believe in an intimate causal relation between gout and neuralgia. He mentions (1.) pains in the eye; (2.) more indefinitely *within* the cranium; (3.) in the stomach, simulating gastralgia; (4.) in the chest, simulating angina pectoris; (5.) in the dorsum of the foot, simulating neuralgia of the anterior tibial nerve; (6.) in a somewhat diffuse manner about the hip and back of the thigh, simulating sciatica. If the pains referred to are not neuralgic, they can only be due to localized attacks of incomplete gout, which should not be very difficult of recognition.

Gout in Relation to Vertigo.

Amongst the symptoms depending on irregular gout is that of giddiness or vertigo. In making the diagnosis, care must be

[1] Neuralgia and its Counterfeits, 1871, p. 270.

taken to exclude any obvious causes not connected with the gouty habit of body. Thus, ocular, aural (labyrinthine), and epileptic vertigo must not be confounded with this form.

Sudden attacks of gout may entirely remove tendency to vertigo of long duration. Assumption of the erect posture may induce vertigo. This occurred for two years in a man whose case is quoted by Trousseau from Boerhaave's commentator (van Swieten). A first attack of gout entirely removed the tendency. John Hunter suffered at one period of his life from this affection, which lasted ten days, during which time he was compelled to keep prone.

Vertigo may be induced in the goutily disposed by irritating ingesta, and some of the cases of so-called gastric vertigo are, no doubt, of this class. Murchison relates the case of a man who had long suffered from gout, and who, as often as he partook of a cup of tea or a glass of champagne, would suffer from vertigo, and be compelled to hold on for support. There was no loss of consciousness, and this attack lasted for a few seconds or minutes. The same writer relates other cases in which vertigo and dimness of vision occurred in association with lithæmia, but not with gout.[1] Dr. Moxon met with a case in which a gouty man suffered so severely from vertigo, that, when seized, he had to go about "on all fours."

Mere dimness of vision may constitute a minor attack of this kind of vertigo. This occurs in the gouty, coming on suddenly while reading or writing, or when out of doors. Trousseau records an instance in which the patient felt as if his eyes were covered with flakes of snow. The attacks are of brief duration, and may recur several times for a day or two. In all these cases there are generally associated gouty concomitants.[2]

The following case occurred amongst my out-patients in 1882:—

J. R. S., æt. thirty-six, a marbler of book-edges, came complaining of tinglings and "hot surgings of blood" in the fingers, hands, and head. This was apt to come on a quarter of an hour after meals. There was much flatulence, and, occasionally, vertigo. The left ventricle of the heart was a little hypertrophied, and the arteries

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[1] Diseases of the Liver, p. 588, 2nd edit.

[2] "I saw a man who for two years was afflicted in this terrible manner; to wit, as oft as he remained sitting and at rest, he perceived nothing; but the moment he got up to stand with his body erect, he was immediately seized with a giddiness, and fell down. Many things he tried by the advice of the ablest physicians, but all without success, till at last a sudden fit of the gout, which he never had experienced before, cured him entirely of this troublesome vertigo."—*Van Swieten's Comment. on Boerhaave's Aphorisms*, Sect. MCCLXII.

somewhat hard. The urine was of sp. gr. 1017, void of albumen, rich in indican. His family was, he alleged, nervous and irritable. The father, aged sixty-nine, suffered from "chalky" gout, one brother, æt. twenty-five, had had gout in a great toe-joint, and a sister, æt. nine years, had been in King's College Hospital for gout in a great toe-joint.

There could be little doubt about the chain of events in this case.

Gout in Relation to Hypochondriasis and Hysteria.

The dependence of hypochondriasis on a gouty habit of body is most plainly recognized in cases where this state yields to, or is mitigated by, an attack of uratic arthritis.

Sydenham noted the peculiar mental conditions sometimes associated with gout. "The mind suffers with the body, and which suffers most it is hard to say. So much do the mind and reason lose energy, as energy is lost by the body, so susceptible and vacillating is the temper, such a trouble is the patient to others as well as to himself, that a fit of gout is a fit of bad temper. To fear, to anxiety, and to other passions the gouty patient is the continual victim, while, as the disease departs, the mind regains tranquillity."

Hypochondriasis is not commonly met with in the fair-skinned, "sanguine arthritic," type of body, where the circulation is vigorous and the mind usually active. It is more apt to occur in lean, sallow, or dark-skinned persons, where the circulation is feeble and nervous energy is less active. In such patients there is often some degree of hepatic inadequacy, the digestion is feeble, and small dietetic errors are quickly and severely felt. Oxaluria is not infrequent. There is gouty heritage, but often incomplete manifestation of the disorder in such sufferers.

It is well-recognized that much depression of spirits, without due cause, is apt to overwhelm some gouty subjects. They are "under a cloud," moody, and lugubrious from time to time. Their outlook is gloomy, and only the darkest side of events is visible for the time being. Such symptoms "grow up," as it were, occasionally, and a purge is the best treatment for this "moping melancholy."[1]

I have learned to regard this condition rather as a phase of incomplete gout than as one alternating with regular arthritic attacks, and as exhibiting a manifestation of the gouty habit derived from inheritance.

Lecorché observes that hypochondriasis in the gouty is not

[1] Milton.

wholly imaginary, but "reposes on a basis of sufferings which are only too real." The varied minor ailments common in incomplete gout become magnified and exaggerated in importance. Regular fits of gout but rarely, and imperfectly, relieve this condition. As pointed out by W. Gairdner, "the same habit which prevents the attack makes a lingering paroxysm." The latter author recorded several instances of hypochondriasis in the gouty, and noted that the sufferers were gluttonous eaters, and that their gout was of an atonic character. He noted every degree of this affection, from the gentlest solicitude about health to the deepest despondency. In common with other careful observers, he referred to the frequency of hypochondriasis and hysteria in women, especially at the menopause.

I have already mentioned the case of a lady of arthritic inheritance who developed, temporarily, symptoms of Graves' disease, and was markedly hysterical throughout her married life, and after the cessation of the catamenia. She was commonly in a desponding and gloomy frame of mind.

Hysteria or neuromimesis, in its many varieties, is, without doubt, a very frequent disorder in the female descendants of the gouty.[1] I have of late regularly inquired as to this point in cases that have come before me, and I can affirm confidently as to the connection between the two conditions in many instances. The symptoms may appear soon after puberty, or at any time up to and after the menopause. The daughters of gouty fathers present the majority of instances. Garrod has noted cases where spinal tenderness and articular symptoms have clearly alternated with each other. Laycock insisted on the fact that the arthritic diathesis predisposed women to anomalous forms of hysteria, and his opinion was the result of long-continued study of the whole subject.

The leading features in these cases are a feeble state of the circulation, and a delicacy of nervous system. It is not essential that luxurious habits should have been indulged in, since cases occur in the families of the poor. Errors in diet and faulty methods of education, both of mind and body, have, doubtless, much to do with the induction of hysteria; but there is probably inherent defect in the generation and flow of nervous energy in all cases.

[1] "It has been supposed by some writers that the daughters of *gouty* parents are more prone than others to hysteria. This may be partly accounted for by deficiency of constitutional energy derived from the parent, and greater susceptibility of the nervous system."—*Dict. of Pract. Med.*, Copland, Art. "Hysteria," p. 283.

The occurrence of hysteria amongst the subjects of arthritic inheritance appears to furnish additional evidence of the implication of the nervous system in the varying manifestations of gouty disease.

In cases of painful joints occurring in hysterical women (arthropathia hysterica) it is not improbable that the localized determination may here be specially significant of arthritic inheritance.

The majority of cases present an asthenic type even when there is present a misleading appearance of robustness.

The Incidence of Gout in Paralysed Limbs.

It has been stated that acute gout does not occur in paralysed limbs;[1] but my own experience, with that of other observers, is distinctly contradictory of the assertion.

M. Landré-Beauvais in his "Thèse" (1800) relates a very noteworthy instance in support of the fact that gout may attack a paralysed limb. It was that of a woman who had three paralytic seizures (right hemiplegia), the first at twenty-five years of age, the second at fifty. After the latter, the right limbs became the seat of vague pains, recurring at intervals. At the age of seventy-four there was a third attack of hemiplegia on the same side, and some months afterwards the pains, which had affected several joints of the paralysed side, settled in the malleoli, with redness, and swelling. Acting on empirical advice, the patient placed her feet in a very hot bath. The pain and swelling disappeared, but in two hours she was seized with violent pains in the stomach and a sense of constriction at the epigastrium. No relief was obtained from treatment, and death followed on the eighth day. At the autopsy, the stomach and intestines were found inflamed, thickened, and sphacelated in places.

In the case of a man under my care in hospital, who was admitted with left hemiplegia of recent onset, general arterial sclerosis, and granular kidneys, acute gout came on in *both* wrists, and followed an ordinary course.

Lecorché records the case of a man, aged thirty-nine, of gouty heritage, who had right hemiplegia after a fall from his horse at the age of eighteen. He had had rheumatic fever when he was sixteen, and syphilis four years later. Gout attacked the great toe of the paralysed leg, and a month afterwards the left great toe; subsequently, he had four attacks in the right great toe.

[1] The late Dr. Leared affirmed this.

Metastasis occurred to the intestines in one attack, and the acute pains there ceased on return of the process to the foot.

The following cases illustrate the incidence of attacks on the sound side. A man, aged seventy, with left hemiplegia, who was under my care, had acute gout in the right great toe-joint. A man, aged thirty-five, admitted into hospital for left hemiplegia, had several attacks of gout in the right great toe-joint, right hand, and phalangeal joints. On one occasion he had some pain in the *left* knee. Both parents were gouty. There was no albuminuria. The man suffered from lead-impregnation, owing to his work as a leather-cutter being done on lead, and he probably had granular kidneys and cerebral hæmorrhage.

In the following case of left hemiplegia, attacks of gout occurred on both sides of the body.

Gout in Paralysed Limbs.

George Ingall, æt. fifty, by trade an optician, was admitted into Mark Ward on February 22, 1881, suffering from well-marked left hemiplegia.

According to his statement, he was fairly well up to the 17th instant, though for a day or two previously he had suffered from a dull headache. Working as usual on the day mentioned, he was seized with trembling and nervousness, and about half an hour afterwards fell down semi-unconscious, and was unable to raise himself, having lost the power of his left arm and leg. His memory seems to have been affected, and he appears to have confused ideas of what happened between this and the time he was admitted into the hospital.

Previous history.—Five years ago he had sunstroke on a voyage from Africa, and was ill for three weeks. Two years ago had a fit (epileptic ?). A month or two ago he noticed a puffiness about his eyelids ; but his feet have never swelled. Of late he has been subject to drowsiness. Has had two attacks of gout.

Family history.—Father died of some liver complaint at the age of forty-six. Mother died at fifty-two. Brothers and sisters healthy. Does not know of any paralysis or gout in family.

On admission he was a man of medium stature, with a fairly-nourished body. Hair dark, eyes brown, complexion sallow, skin soft and dry.

The paralysis, which affected the left side of the body, was more marked in the upper extremity than in the lower. The arm was swollen and painful, and any attempt to move it caused pain. Though motion was much impaired in the leg, he could move it voluntarily, but was quite unable to stand. Sensation, though impaired over the paralysed side, was still preserved, though discriminating power, for the most part, was lost.

Patella-reflex present, and also skin-reflex. Ankle-clonus absent.

The left side of the face was also paralysed, with the exception of the occipito-frontalis and orbicularis. Articulation was affected, but deglutition was performed without difficulty.

Chest.—Inspection showed that the left side expanded to a less degree than the right. Palpation, percussion, and auscultation normal.

Heart.—Area of dulness increased downwards and outwards. Apex beat two inches below the nipple. First sound reduplicated at apex. Second sound short and sharp. Sounds of reduplication become less marked towards base, and at base cannot be heard.

Abdomen natural. Some slight tenderness over liver.

Tongue slightly furred. Appetite good.

Bowels regular. Urine, acid, sp. gr. 1015; albumen $\frac{1}{6}$th.

Treatment—confined to bed and ordered milk diet.

February 24.—The right hand and wrist swollen, inflamed, and painful. The left hand in the same condition. Has passed a good night. Pulse 70. Tongue furred. Albumen in urine $\frac{1}{12}$th.

February 25.—Slept well. Hand as yesterday. Has had it wrapped up in cotton wool. Albumen in urine $\frac{1}{6}$th. Ordered tinct. colchici ♏xx. out of haust. calumb. alkal. ter die.

February 26.—Slept well last night, and sleeps a good deal in daytime. Very little pain in left hand. Right hand still painful. Last night passed water in bed unconsciously, but micturition normal this morning. Tongue furred thickly. Temperature 100.6° last night, normal this morning. Pulse 84. Bowels not open since yesterday. Ordered haust. sennæ co. ℥iss. Albumen $\frac{1}{8}$th.

February 28.—Has slept well. Tongue furred. Hands much better, no pain. Albumen in urine $\frac{1}{20}$th. Bowels open. Temperature normal.

March 1.—Continues to sleep well. Bowels open. Tongue still furred. Both hands in much the same condition as 28th ult. Urine sp. gr. 1015. Albumen a trace.

March 2.—Has passed 1¾ pint of urine from 10 A.M. yesterday to 10 A.M. to-day. Albumen a trace. Hands somewhat better.

March 4.—Much better. Swelling in wrists nearly subsided. No pain except a slight twinge now and then. Tongue clean. Pulse 72. Bowels not open for two days. Ordered haust. sennæ co. ℥iss.

March 7.—Hands nearly all right. Passes from oiiss. to oiij. of urine per diem. Albumen a trace.

March 10.—Hands quite well. General health good. The colchicum mixture stopped. Patient can move his leg much better, and the arm somewhat. Face also much improved.

On the evening of 24th, patient had a slight attack of gout in *left* great toe, up till which time his condition had greatly improved. Colchicum mixture was again ordered, and patient was all right again on 26th.

From this time up till the day he left the hospital (April 19) he greatly improved. He had no more attacks of gout, and left not cured of his paralysis, but greatly relieved by treatment.

It is not improbable that gout may sometimes have been suspected in cases of paralysis where arthritis has come on in connection with the cerebral lesion, constituting examples of Charcot's *arthropathie des hémiplégiques*.[1] Scott Alison directed attention to such cases in 1846 (Lancet, January 16).

Charcot relates a case of a woman, aged forty-nine, suddenly seized with hemiplegia. Some days afterwards pain, heat, and swelling occurred in the wrist, then in the knee and foot of the paralysed limb. The limbs were slightly rigid. Cerebral softening was found after death. In the renal pelves were numerous uric acid calculi. The same author also records the following cases:—A man, aged fifty-four, a painter, suffering from saturnine gout, became suddenly hemiplegic. Shortly afterwards, the wrist, hand, and foot of the paralysed side were attacked with gout.

[1] *Leçons sur les Maladies du Système nerveux*, tom. i., Paris, 1877, p. 114.

The affected limbs were rigid. Hæmorrhage was found in the brain.

A woman, aged about forty, had right hemiplegia with aphasia of three years' duration, the limbs being very rigid. Attacks of pain and swelling occurred in several of the joints of the paralysed side. At the autopsy, signs of old cerebral hæmorrhage were found, and the cartilages of the affected joints were encrusted with crystallized and amorphous urates. There were no deposits in the cartilages of the joints of the sound limb, but some were found in the kidneys.

In all these cases there can be little doubt as to the truly gouty nature of the concomitant arthritis, and of its determination to the paralysed limbs. M. Charcot is careful to state in respect of these examples that they were quite exceptional, and very different from those in which arthritis occurred without rheumatic or gouty taint, and, solely, as a concomitant of the brain-lesion.

The following case, recorded by M. Bourneville, is important.[1] It was that of a woman, aged fifty-four, who had had an attack of right hemiplegia five years previously with aphasia, probably due to cerebral hæmorrhage. A second and fatal apoplexy occurred. At the autopsy, a large fresh hæmorrhage was found in the brain, extending into all the ventricles. In the knee and great-toe joints of the right (paralysed) limb were found uratic encrustations, but none were detectible in the corresponding joints of the left limb. The kidneys appeared natural, with the exception of uratic streaks in some of the tubules. In the knee the synovia was bloody, and bony outgrowths, with "lipping," were found. M. Bourneville was of opinion that the deposits were locally determined by the paralytic state of the limb.

I fully recognize the occurrence, occasionally, of arthropathy in hemiplegia, and I would suggest that the same theory of its production be entertained in cases where gouty arthritis supervenes in paralysed limbs. The nervous element appertaining to gout is here often, if not always, the determinant factor in localization. Exceptions occur, but they do not invalidate the general rule in such cases.

My hospital experience has supplied me with other instances in which gout has alighted on paralysed limbs.

[1] *Études cliniques et thermométriques sur les Maladies du Système nerveux.* Paris, 1872, p. 58.

Recoverable Paraplegia in a Gouty Man.

Dr. Wilks records a case of recovery from paraplegia in a man, æt. fifty-two, who was subject to gout and had not had syphilis.[1] A month before he went into Guy's Hospital he had gout in the feet, and after two weeks was losing power of the legs and bladder. The paralysis rapidly increased. On admission he could not move his legs. There were partial anæsthesia as high as the umbilicus, and well-marked reflexes. The urine was drawn off twice daily and was ammoniacal. Girdle-sensation and numbness ensued, passing down arms to fingers. The patient got worse, became feverish, had rigors, hiccup, and a red tongue. A bed-sore formed, and the mind was clouded. Suppurative nephritis, by extension from the bladder, was suspected. Was very ill for some days, when symptoms abated, and some power in legs returned. Rapid recovery followed, and use of catheter became unnecessary. The patient began to stand, and to walk on crutches, and left the hospital two months after admission, or six weeks after his paraplegia was complete.

It is not easy to surmise the cause of the myelitis which occurred in the case just recorded, and we have no knowledge, as yet, to warrant the belief in meningo-myelitis due to gouty inflammatory change. I conceive it to be possible, however, for such a disorder to occur.

Idiosyncrasy in Relation to Gouty Proclivity.

One of the most noteworthy features attaching to gouty tendency is that of idiosyncrasy. The whole subject is a large one, and would well repay more accurate and scientific study than it has yet received. It can only be generally referred to here. I allude to it in this chapter because I conceive that the nervous system is essentially concerned with the special peculiarities under consideration. There are always present the personal and individual factors, each person being a law to himself.

In respect of gout, the very general inability to partake of certain alcoholic fluids is idiosyncratic to the mode of tissue-metabolism attaching to persons thus impressed.

The variety of personal peculiarities in respect of food (including flavours and odours), air, water, medicines, climatic and other environments, met with in the gouty, is great and quite remarkable. For purposes of treatment, it is important to take note of any one of these that may be manifested. I suppose that no other known diathetic state has so much of idiosyncrasy attaching to it as gout.

Many of the dietetic peculiarities recognized are met with in the gouty or their descendants. These may occur at an early period of life, or may supervene at any time, and it is certain

[1] Diseases of Nervous System, p. 229, 1878.

that they may last for a term and completely disappear. We have, therefore, to note a certain periodicity as attaching to idiosyncrasy, inability present at one time of life yielding completely by lapse of time. Such periodicity is also seen in certain gouty ailments which are the appanage at one time of youth, and pass off, to yield, perhaps, to fresh proclivity or idiosyncrasy. Examples in proof of this are recognized in the tendency to sore throat, hepatic disturbance, loathing for animal food in childhood and early adult age, in megrim-tendency of early and adult life, which yields in middle life, and in carbuncle and glycosuria, which are apt to supervene in more advanced life.

The facts relating to gouty idiosyncrasy throw a strong light on the nervous element which figures so largely as a factor in the whole disease.

It may be boldly affirmed that there are no known means whereby an idiosyncrasy may be overcome. The peculiar nervous potentiality is in possession so long as the peculiarity endures.

Our knowledge of its natural history affords warrant for the belief, or at any rate for the hope, that in course of time it may yield and pass away. While it is present, it has to be reckoned with as a dominating factor, and must be regarded as a special form of nervous impressibility—a part, as I believe, of the peculiar gouty neuro-trophic habit.

CHAPTER XI.

SUGGESTIVE METHOD FOR INVESTIGATION OF CASES OF GOUT.

Gout—Clinical history—Evidence of family predisposition—Heredity—Modification by transmission—Association with other diseases; e.g., *commingling with rheumatism, struma, cancer; gout cross-tainted with syphilis or other diseases.*

Initials of patient. M. or F. Age.

Residence.

Occupation of patient and parents (especial reference to alcoholic excess, or exposure to lead- or lime-impregnation).

Total abstainer, how long?

Stout, moderate, spare. Strong, moderate, weak.

Complexion, vascularity, pallor.

Head, large.

Integumentary System.—Complexion, vascularity, pallor, mixed. Hair, colour, date of greyness, baldness. Head, large. Temporal artery, tortuous. Skin, thick, smooth, very smooth. Ears, undue hardness of, tophi, condition of meatus, deafness. Eyes, arcus senilis, conjunctival œdema, suborbital œdema. Nails, coarsely striated, smooth, brittle. Nose thickened at end. Eczema, eczematous ulcer, painful at night. Urticaria. Lichen. Herpes. Psoriasis. Pruritus. Pruritus ani. Pruritus vulvæ.

Eyes.—Condition of iris, old iritis, adhesions. Cataract. Retinal changes. Flame-shaped hæmorrhages on disc. Glaucoma. Conjunctival ecchymoses, episcleritis, sclerotitis, ophthalmitis.

Articular System.—Suppleness generally in neck, spine, limbs, and extremities. Cracklings on movement, *e.g.*, in spine. Condition of metacarpo-phalangeal and phalangeal joints, as to knottiness, distortion, altered axes. Fluid or tophitic effusions, bony enlargements, œdema, ankylosis. Heberden's nodes in true gout. Deflection of digits to ulnar aspect, or not. Great toe-joints, deflected outwards, or not, enlargement, crackling. Condition of bursal sacs as to thickening, effusion,

tophi. Pellucid cysts near joints. Ankylosis, true, (synostosis) or false. Periostitis. Dupuytren's contraction of palmar fascia. Nodules.

Lymphatic System.—Condition to be noted in cases untainted by struma. Leuchæmia, splenic and lymphatic.

Circulatory System.—Heart, position of apex-beat; signs of enlargement. Sounds, pure, reduplicated, aortic second sound, character. Valvular lesion. Pulse, quality, rhythm, condition of arterial coats, tension. Angina pectoris. Veins, venous remora. Varix. Phlebitis. Palpitation, cardiac, thyroideal, and in abdominal aorta. Sphygmograms. Hæmophilia. Graves' disease.

Respiratory System.—Tendency to quinsy in early life; one-sided tonsillitis. Laryngeal catarrh. Bronchial catarrh. Emphysema. Pneumonia and character of sputa. Dry pleurisy. Hæmoptysis. Epistaxis in early life, or at any period. Spasmodic asthma. Phthisis, family history. Commingling of gout and phthisis.

Digestive System.—Teeth, regular, massive, small, well-enamelled, colour, worn down, freedom from caries, or reverse; sound teeth ever extruded; "buck teeth;" alveolar absorption, tenderness, gums retracted, blue line (lead), tartar. Tongue, conditions of: undue dryness at times, deep-seated pain in; psoriasis of; neuralgia of. Fauces, unduly red, coarsely granular. Uvula, long, glossy. Parotitis. Saliva, sulphocyanide of potassium in excess. Pharynx, condition of mucous membrane. Œsophagismus. Gastric digestion, acidity. Intestinal digestion, much flatulence, effects of various wines and beer, cardialgia. Idiosyncrasies as to food. Gastralgia. Liver, natural, tender on palpation. Hepatic colic. Gall-stones. Enteritis, colic. History of bilious attacks; hypochondriac fulness; shoulder-tip pains; omentum and abdominal parietes fatty. Bowel-evacuation, colour and character of stools. Diarrhœa. Hæmorrhoidal tendency.

Genito-Urinary System.—Renal pain, as distinct from lumbar pain due to other causes. Calculi. Gravel. Urine, character, quantity, &c., before attack, during, and in intervals, colour, reaction, sp. gr., deposit, casts, albumen, glucose, urates, oxalates, urea, uric acid; cystitis. Quantity. Nocturnal micturitions. Bladder, irritable. Hæmaturia. Prostatitis with retention of urine; stricture; fibroid thickening. Priapism (nocturnal). Urethra. Thrombosis in corpus cavernosum. Herpes or eczema of glans. Excessive venery in early life. Varicocele. Orchitis. Gonorrhœa, gleet, characters of, if intractable. Menorrhagia. Amenorrhœa. Vicarious hæmorrhages.

Nervous System.—Temperament, cheerful or melancholic disposition. Hypochondriacal tendency. Hysteria. Temper. Energy. Sensitiveness to pain. Mental power and capacity. Neurotic history in relatives or patient (insanity, epilepsy, hemicrania, neuralgia, angina pectoris, spasmodic asthma). Sleep, tooth-grinding during, startings in, somnambulism. Cramps in calves or elsewhere. Fleeting pains in knuckles, &c. Pain in heel, tendo Achillis, in xiphoid cartilage. Sensations of burning

in palms, thighs, and soles. Tickling and pain in palate. Lumbago. Sciatica. "Pins and needles" sensation. Numbness. "Dead fingers." Vertigo. Neuralgia of fifth and great occipital nerves. Neuritis. Paræsthesiæ. Convulsions.

Nervous symptoms premonitory of attack.

Family History.—Gout. Osteo-arthritis. Rheumatic fever or any arthritic condition. "Chalky" gout. Diabetes. Dietetic habits and residence of ancestry. History of any members who have lived out of British Islands. Ages at which gout appeared. If younger members suffered earlier and more severely. Medical history of ancestors as fully as possible.

In Women.—Health during menopause. First gouty manifestations, if articular, where?

Chronic Gout.—Tendency to polyarthritis, simulating general rheumatism. Modifications of attacks. Alternations with any neurotic attacks; substitutive manifestations. Glycosuria.

Visceral Attacks.—Gastric. Hepatic. Enteric. Pulmonary. Vesical. Encephalic.

Climatic Influences.—Season, special influences of cold, east wind, damp; of mountain and sea air. Town and country life. Gout in India, Tropics, and Colonies.

Dietetic Influences.—Fish-eaters. Vegetarians. Meat-eating.

Occupation.—Profession. Habits, active, sedentary. Open-air life.

CHAPTER XII.

PREMONITORY SIGNS OF GOUT—CLINICAL VARIETIES OF GOUT—ACUTE (REGULAR) AND CHRONIC (*A.* TOPHACEOUS, *B.* DEFORMING) GOUT—GOUTY CACHEXIA—IRREGULAR (INCOMPLETE) GOUT.

Premonitory Signs of Gout.

THE precursory symptoms of sthenic gout in the earlier attacks are few and little marked. In later attacks these are more pronounced, and also better heeded by the patient. The earliest warnings come from the digestive and circulatory systems.[1] Some degree of dyspepsia is commonly noted, accompanied by a sense of fulness at the epigastrium and in the hepatic region. Heartburn, sour eructations, and flatulency are the leading dyspeptic indications. There may be headache. The urine is apt to be charged with lithates. Irregular action of the heart is observed, with throbbing and palpitation, these irregularities of impulse being felt in the head, especially on exertion. The pulse is apt to be firm or tense. Sighing expiration is sometimes present. Hæmorrhoids may occur. On the side of the nervous system are found mental depression, neuralgia, hemicrania, drowsiness and yawning, deep-seated pains in various parts, sometimes violent, and of momentary duration. These may be felt in the limbs or feet, or in the joints of the fingers. Pain in the calcaneum and plantar fascia is characteristic of gout, and so is general pruritus.

Lumbar pain, toothache in sound teeth, sharp pains in the tonsils and many other parts may likewise be warnings of a paroxysmal attack. Some sallowness of the face and icteric tint of conjunctivæ may be observed. Ptyalism was noted by Scudamore. The bowels are often confined, and the stools pale. As articular pains supervene, dyspeptic symptoms may pass away. The appetite has often been observed to be unusually good the

[1] "Its only forerunner is indigestion and crudity of the stomach, of which the patient labours some weeks before."—*Sydenham, op. cit.*

day before the paroxysm,[1] and a general sense of *bien-être* or euphoria—too commonly a bad sign in many diseases—may be experienced. As a rule, scanty secretion of concentrated urine is observed before an attack; but free urination sometimes precedes a gouty fit. I have observed this sometimes, and Scudamore believed that it seldom occurred except in persons of nervous temperament whose constitutions had been much weakened by gout. My experience supports this view. Graves recorded two examples of this in hereditary cases.[2]

Slightly marked attacks of ophthalmia, affecting the sclerotic tunic and the conjunctiva, have been sometimes noted as precursors of gout. The congestion is not usually wide-spread, and is chiefly seen near the insertions of the orbital muscles. The nights may be disturbed by various dyspeptic symptoms, and the sleep is not refreshing. The mornings are unwelcome, the appetite fails, errors in diet are quickly recognized, and there is disinclination to face the common duties of the day. As psychical symptoms, peculiar irritability and shortness of temper may be witnessed, and mental effort is difficult and laborious, or impossible.

As might be expected, these symptoms will vary according as an outbreak is determined by any special cause, or led up to by some peculiar train of circumstances. As a rule, all of them disappear with the onset of a paroxysm of arthritis. Such predetermining causes are well-recognized. Prolonged mental labour, undue strain, anxiety, pressure of business, confinement in-doors, absence of wonted exercise, a succession of indulgences at the table, any one of these, or, not infrequently, a combination of two or more of them, may readily lead up to a sharp attack of gout.

On Determinants of Gouty Paroxysms.

Allusion has already been made to several conditions which appear to determine articular attacks of gout.

Traumatism, shocks, bodily and mental, loss of blood, and various ailments are amongst some of the recognized determinants. Many of these doubtless induce changes in the nervous system, and are of a character to upset the general equilibrium of nutritive processes throughout the body.

Attacks of severe influenza have been noted with some frequency to be followed by paroxysms of gout. Amongst the many

[1] Scudamore records a case where there was excessive appetite for meat two or three days before a fit.

[2] Clin. Med.

proclivities of those suffering from incomplete gout is a tendency to such catarrhal attacks as are commonly called "bad colds," which occur especially in the form of coryza and gravedo, with sore-throat, leaving the patient weak and depressed for some weeks.

Severe purging is a determinant, especially in well-established gout. Excessive venery is thus recognized. Changes of accustomed habits in respect of exercise and diet, be they either in the direction of excess or deficiency, are readily provocative in those goutily disposed. Exposure to cold with check to perspiration is sometimes an efficient cause of a fit of gout.

Paroxysms have been induced with remarkable suddenness by indiscretions in diet. A single glass of champagne has been known to induce an attack within a few minutes. In such a case there must have been all the necessary conditions ready for the induction of a fit. The train was fired, as it were, by the last addition to the blood of such matters as sufficiently reduced its alkalinity. The balance of health is, therefore, very readily upset in these instances, and where such instability exists, other than dietetic errors may be effective as determinants, such as shocks, strong emotions, and, indeed, any conditions capable of producing profound impressions.

Hydropathic treatment is frequently a cause of acute paroxysms. This is well recognized at the various Spas. Change of habits in diet and general regimen, together with the special influence of baths and water-drinking, suffice to induce attacks, and these are sometimes found to be so far salutary as to clear the system, and secure a more lasting immunity from future paroxysms.

Inflammation, as commonly understood, forms no essential part of the pathology of gout—neither does pain. Instances of unequivocal gout occur in which changes in joints take place without any of the classical symptoms of an acute paroxysm. There may be intense pain, but no heat, redness, or apparent change in the affected part, and the pain may come on without any warning.[1] I have already described a case where "quiet" gout occurred

[1] Dr. W. Gairdner was familiar with such cases, and gave an account of such an attack, during dinner, in a gentleman, in whom sudden agony occurred in a great toe-joint. The pain lasted half an hour, and passed off as suddenly as it came. No change was observable in the joint. No warning symptoms had been present. Very early the following morning the other great toe-joint was similarly attacked. Three weeks later a typical attack occurred in the heel, with every symptom of true gout. He quotes another case from van Swieten's Commentaries, and in this instance unequivocal gout followed in twelve months. From these and other cases Dr. Gairdner sought to prove that "the nature of gout is the very reverse of inflammatory."—*Op. cit.*, 3rd edit., p. 139.

gradually, leading to severe crippling with much uratic deposit, and where no pain or overt gout ever occurred in the parts.

Prolonged mental labour and intense study may precipitate a fit. As Sydenham remarked, "Quoties enim ad hæc studia me recipiebam, toties et podagra recurrebat." Venæsection and loss of blood are to be reckoned amongst determinants.

Acute Gout.

"We learn acute disease from seeing it as a whole; from seeing it as it is acted and suffered through all its stages by the same individual men and women. Being an affair of a few days or a few weeks only, we are often present as eye-witnesses of it from first to last. Thus our knowledge of it is drawn from single and complete histories."—PETER MERE LATHAM, *Diseases of the Heart*, 1846, Lect. xxxvi.

This constitutes the form in which gout is commonly regarded, the classical arthritis or podagra.[1]

When typically manifested, its character and symptoms are readily recognizable, and should not be confounded with any other condition.

In a first attack, however, the patient himself is not unfrequently sceptical as to the diagnosis. It may be, and often is, his first serious ailment of adult life. He has, perchance, prided himself on his robust constitution, on his activity and muscular capacity, and would fain believe that he is as yet too young to be the victim of a trouble which he conceives to be the appanage of advanced years, and a Nemesis for over-indulgence in the good things of the table.

He would rather believe that he had suffered some sprain or direct injury to the part, or that he was merely "rheumatic." A second attack commonly brings conviction to the sufferer, and he forthwith acquiesces in his fate.

A careful consideration of the several features of a well-marked case should prevent mistakes in the diagnosis of the local affection, for it may be asserted that no other known malady attacks a joint after the manner of gout. Other forms of arthritis may affect the gouty, and, thus, difficulty be met with,[2] but the rule will commonly hold good—a suddenly induced acute monarthritis

[1] Various epithets are applied to this form of gout, *e.g.*, "regular," "articular," "frank," "paroxysmal," "sthenic."

[2] Such an instance is related by Paget,* where a pyæmic abscess formed near a great toe consequent on tying piles.

* Clin. Lect. and Essays, 2nd edit., p. 358.

is, in the majority of instances, an unequivocal manifestation of true gout.

The leading features of a regular fit of the gout relate more especially to its peculiar mode of onset, the specifically intense painfulness, and the locality most apt to be the seat of first attacks.

No description, however minute, will apply to the symptoms met with in each case of a fit of gout. In each individual affected by disease there is always a personal element, and the manner of his suffering depends on one or more factors pertaining to his peculiar habit of body, inherited tendencies, age, and circumstances. The symptoms are much modified by the special constitution of the nervous system as to sensitiveness and reaction in each person. The classical attacks described by Sydenham are by no means the rule, even in the first onset, and in cases that may be termed sthenic. The nocturnal paroxysm is, in particular, less often met with than would be imagined if the ordinary accounts of the text-books were to be implicitly relied on. Suddenness of onset, also, is often not met with in cases that within a few hours become very acute. A classical attack of *podagra*, at all events in the earlier history of a patient goutily disposed, is not infrequently characterized by its suddenness, by its excruciating torture, and by its incidence in the proximal joint of one or other great-toe.

Most physicians have agreed as to the fact that in most cases the four cardinal symptoms of inflammation are present, and have asserted that there may, thus, be found *tumor*, *rubor*, *calor*, *et dolor*. To this concise definition should be added the very frequent occurrence of the attack in the early hours of the morning.

Statistics in proof of the accuracy of the above statements are not wanting,[1] and the experience of all practitioners daily confirms it.

Exception has been taken to the assertion that a noteworthy amount of inflammation is invariably present,[2] and I conceive that such an objection may be made by those who see a great deal of gouty disease; but there can be no doubt that in the majority of cases of acute, regular, and localized gout, there is present, as part of the process, an obvious measure of inflammation.

In an early fit of acute, regular, sthenic gout, the patient, who

[1] Scudamore, *op. cit.*, 3rd edit., 1819, p. 25.

[2] *Vide* Gairdner on Gout, 2nd edit., 1851, p. 108.

may have previously had premonitions of an attack—*e.g.*, violent cramps in the calves—usually retires to rest feeling no particular disturbance of health, or even in better condition than is his wont. He is awakened in the early hours of the morning by violent pain, commonly in one great toe-joint. A slight rigor sometimes occurs at this time. The pain increases to positive agony,[1] and there is much restlessness. A sense of extreme tension, sometimes with throbbing, is experienced. An easy position for the foot is sought in vain. The slightest vibrations aggravate the pain, so that the sufferer resents even the movement of the bed-clothes, or the tread of a person on the floor of the room. The pain is excruciating, and quite peculiar to the earlier processes of gouty arthritis. Nothing at all like it occurs in any other joint-disease.[2] After some hours a measure of relief is obtained, sometimes gradually, or quite suddenly; perspiration occurs, and sleep follows. On the following day the affected joint is found swollen, dusky red in colour, tense, shining, and very sensitive to touch. The pain is apt to continue with more or less intensity during the day, and to rage again towards evening. The dorsal veins of the foot are observed to be turgid. The pulse is quickened, 80 to 100, and the temperature may rise from 101° to 102°. It seldom rises higher. I have several times met with 103°, and Garrod has once found it 104°.

The temperature of the inflamed joint, though feeling preternaturally hot to the hand, may be below normal. I have found it 97°, while that in the mouth at the same time was 100°, and the corresponding unaffected joint was 96.3°. In another instance, with a temperature of 100.4° in the mouth, I found the joint registered only 95.6°. These temperatures were taken by wrapping up the joints in cotton-wool and inserting the thermometer for twenty minutes.

The symptoms just described may recur for several days. Œdema of the superjacent integuments sets in gradually, and increases up to the fourth or fifth day, when it is at its height, and readily allows dimpling. There may be ecchymoses. The

[1] "Tanquam ossium dislocatio."—*Sydenham.*

[2] "The pain is altogether disproportionate to the other signs of inflammation, and, even more, to the consequent structural changes in the inflamed part."—*Paget.*

"The inflammation of the gout is very different from the adhesives and suppurative in its sensation. It seldom throbs; it is a pricking, cutting, and darting pain, besides which there is a pain which feels as if the inflamed parts were all moving, and in that motion there was pain; therefore the action which is the cause of the pain must be very different, and is most probably from the action of the vessels, not from their distension, as in the suppurative inflammation."—*Treatise on the Blood and Inflammation*, John Hunter, p. 266, 1794.

redness also passes off gradually with the venous turgescence. With the onset of œdema comes relief to pain.[1] Severe cramps in the calves sometimes occur. There is commonly a furred tongue, dirty yellow in colour, a foul breath, much thirst and aversion from food. There may be a bitter taste in the mouth. Hiccup and eructation may occur, but no vomiting. The bowels are usually constipated; if not, there may be pale or very dark offensive stools. The urine presents the ordinary febrile characters, being dark in colour, scanty, and concentrated, and depositing red lithates or crystals of uric acid. There may be a trace of albumen lasting through the attack. The cuticle is next observed to crack and degenerate, this process being accompanied with much itching. In the case of a small joint, it is not possible to detect effusion within it; but in the case of the knee or ankle, this sign is evident.

The arthritis having passed off, the patient rapidly recovers his wonted health, and feels better than for some time previously. Some weakness and stiffness remain in the joint for a few days, and recovery is established. The whole attack generally runs its course within a week or ten days.

I have already remarked of the mode of onset of a gouty paroxysm that this peculiarity stamps a nervous character upon the malady, and allies it with other explosive neurosal disorders.[2]

The intense painfulness is specific, and pertains, as I have stated, to no other form of arthritis. Scudamore noted that there was most sense of throbbing when the great-toe was involved; that there was more sense of weight and loss of power when the tarsus was the seat of the attack, and that the feeling of tightness was most urgent when the elbow and carpus were implicated. Gene-

[1] Attention has been pointedly directed to the occurrence of œdema as part of the inflammatory process. It is commonly present, but I cannot agree with those observers who regard it as absolutely pathognomonic of true gout. A measure of œdema is always present in acute inflammation, and constitutes the outermost ring or zone of simple (serous) excitement, as the late Professor Miller, of Edinburgh, termed it. A slight degree of such swelling is certainly found in connection with rheumatic inflammation, and in other forms of arthritis, albeit it is often a marked feature in acute attacks of gout. In chronic forms of both gouty and rheumatic inflammation œdema is not recognized. The desquamation of the cuticle, which very commonly occurs after acute gouty inflammation, is certainly peculiar and characteristic. In many instances this process is, I believe, at least encouraged by the various applications which are used to mitigate the pain during an attack, and the degree of previous inflammatory distension, as in the case of erysipelas, may also determine the amount of it. Trousseau aptly likened the appearance of a part recently affected with true gout to the outer pellicle of an onion.

[2] *Vide* p. 48, chap. iii. The time at which seizures commonly occur is in the early hours of morning. At this period there is the greatest cessation of activity of the nervous system, predisposing to sleep.

rally, it may be affirmed that gout of the lower extremities is more insupportable than that of the upper limbs, and an experienced sufferer declared to Scudamore that the two most painful parts to be involved were the ham-strings and ligament of the patella; and this I can readily believe, since it is very difficult to prevent motion in these structures. Gout in the nape of the neck is also extremely painful. Sydenham truly noted the constant tendency of the pain to increase at night and remit in the morning.

The special implication of a great toe-joint, especially in early attacks of gout, has long attracted attention, and been much discussed. The most satisfactory explanations to my mind relate to the inordinate exposure of these joints to hard work, compression, and injuries, whereby there is always tendency to damage of the integrity of their component structures. The entire weight of the body is borne by these joints. In healthy persons, as our observations at St. Bartholomew's amply prove, it is common to find erosion of the investing cartilages, more especially about the centre of the cup of the first phalanx. Such a part is therefore predisposed to uratic infiltration and deposit as a *locus minoris resistentiæ*. Next to the great toe the knee-joints are most apt to suffer erosions, and, so, are similarly predisposed to attacks of uratic arthritis.

I have already stated that too much blame must not be attached to tight boots, since gout has from all time shown predilection for the great toe-joints. Improperly fitting boots, however, may very well cause damage to these structures.

It must, however, be stated that attacks of acute articular gout are not always violent or paroxysmal, even in the first instance. They may supervene gradually, becoming, however, fairly "classical" at last. Whether such shall, or shall not, be the character of the attack depends probably on the degree of goutiness present at any given time, and no less on the special proclivity of the individual and the determinant of the fit.

Many attacks begin during the day, and this is perhaps more often the case after the disorder is fully established.

After a primary, regular fit, some time may elapse before a second one, much depending upon the degree of heredity, the treatment, and the fortitude of the individual in altering his habit of life.

Cases are met with in which one or two attacks are all that ever occur in a lifetime. It is more common, however, once the gouty habit is declared, for renewed paroxysms to supervene, and these tend to recur not only in the previously-affected localities,

but to involve other joints centripetally, seizing on the tarsus, ankles, knees, hands, and elbows.[1] The hip-joint and shoulder are singularly immune, but not altogether exempt from uratic arthritis, herein presenting a noteworthy contrast to the course of chronic rheumatic arthritis. In some cases, fresh attacks supervene with very brief intervals, and the general health is seriously impaired. Primary attacks are not uncommon in other than the great toe-joints, the knee being very prone thus to suffer, likewise the tarsus and ankles. In such cases this departure from the common habit of the disease may be sometimes explained by previous injuries or strains of the parts affected, or by certain occupations which lead to over-use of and damage in joints.

An attack of acute gout which passes off completely in a few days, probably leaves very little permanent change behind it. Acute rheumatism is known to leave none. We are here in face of the fact that each attack, so far as is known, leaves its trace in the form of some amount of uratic deposit on the articular cartilage or other tissue of the joint. Naturally our knowledge is, and must be, very limited as to this. There is reason to believe that acute attacks sometimes supervene in parts which have long been the seat of quiet deposit, with or without subacute inflammatory reaction. In such a case there would be *post-mortem* evidence of chronic arthritis, with more or less uratic deposit, and the clinical symptoms of the case would alone have given token of any disturbances prior to the acute attack. In such cases there may have only been occasional twinges of pain or of uneasiness in the joint. Sometimes, actual deformity has occurred before any acute attack supervenes, due to ostitis and some erosion or damage to the cartilages. These are really examples of chronic gout with acute inflammatory exacerbations.

I am here discussing the permanent coarse changes due to acute attacks, and for these we must look alone to the joints, for we cannot expect to find elsewhere any traces of the malady in its early stages.

Early attacks may leave behind them permanent crippling and deformity. True ankylosis may occur, and this is peculiar to gouty arthritis. Bony union—synostosis—is never met with in rheumatic disease. I have twice seen complete synostosis of the first metatarsal bone with the phalanx. "Lipping" of the edges of the joint may occur, giving rise to bunion, and to extreme deflection of the digit to the outer side of the foot.

[1] "After it has attacked each foot, the fits become irregular, both as to the time of their accession or duration."—*Sydenham.*

It is noteworthy that repeated attacks of true gout may in some cases leave no traces behind in the form of uratic deposit. I have recorded one such instance.[1] The case was of great interest because of a rare form of cardiac disease in which reflux occurred through the pulmonary valves. It was that of a man, æt. forty-nine, who had had many attacks of gout in both great toes, some under my own observation. At the autopsy no deposits were found in either toe-joint.

It is rare for gouty fits to involve more than one or two joints in its earlier periods, but cases occur in which many articulations are seized (polyarthritis uratica). The diagnosis then becomes somewhat difficult, since such a manifestation resembles acute rheumatism. The history of the illness, the personal history, the age of the patient, the temperature, the absence of sour sweats and of cardiac implication should avail to render the diagnosis almost certain. There is no incompatibility between true rheumatism and true gout in the same individual, and the previous occurrence of the former may possibly tend to the establishment at a later period of gouty polyarthritis; but such cases are certainly not often encountered in practice. The existence of tophi anywhere should promptly throw light on them.

Garrod's blister-serum test may be had recourse to in any uncertainty. I have met with two or three of these cases. They do not respond promptly, if at all, to the influence of sodium salicylate, colchicum being much more efficacious, and, thus, indicative of their true nature.

Repeated regular attacks may occur, and no indications of uratic deposit be met with in any part, even after many years. Patients vary infinitely in this respect.

Where there is great tendency to formation of tophi around joints or in the ears, the encrustation may proceed to great excess without painful paroxysms bearing any proportion to the deposition. There is, in fact, a special clinical type of gout in which this tendency predominates, the upper extremities being by far the most involved, and the deposit proceeding, as it were, quietly but ruthlessly towards the most hopeless crippling of the parts, the fingers assuming the "parsnip-type" and becoming enormous.

An acute attack may not pass off completely in a few days. After repeated attacks there is tendency sometimes for gout to linger for weeks, and even months, in the part. This is mostly seen in older persons with enfeebled constitutions, but it may be met with in patients under fifty years of age, still robust. Syden-

[1] Clin. Soc. Trans., vol. xxi. p. 18, 1888.

ham considered such an attack to represent a series of minor fits rather than a prolonged paroxysm.

Acute gout in the foot is probably the least harmful form in which the disease occurs, because this indicates the most regular course.

The mental faculties remain clear, as a rule, during the progress of an acute fit, and sometimes there is full capacity for intellectual labour during the paroxysm.[1] The influence of the mind and of a strong will in "fighting down" the gout is in some instances very remarkable, so much so, that the usual progress of an attack appears to be modified or even arrested.

An indication of past attacks is sometimes met with in the form of transverse depressions on the nails of the toes, as described originally by M. Beau,[2] who noted their occurrence after acute general illnesses as well as after severe local disturbances. As a complete nail takes six months to grow, the site of these depressions affords a fairly accurate indication of the date of the past attack.

W. Gairdner believed that the left limbs were more often affected than the right, and he found metastasis more common from the right to the left limbs. Laycock and Gull taught that all actions, both healthy and morbid, were less energetic on the left than on the right side of the body.

Scudamore, Garrod, and Braun, of Wiesbaden, have proved by their statistics the very marked predilection for the great toe-joints in early attacks. These are the parts commonly affected first in hereditary gout, and it is rare for the upper extremities to be the seats of primary attacks.

Acute and Regular Gout less common than formerly.

It is certain that instances of acute gout—cases, that is, of classical podagra—are less frequently met with than was the case in Sydenham's time, or even a century ago. An inquiry into the cause of this infrequency must yield assistance in studying the ætiology of gout, and a ready explanation is at once afforded by the marked change in dietetic habits now prevalent. The gross over-eating and disgusting alcoholic intemperance which were common amongst the wealthier classes of society at the time referred to, happily, no longer exist.

It may also be regarded as certain that the excesses of the last century have left an ample legacy of gouty disease in this country,

[1] *Vide* chap. xxi.

[2] M. Beau pointed these out to me in his wards in the Hôtel-Dieu in 1862.

but it is highly probable that the amended dietetic habits of more recent times, and in particular the diminished consumption of the strong wines of Spain, Portugal, and Madeira, have much to do with the comparative infrequency of sthenic gout at the present time. Sir George Burrows informed me twenty-six years ago that he then saw fewer cases of acute gout than he was accustomed to see in his earlier practice. Amongst subsidiary causes for the diminution of acute gouty disease, may probably be reckoned the better ventilation and hygiene of modern times, and the great increase in the consumption of water, especially in the forms of various mineral waters. The increased use of the lighter and thoroughly fermented vintages of the Médoc, in place of heavier and incompletely fermented wines, and of strong ales, together with the results of the several earnest crusades against alcoholic intemperance, may also be credited with noteworthy results in this direction.

Although there may be less acute and typical gouty disease than formerly, the opinion gains ground that many of the manifestations of gout at the present time are modified by inheritance and change in habits, so as to be less easily recognized. Thus, there are now met with cases of incomplete or imperfect gout, as well as a variety of disorders, which, by reason of their constant association with gouty inheritance, mode of onset, response to specific treatment, and their general habit, are fairly to be reckoned as the outcome of gout and as indications of it.

That gouty diseases and manifestations are very common at the present time cannot be doubted, and when all allowance is made for the *dicta* of, so-called, "fashionable physicians" respecting the great prevalence of gout, it must be affirmed that many of the ailments of the affluent classes in this country are modified by, and often dependent upon it.

Chronic Gout.

"We do not learn chronic disease from seeing it as a whole, as it passes through all its stages in the same individual men and women. Being an affair not of days or weeks, but of months, and many months, and oftener of years, and oftener still of many years, we are indeed very seldom present as eye-witnesses of it from first to last. Thus our knowledge of it is not drawn from single and complete histories, but put together piecemeal from numerous imperfect ones. . . . The most useful and perhaps only just representation that can be made of chronic disease is not in the way of description, but of commentary."—PETER MERE LATHAM, *op. cit.*, p. 358.

It is not easy to determine precisely when the acute form of the disorder passes into the chronic variety. The latter condition

represents a stage when acute paroxysmal attacks have become frequent and well-nigh continuous, complete recovery in the parts affected, and of the system generally, not being attained.

Recurring fits of lesser intensity gradually lead up to the state recognized as chronic gout. If the first fit occurs late in life, there may be long intervals. The latter may also occur in early life; but where the first attacks are developed before forty years of age, the disease is very apt to recur soon, and to manifest an atonic form with various tissue-degenerations. Such examples are significant of strong hereditary tendency or of feeble constitution, or, indeed, of both.

The influence of the disease is in direct relation to the general vigour of nutrition and the force of tissue-resistance. Hence, old persons may be met with of originally powerful constitutions who may have been gouty for many years, and yet have resisted the degenerations so commonly associated with the disease, and, in particular, show no signs of uratic deposit at any point. They are, in fact, little the worse for the gout, and reach a ripe age. In persons of less vigour, the same gravity of the disease will suffice to induce decided gouty cachexia. Good constitutions may, however, be ruined and yield to prolonged excesses and indulgences, whence, even in early life, such persons are no better than the heritors of an originally frail habit of body.

In chronic gout the local processes linger and fail to leave the joints. Effusions, enlargements, stiffness, and crippling gradually supervene. Slight paroxysms are readily provoked, and add little to the abiding discomfort. Several joints become thus implicated, and the patient grows more and more infirm. The gouty habit is pronounced, and manifested by many untoward symptoms. One ailment sets in after another, now of the digestive organs, now of the heart, or of the kidneys. Thus come into prominence many of the troubles recognized as incomplete gout. As has been said, "The patient now no longer has the gout, but the gout has the patient." The natural look of health departs, and the face becomes sallow or pasty, the circulation is feeble, and a general aging is manifest. Serous circulation is slow, whence tendency to slight œdema of eyelids and extremities, although the kidneys may not yet be seriously involved. The tendency to various discomforts and pains indicates a disposition towards wandering or retrocedent gout, and when these rapidly change their place, the term "flying gout" is sometimes applied.[1]

[1] As pointed out by my friend Dr. Wynne Foot, of Dublin, the term "flying gout" appears to have been first used by Mrs. Hunter, wife of John Hunter, in a

In treating of chronic gout, I shall describe two main varieties, which deserve to be treated separately, because they fairly represent two clinical types. I take first, therefore, (*A*) the tophaceous variety, and (*B*) the deforming variety. Both forms may, and do, occur together, but in practice the two may best be separately regarded.

A. **Tophaceous Variety.**—It is in the chronic form of the disease that tophi are most apt to be formed in various situations.[1] This "tartar of the blood" or "gravel of the skin" mostly appears in the vicinity of affected joints or in the integument of the ears. It is presumably present in the encrusting cartilages of the joints, or in their synovia or ligaments. In the latter case it may be felt if not seen, and in extreme degrees of this deposition, hardly any part of the integument may escape it. The nature of this is now fully understood and recognized by appropriate tests. The only morbid appearances at all resembling tophi are small sebaceous tumours, such as milium, found on the face near the eyelids, or larger ones which may occur on the neck, chest, upper arms, or scrotum. Deposits on the eyelids may somewhat simulate patches of xanthoma. The microscope will clear up any dubiety, or failing this, the employment of the murexide test.[2]

The laity never fail to recognize this form of the disease, which they term "chalky gout," and if history be given of ancestors or

letter to Dr. Edward Jenner about her husband, dated Bath, September 13, 1785. She mentions that "he has been tormented with a flying gout since last March."—*Works of John Hunter*, edited by James F. Palmer, vol. i. p. 96, 1835.

It is of interest to note that this great man was a sufferer from gouty ailments. At the age of forty-six he had attacks of spasm in the region of the pylorus, and of failure of cardiac action. At forty-nine he had severe vertigo for ten days, being unable to raise his head from his pillow. Jenner, who saw him at Bath, made the diagnosis of angina pectoris, and wrote his opinion to Heberden.* At fifty-seven he had slight symptoms of regular gout, which were followed by irregular spasms of the face, arm, stomach, and heart—"flying gout." At sixty-one he had attacks of amnesia, lasting half an hour, coming on suddenly. Afterwards he was subject to angina pectoris on exertion or emotion. He was always irascible and explosive in temper. His death was, as is well known, sudden, in an anginal paroxysm, provoked by deep emotion, at the age of sixty-seven. His aorta, coronary, carotid, and cerebral arteries were calcified, aortic and mitral valves thickened, heart-structure "pale and loose." With the knowledge of to-day, it is easy to read the pathological lessons of his case. He was a temperate man.

[1] "Abeuns in cretam, calcemve."—*Boerhaave's Aphorisms*, 1261.

[2] "Callosities also form in the joints: at first they resemble abscesses, but afterwards they get more condensed, and the humour being condensed, is difficult to dissolve; at last they are converted into hard white tophi (πῶροι στερροὶ λευκοὶ), and over the whole there are small tumours like vari and larger, but the humour is thick, white, and like hailstones."—*Aretæus*, Περὶ Ἀρθρίτιδος (second century A.D.).

* He, however, withheld this letter.

relatives thus affected, there need be no hesitation in pronouncing for true gout amongst them, and for the probable gouty nature of such arthritic ailments as may be complained of by the patients under examination.

With the presence of tophaceous deposit is associated a peculiar soft and satin-like condition of the skin. The integument of a labouring man may, thus, come to resemble in texture that of a delicately-nurtured woman, the skin being very smooth and glossy. In the vicinity of tophi there is usually a dusky pink or red colour of integument, through which subjacent uratic deposits may glisten, a little pressure being sufficient to make the latter plainly visible, even where they are situated somewhat deeply.[1]

In chronic gout the skin of the limbs may become very soft without the occurrence of tophi, or when these exist only in the ears. In the latter situation tophi may sometimes be well-demonstrated by holding a light behind the auricle, when deposits are disclosed as black spots, which may be hardly recognizable by reflected light. When large accumulations of urates have been formed around joints, the integuments become so stretched and attenuated that they give way, and afford exit to a creamy pultaceous fluid, consisting almost wholly of densely-packed crystals of sodium urate, but also of calcic urate and phosphate, sodium chloride, and animal matter. Masses of urates may be discharged from time to time, and after a free evacuation there may be a lull, the broken surface presenting the aspect of a flabby or indolently granulating ulcer, with serous or slightly opalescent fluid exuding from it. Deep in the ulcer may be seen other more solid deposits in course of breaking down or of extrusion. As a result of such discharge, a joint may come to assume a more shapely appearance, and the ulcer heals up. Meantime, other deposits may break down elsewhere in similar fashion. The size attained by these masses is sometimes very great. In Fig. 16, p. 84, I have depicted the most extreme instance that I have met with. Some of the fingers were as large as turkey's eggs.

[1] Concerning this variety of gout John Hunter wrote as follows:—"Chalk is not necessarily an effect of the gouty inflammation, for in a gouty habit we have chalk formed where there never had been any gouty inflammation. . . . The chalk shall remain for years without producing inflammation, and seldom produces it at all, but from quantity. And when the interior surfaces are exposed, they hardly take on common inflammation and suppuration, healing more readily than a sore of the same magnitude from any other cause; even a joint shall be exposed, yet common inflammation shall not come on, nor shall it suppurate, only a watery fluid shall come out, bringing with it the chalk occasionally, and it shall heal up kindly."—*Op. cit.*, p. 268.

In Fig. 21 is depicted a less grave form of tophaceous arthritis, which, however, induced much crippling of the fingers.

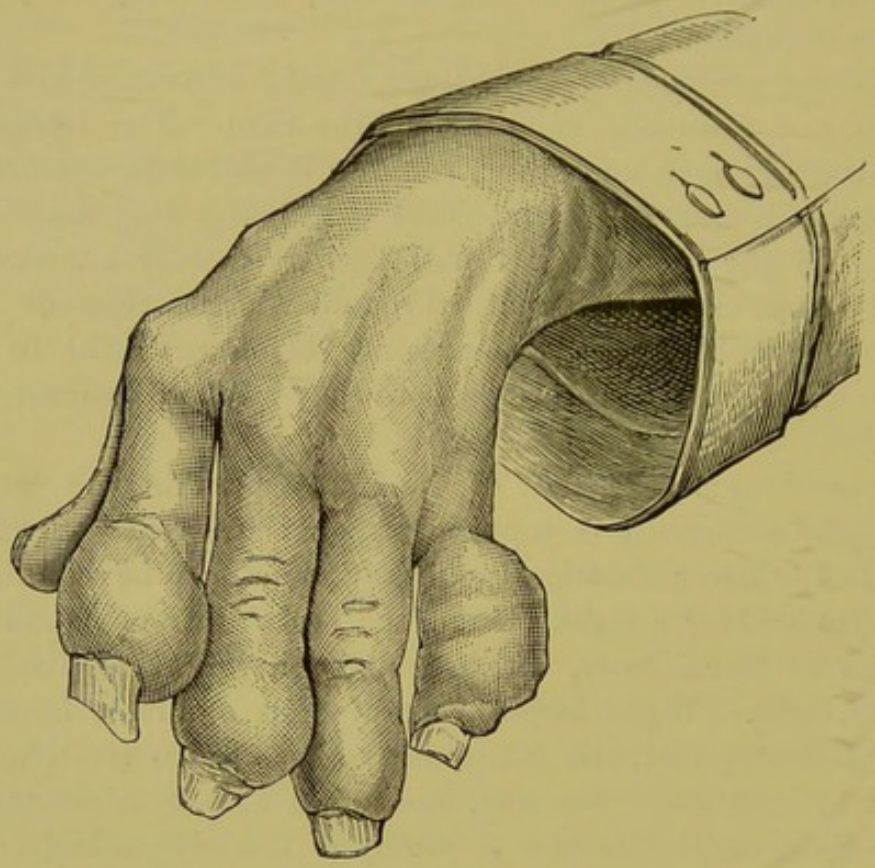


Fig. 21.

Uratic deposits are not, however, always painless during their formation. After acute attacks of gout have passed off, there may follow renewed pain in the neighbourhood of the joint, and later there is discovered a nodular or a soft swelling. In the latter case there may be fluctuation, indicating a liquid collection of urates. This should never be opened. In a few weeks this tumour tends to indurate and grow more compact, and a so-called "chalky" concretion is established. With renewed accessions of gouty attacks fresh deposits may be laid down. When these are small and flattened, they do not tend to cause ulceration, and remain as streaks or plates scattered in the integuments.

Deposits in the vicinity of joints may disappear during renewed attacks of arthritis, and others form elsewhere around the joint. Bursæ over joints are common sites for deposit. That over the olecranon may become the site of a large fluctuating tumour full of synovial fluid impregnated with urates, which may in time become firm and compact. Those over the fingers and the patellæ are frequently involved. Abarticular tophi are not, as a rule, accompanied by any painful process. I have seen many cases

illustrating this fact, of which the commonest example is that supplied by deposits in the ears.[1] The following was a very remarkable instance :—

J. W., æt. fifty-eight, a retired lighterman, robust-looking and of large frame, came to the Hospital to my colleague, Mr. Butlin, on account of numerous small tumours of the scrotum. There were about five-and-twenty of them, situated chiefly at the sides of the purse, the skin of which was melasmic as seen in advanced Addison's disease. In colour the tumours contrasted markedly with the dark integument, being bright pink for the most part, with, in some instances, whitish specks on their surface. They somewhat resembled a large crop of sebaceous mollusca, but had no umbilicated hilum. They proved to be tophaceous, crystals of sodium urate being found in the milky pap which was forced through a puncture made in one of them. Fatty matters and cholesterine crystals were also found. The murexide test was also satisfactorily obtained.

The history was, that these tumours had begun to form at the age of nineteen, and had since grown, some having discharged and disappeared. Attacks of frank gout had been frequent, not very painful, beginning in the right great-toe, then in the left, forty or fifty such fits having been experienced. The knees had suffered, and the metacarpo-phalangeal and phalangeal joints of the right fore and middle fingers. *No distortions existed in any joints, and no tophi were found elsewhere than on the scrotum.* There was optic neuritis in the right eye, and a large detachment of the retina at lower part. In the left eye were patches of choroidal atrophy with myopic crescent.

This man had drunk all kinds of liquors, but not, according to his account, very intemperately. He knew of no history of gout in his family. His mother, he believed, had had rheumatic fever. A coloured drawing was made for the Museum.

It is not easy to account for the occurrence of so many tophi solely in a part for which gout has no special predilection, and which is commonly endowed with a vigorous circulation. I showed this patient at the Clinical Society, in February 1889. It was suggested by some members that these growths had originally been of the nature of molluscum fibrosum. Such a case well-illustrates the fact that tophaceous gout runs a different course from the more painful deforming variety of the disease. Tophi sometimes precede by some years, as in the foregoing case, the development of gouty attacks in joints. The same is true, also, of auricular tophi. In persons suffering from frail health (gouty cachexia), abscesses may form around the deposits, and give rise to discharge of pus and urates, and whenever ulceration and flow of tophaceous matter occurs, it is rare to meet with paroxysmal attacks anywhere in the body. With the cessation of the discharge renewed fits may supervene. Cases of extreme tophaceous deposit may be met with in persons who have always abstained from alcohol. Sir William Gull has

[1] Sometimes auricular tophi are a little painful at an early stage.

told me of one such case, which occurred in the son of a very gouty father who also was a total abstainer from alcohol.

Tophaceous gout is commonly seen in the male sex, but well-marked examples have come under my notice in women. Traumatism sometimes determines the localization of a deposit, but most instances cannot be so traced.

As pointed out by Garrod, the rule commonly holds good that where extraordinary tophaceous deposition prevails, the kidneys may be regarded as unsound and in progress of contraction. Such cases are more correctly included in the category of gouty cachexia, under which head I shall more particularly refer to them. It is, however, not very uncommon to meet with tophi in the ears of patients who enjoy fairly robust health, and are capable of mental and bodily activities. It is not within my experience that any morbid growths in gouty subjects, such as innocent tumours or scars, ever become infiltrated with urates. When I exhibited the patient with scrotal tophi at the Clinical Society, several members reported that they had seen similar cases, and the question was raised as to these having been originally molluscous tumours which became subsequently infiltrated. If this was really the case, the occurrence must be of extreme rarity.

B. **Chronic Deforming Gout** (*Arthritis deformans uratica*).—With, or without, much uratic deposit may occur various deformities in the affected joints in chronic gout. Many of these I have already described in the chapter on the morbid anatomy of articular gout, and they have long afforded matter for discussion respecting the rheumatic element which some authorities allege to be mixed with gout in such instances. I believe that, for the majority of articular deformities and distortions met with in uratic arthritis, gout is solely responsible, and that many of the changes thus wrought are similar to, but not the same as, those induced by rheumatic disease. The evidence as to *causation* is not afforded by a study alone of the *results* of either disease.

The most obvious changes relate to enlargement and distortion of the component structures of the affected joints. The degree in which slowly progressive ostitis, chondritis, and induration of synovial membrane occur is dependent not only on the severity of the irritant gouty process, but also on the textural peculiarities and special vulnerability of the individual affected. Hence, the explanation of the apparent paradox of slight or no articular deformity in cases of repeated and violent paroxysms, and the gross deforming changes sometimes met with after a few and less intense attacks of local gouty disease. These varying degrees

and results of arthritis are not more remarkable than the occurrence of the tophaceous form of gout already treated of, in which there must also exist some personal factor or idiosyncrasy.

The fingers, hands, and wrists show various deformities, depending on overgrowth of articulating ends of bone, cartilage, ligaments, and bursæ. These may be complicated with visible or invisible tophaceous deposits. Partial dislocations of phalanges may occur, and deflections of these in various directions. A common site, even in slightly pronounced cases, is the metacarpo-phalangeal joint of the first finger. As I have mentioned previously, ankylosis may occur, both true and false, the former being quite peculiar to gouty arthritis. Superjacent bursæ tend to become large and loose, or may contain uratic deposits, and the latter may occur in nodular form in the pulps of the fingers and thumbs. The affected joints are apt to crack audibly on movement, and a crunching sensation is imparted to the hand if placed over a large one, such as the knee. This sign is, however, not peculiar to chronic gouty arthritis. It is very rare for the deformities of true gout to attain the gross characters peculiar to chronic rheumatic arthritis; they are altogether of lesser degree in the majority of the worst instances. Hydrarthrosis is less commonly due to gout than to rheumatism. Some degree of synovial effusion is very often met with. Gouty arthritis will, however, provoke exostoses, which are due rather to irritative ostitis than to proliferation of encrusting cartilage.

The patellæ may enlarge considerably and lose their sharp edges; their cartilages are rarely found intact, but are eroded, cracked, and often encrusted with urates. The sufferers sometimes complain more of helplessness than of painfulness in chronic gout of the knee, the leg having a tendency to yield and give way suddenly on exertion.

In chronic gout affecting the feet, there are both pain and weakness. The ankle is a site for long-abiding gout, and so, too, are the component joints of the tarsus. Repeated subacute attacks are very prone to seize upon various parts of the feet, as on the outer or inner aspects of the tarsus, the heel, tendo Achillis, and the plantar fascia. Painful states of single tarsal bones occur with nodular swelling. Such patients are sorely crippled and hampered in their efforts at locomotion. Varied deflections of the toes occur, as in the fingers, but the tendency is for distortion outwards of each great-toe, sometimes to an extreme degree, whereby the two component phalanges quite overlap the other digits. This is a permanent and irremediable

deformity. I have already described the formation of gouty Heberden's nodes, which may be seen in both sexes, but with greater frequency in women; also the tuberous or knotty state of the small joints in the latter sex, which afford, as I believe, strong indications of a gouty habit of body. With these are found various deflections of the last phalanges, especially of the little, ring, and fore-fingers. Both the nodes and distortions in such cases belong rather to the category of incomplete than of chronic gout, since they are rarely attended by any regular manifestations or sharp paroxysms, but are associated rather with hemicrania, and various vascular and nervous symptoms, and sometimes with glycosuria.

Deformities such as I have described may be borne for many years in persons of originally vigorous constitution, and advanced age may be reached in spite of severe crippling.

With the establishment of chronic gout in either its tophaceous or deforming varieties, it is rare to find the kidneys in a healthy or adequate condition. In the earlier paroxysms these organs may be little, if at all, involved. The patients may notice occasional uratic deposits, but it is a common experience that, as the gouty fits lessen in intensity and frequency, the urine becomes less apt to be loaded, and is observed to be more plentiful and clear. This symptom may be noted by the patient, and is not seldom regarded by him as a satisfactory indication of better general health and of less goutiness. The observant physician will form a different opinion on this fact, and take note of the condition of the urine as to its quantity, specific gravity, and the percentage of urea and uric acid contained in it. In particular, he will ascertain if albumen be present in even small amount. Albuminuria has been observed to set in within a year or two of the first overt gouty attacks. In such cases it is only too common to find that with some polyuria there is low specific gravity, 1006 to 1015, a deficient amount of urea and uric acid, and a small quantity of albumen. The latter may be absent for long periods, but is usually fleeting if in small amount. These qualities betoken the onset of interstitial nephritis, with some degree of tubal catarrh, and indicate one of the gravest complications or phases of chronic gout. It is then certain that one variety of visceral gout has supervened, and the general disease is thus rendered more grave and less amenable to treatment. This phase is more apt to appear in cases of tophaceous gout, and some ratio is established between the degree of renal inadequacy and the amount of uratic deposit laid down in or around the joints. Microscopic examina-

tion of the urinary sediment reveals in these cases a few casts of the tubules, epithelial or granular. These are not constantly detectible, but may be found at intervals, and especially during subacute articular attacks. Unless tubal catarrh is present to a considerable extent, no large amount of albumen is met with in the urine in cases of chronic gout. The morbid appearances in the kidney have already been described in Chapter iv., p. 99. I do not believe that uratic deposits in the tubules, or outside them, as found sometimes in the pyramids, are, as is often affirmed, an important cause of albuminuria. Intimately connected with, and indeed dependent on, the progressive renal changes are certain cardio-vascular alterations which have in recent times received much attention and close study from many good observers. I have given an account of these so far as their morbid anatomy is concerned. The associated symptomatology of these changes may be noted here. Indications are afforded of the hypertrophy of the left ventricle of the heart by the ordinary physical signs of forcible impulse, displaced and diffused apex-beat, and by the noisy quality of the first cardiac sound. This is often replaced by a reduplicated first sound, heard at the apex, over the septum of the ventricles and at the base. The aortic second sound is apt to be loud or accentuated. The systemic arteries become hardened, and, where superficial, visible, tortuous, and possibly atheromatous. The pulse is of high tension, full between the beats, and firm—*pulsus durus*. This is commonly an abiding condition, but the tension may vary, and the pulse become relaxed or compressible in response to several conditions.

Such symptoms differ in no way from those commonly associated with interstitial nephritis, however induced. Hence, they are not peculiar to the gouty, though so often met with in such persons. The longer this condition persists, the greater the risk for the patient, since he becomes liable to the several untoward accidents of arterial sclerosis with high arterial blood-pressure,—to wit, hæmorrhages in vital or essential organs, and especially in the brain or retina; and, in truth, the condition tends to progress *pari passu* with the advance of renal contraction.

Todd related a case where albuminuria occurred two years after a primary attack of gout, and death ensued two years later after uræmic eclampsia and coma.

In chronic gout there is always defective excretion of uric acid by the kidneys, and an excess of this acid in the blood.[1]

[1] In respect of the intimate pathology of cases of cardio-vascular disease in relation to interstitial nephritis, a new view has been taken by Dr. Da Costa of Phila-

Pulmonary emphysema is another textural degeneration, which tends to proceed with much risk and suffering to the gouty, adding to the difficulties of the circulation, and aggravating the cardiac trouble, already existing, by causing dilatation and leather-like induration of the right ventricle.

Gouty Cachexia.

This condition supervenes gradually in cases of chronic gout, and represents the final stage of the various evils wrought by this malady on the constitution. Gout occurring at an early age, and, therefore, especially significant of strong hereditary tendency, may within a few years induce this state, and, so, men in the prime of life may be the subject of it. Women under fifty years of age very rarely afford examples of it. I have met with at least one instance in a woman aged forty.

Regular gout in robust men who have not abused their health by excesses of any kind seldom passes on to the stage of cachexia. The case is very different when the disease is implanted or induced in persons of originally feeble constitution. In such instances the gouty processes are atonic, and indicate a generally asthenic condition. The health fails, the blood becomes impoverished, the circulation flags, signs of early senility supervene with widespread textural degenerations, and the kidneys become rapidly inadequate for their functions.

Gouty cachexia may form the last phase of either tophaceous or deforming gout. The cases of extreme "chalky" gout are also examples of gouty cachexia. Subacute attacks in the joints may occur from time to time, tending to linger long in the parts,

delphia.* He and Dr. Longstreth have carefully examined the nervous ganglia of the renal plexus and the cervical ganglia giving origin to the cardiac nerves in cases of this nature. They found well-marked changes, showing increased fibrous tissue and atrophy of the ganglionic cells. Dr. Saundby has also noted this alteration in the renal ganglia. Dr. Da Costa asks, "Is it going too far to assume that these changes are an integral part of the disease, and in the case of the cardiac ganglia determine the hypertrophy? . . . What starts the change? What is the cause of the degeneration? Is it not fair to look beside the blood, to a cause so predominant as the nervous influence, which is everywhere? What the ultimate cause of the lesion is cannot be stated, nor need we assume that one cause alone will determine it. It may be gout, it may be lithæmia, it may be rheumatism, it may be lead, it may be purely perverted nervous function from worry, from strain, from anxiety." He concludes, that, "the cardiac hypertrophy in Bright's disease is not in any sense the consequence of that disease, but an integral part of the same general morbid process." This is the view of Gull and Sutton from another stand-point.

* The Middleton-Goldsmith Lecture, April 1888. Med. News, May 1888.

and to subside imperfectly, leaving œdema and tenderness behind them. Slight provocations suffice to induce renewed attacks, trivial injuries and exposure to cold being often determinant of such. The patient is crippled, and unable to take any kind of exercise beyond that of conveyance in some vehicle. He is a confirmed invalid. Irregular forms of gout may be also induced by slight causes in various parts of the body.

The renal inadequacy is often very manifest, and sets up dropsy with minor, or even grave, uræmic symptoms. Pulmonary œdema, bronchitis, the many troubles inseparable from cardiac failure, dilative hypertrophy of both ventricles, hepatic engorgement, gastric catarrh, and diarrhœa may all gradually supervene, and portend a fatal issue. These conditions may be successfully met again and again by treatment, but surely wear out the patient; or death may rapidly be brought about by an attack of pneumonia, especially when it seizes on a lung already emphysematous.

The onset of gouty cachexia in any case is always to be dreaded, and, therefore, to be anticipated early by preventive and other modes of treatment. So long as regular attacks occur at long intervals, there is little fear of drifting into the cachectic stage. The tendency varies exactly with the intensity of the gouty habit, the general management of the case, and the fortitude of the patient in respect of self-control. The wilful libertine, if gouty, is likely soon to become cachectic, while the prudent man may altogether avoid this state, or avert its evils for many years or decades of years.

The anatomical basis of gouty cachexia is represented by widely spread tissue-degenerations, of which I have already treated. Arterial sclerosis, fatty changes with fibrosis in various viscera, interstitial nephritis, degeneration of cardiac walls, pulmonary emphysema, and catarrhal states of mucous surfaces generally, are the essential pathological lesions. It is readily intelligible that the symptoms arising from these conditions must be manifold and infinitely varied, some appearing soon, and others later. I will enumerate the most characteristic of these. They may not all be met with in any one case.

Anæmia is apt to supervene in chronic gout, though forming no part of the disease in its acute forms. Some degree of dyspnœa is common, being variously induced by renal, cardiac, or pulmonary degenerations, or by a combination of all three. The pulse is irritable and easily rendered unduly frequent. There may be palpitation, and various cardiac symptoms dependent on the existing lesions and degree of failure of the heart's walls.

More or less bronchial catarrh is frequent, and œdema of the bases of the lungs. Slight provocations readily induce bronchitis.

The digestion is feeble and painful, flatulency and indications of gastro-enteric catarrh being present. Pharyngeal catarrh may be troublesome, exciting hacking cough, especially in the morning. Diarrhœa may occur from time to time. The condition of the urine is closely dependent on the condition of the kidneys, and presents generally the characters common to chronic sclerosing nephritis. Polyuria may be present for short periods, and remit. Glycosuria in varying degree, or fugitive, may be sometimes noted. Albuminuria also varies in degree, and may remit for long periods.

The cerebral or psychical state is not constant. There may be much nervous irritability and varying degrees of amnesia. The latter is sometimes very marked. Gloomy and melancholic states of mind may prevail, with miserable dejection of spirits. Stupor, loss of consciousness, and a state of catalepsy with vacant stare, have all been noted as temporary accompaniments of gouty cachexia.

Somnolence, especially after meals, may occur, also vertigo, syncopal tendency, tinnitus aurium, and diplopia. Fatuity, hebetude, or muttering delirium, are sometimes met with in the last stages. The complexion may become sallow and dirty yellow, as in chronic nephritis. The hair is apt to whiten prematurely. Ultimately, signs of dropsy, due to cardiac failure for the most part, tend to appear in the lower limbs. A cerebral hæmorrhage may induce hemiplegia, or prove rapidly fatal, or the end may be reached after the onset of bronchitis or pneumonia. Death may also occur from syncope, or from rupture of the left cardiac ventricle, which has become fatty.

Gouty Vascular Cachexia. — Sometimes, the onset of gouty cachexia is manifested by a general feebleness of bodily power, inability for wonted exercise being manifested. In this way, vigorous old men, who have been long more or less gouty, begin to break down. There may be no uratic deposits, but there is often polyuria, at times paroxysmal and nocturnal, and the urine is indicative of granular kidneys. There is reason to believe that some of these failures are largely due to vascular degeneration, which is wide-spread, and thus involves the intimate nutrition of the cerebro-spinal centres. Some bodily wasting is observable, together with muscular and nervous enfeeblement.

Irregular (Incomplete) Gout.

Many terms have been applied to phases of gout which do not manifest themselves in classical fashion in a joint.[1] One more may be added, which will concisely express the relation which these forms—for they are many—bear to the acute and regular fits of the disorder, viz., abarticular. Gout manifesting itself anywhere but in a joint is to be considered irregular or incomplete. Such phases of the disease may be anomalous, but they are very common, and as such are so far regular as to comprehend a number of ailments which pertain chiefly, and in some cases exclusively, to persons of gouty heritage and diathesis.[2]

In discussing these varieties of gouty disease, it is of the utmost importance to seek exactness, and only to include in the category such ailments as may legitimately find a place there. Without doubt many morbid states have often been flippantly or erroneously set down to irregular gout which owned no such designation, and thus a cloak for ignorance has always been at hand to throw over careless observation, ignorance, or wilful misinterpretation of symptoms. As a consequence of such errors, some have come to regard even truly gouty manifestations, when not articular, as actually non-existent, and to deny the dependence of such upon a gouty habit. The latter error is no more to be condoned than the former, and it may be fraught with mischief to the sufferer.

Senator expresses his belief that irregular gout is evolved from the typical form when the latter has existed for many years, and that it is met with chiefly in elderly people.[3] This is without doubt the case in a certain proportion of instances, but this view will not explain the existence of symptoms and numerous ailments which occur in the persons of those who are goutily disposed, who are entitled to gout, and whose bodily disorders are plainly impressed with the gouty type. The minor, incomplete, and less well-marked forms of gouty trouble are precisely those that have so long escaped exact recognition, and which even now do not readily receive this explanation.

Many of the irregular phases of gout are recognized by some

[1] On this subject, "Arthritis Anomala," the masterly treatise of Musgrave, written in 1707, is well worthy of study.

[2] Such epithets are "lurking," "latent," "misplaced," "undeveloped," "atonic," "suppressed," "masked," "imperfect," "incomplete," "asthenic," "vague," "erratic," "anomalous."

[3] Art. in Ziemssen's Cyclopædia.

as results of lithæmia, and in no special relation to gout.[1] But lithæmia or urichæmia is an uric acid disease, and—no lithæmia, no gout.

I have already expressed my belief that in many instances the disorders attributed to lithæmia are truly expressions of incomplete gout. They portend, if they do not ultimately lead up to, true gout. Some persons are only so far gouty as to be lithæmic without manifesting any joint-disturbances. They may never be the subjects of regular gout, or many years may elapse before this event supervenes. Allusion has also been made to many varieties of irregular gout in Chapter viii.

Manifestations of it may be met with in both sexes, women, however, being especially prone to them, more so at, or soon after, the climacteric period. So multiform are these, that the epithet "Protean" has been applied to them.

Much difficulty attends the diagnosis of minor gouty ailments in many cases, because regard is mostly had to some very plain tokens of the disease. Thus, it is common to hear objection made to an opinion as to the truly gouty nature of a case, because no tophaceous deposits can be found, or no history of an attack in a big-toe at some previous period is obtainable. And again it is objected that there can be no gouty element because the patient has earned the right to the ailment neither by heredity nor by his personal habits.

In opposition to doubts of this nature, I am most deeply impressed with the fact that it is just in cases where no marked coarse objective signs of gout exist that we should look for the presence of the minor tokens of the affection, and just in such cases that we commonly find them. Irregular and incomplete gouty symptoms occur both in those who are the victims of regular, exquisite, attacks, and in persons who have never had, and, perchance, may never have, a typical precipitation of regular gouty inflammation. The most marked instances are certainly to be found amongst the latter class, although even in these it is never safe to predict immunity from a regular attack, since such may not supervene till the ninth or tenth climacteric period of life.

A study of the irregular phenomena of gout is of much

[1] Some physicians will not regard any disturbance as truly gouty unless positive demonstration of aberrant relations of uric acid be made, and for them nothing is gout that is not essentially connected with paroxysmal attacks in joints. This is, in truth, but an elementary fact in the whole pathology of gout, the first conception of the malady as defined in simple form for a junior student. Careful clinical study teaches many other facts about the disease and its relationships.

importance, and tends to shed light upon the nature of the disorder, or at least to clear the way for a better knowledge of its laws.

It is to be noted that phases of irregular gout may occur in those who suffer occasionally from regular attacks; but probably the majority of cases are seen in persons who have never had frank gouty arthritis. Hence, it may be affirmed that, for the most part, regular or sthenic attacks are preventive of the irregular or masked phases of the disorder. Some patients become aware of this fact, and endeavour, imprudently, to bring their gout out, or to focus it, as it were, in a regular fashion in some joint. They have learned to dread its insidious actions, and prefer the honest malady in a classical form.

Many of the ailments due to irregular gout are often regarded, both by the practitioner and the patient, as "rheumatic" or "neuralgic," and much confusion has arisen in consequence. The diagnosis is, in truth, often very difficult, demanding nice and painstaking discrimination if we would be accurate and honest. I have already remarked that not every ailment or illness in a truly gouty person is of gouty nature; much less so is this the case in the subjects of gouty tendency which is only slightly impressed upon them.

The true nature of the symptoms is to be ascertained by a consideration of the physiognomy, family and personal life-history of the individual, and by attention to the type of the particular ailment. The crucial test as to the presence of uric-hæmia is seldom practicable in any but hospital patients, so that we must often be content to act in ignorance of this important fact.

Considerations as to family proclivity, personal habits, age and sex, will usually avail materially, if not absolutely, to throw light on the true characters of the symptoms presented; but cases will occur in which the best observers may be misled, and a correct diagnosis only be possible on the onset of some unmistakable gouty manifestation elsewhere. When the latter is not forthcoming, the clue is sometimes at hand by therapeutic tests. A large bedside experience is commonly requisite to unravel some of the strange problems presented by masked gouty processes. This necessarily entails a wider grasp of all forms of morbid phenomena than is possible for him whose mind is mainly directed to the study of one disease, and thereby warped.

A study of the multiform features of irregular gout is of great importance, since they often betoken grave states of ill-health,

leading more surely to death than any number of frank attacks of gout. Hence the dictum of Musgrave[1] should be borne in mind, "Arthritis raro occidit Regularis, raro nisi prius degenerans in Anomalam." Sir Thomas Watson quoted some French author for the following aphorism, which would serve as a fair translation of Musgrave's sentence: "La goutte articulaire est celle dont on est *malade*, et la goutte interne est celle dont on *meurt*."

It is an error to suppose that the irregular manifestations of gout are mostly witnessed in the affluent classes of society. Hospital practice furnishes numerous examples, if they be sought for and detected. Hard-worked men living in towns, whose occupations are mainly sedentary, suffer in considerable proportion, and especially are those affected who use their brains and undergo great mental labour. In such instances there is often found to be a large appetite for food; for brain-work and wear of the nervous textures will, equally with muscular energy, create this; but a limit is placed to sufficient oxydation by reason of the necessary urban and confined life, and the consequent defective aëration. If no relief be afforded, a measure of dyspepsia ensues, usually of catarrhal form, and pains and fulness are complained of in the liver. A sort of cumulative plethora is thus from time to time set up, and it is at such crises that a sudden precipitation of gouty inflammation may be looked for. A regular attack of acute gout may occur, or, if no special depressing cause comes into play, then some minor or irregular token of gout appears.

In the cases of those who lead very uniform and regular lives, these troubles occur if the gouty taint is present; but the proclivity is much aggravated by indiscreet indulgence in food or drink, or by undue exposure to chill and changeable weather.

Sir Prescott Hewett's remarks on some of the irregular manifestations of gout are particularly apt and instructive.[2] He refers to "dyspepsia, more or less troublesome; frequent deposits of lithates; slight eczematous eruptions from time to time; anomalous pains in various muscles; sharp, deep-seated pain in the tongue, existing for two or three days, and then disappearing altogether for a while; crackling about the cervical spine in slight movements,[3] more or less; sometimes a mere suspicion of knottiness about the smaller joints of the fingers." And he adds, "The great difficulty in such an investigation is to get at

[1] *De Arthritide Anomalâ*, Corollaria ii., p. 474. Exon., 1707.

[2] Clin. Soc. Trans., vol. vi., 1873.

[3] Crackling sensation in the upper part of the spine was recognized by Brodie in 1842 as a gouty symptom, and he related having met with several cases. I have also observed it.

a clear recognition of such trifles, for, disappearing as they do for a while, they are forgotten until recalled to the mind."

I believe that many of the anomalous pains above described, in muscular, fibrous, synovial, and articular structures, are truly gouty in their nature, but they are more frequently ascribed to "rheumatic" influences.

Amongst minor signs of incomplete gout are the nodosities of the fingers already described, with or without more marked distortions of the phalanges or entire digits.

I have, on several occasions, had the opportunity of watching the course followed by small gouty formations, resembling a crab's eye, over Heberden's nodes. Garrod refers to these.

In the earliest stage a small eminence was observed, which was slightly tender. It became full of a clear fluid, and was prone to ache and cause a sensation of burning. It enlarged and burst at intervals, emitting a pellucid and sticky fluid. It was solitary, and in no way connected with the last phalangeal joint. On examining the fluid microscopically, I could find no uratic formations, and no chalky matter appeared. The whole subsided for months together, and re-formed exactly as before. These formations can hardly be called tophaceous, inasmuch as no salts of uric acid are deposited. They occur, probably, in small bursal sacs. Sir James Paget has described these growths, and deprecates any surgical interference with them. If let alone, their tendency is to rupture and to subside along with the quiescence of the gouty activity that gave rise to them. Some little thickening of the integument is all that can be found in the intervals.

The gouty nodosities are often red, and are prone to become hot and painful from various causes. Fugitive achings are frequent in them whenever a threatening of fresh attacks supervenes, and they are specially apt to be troublesome after partaking of bad champagne or other gout-inducing wines.

The metacarpo-phalangeal joint of the right thumb is frequently enlarged, and excessive use of this part in writing, or in other manual labour, has possibly much to do with its special liability to suffer.

These deformities of the finger-joints are to be met with in women, who seldom present, in as marked form as males, either the physiognomical features or the tissue-changes of the gouty diathesis. I have met with them before the menopause. They are to be distinguished from true gouty deformities of the fingers in both sexes, both by their tuberous form and by the fact that

they are not always the result of an acute (gouty) arthritis. And they must not be confounded in any case with the grosser changes brought about by rheumatic osteo-arthritis, in which also the axes of the fingers commonly diverge to the ulnar side of the forearm.

It has long been recognized that gouty manifestations in women are different from those observed in men. Distinct gouty inflammation is not usual in women till the "change of life" has occurred. But it is found that gouty headache, migraine, and dysmenorrhœa occur before that period in the subjects of inherited gout, and are indeed sometimes the only expressions of such hereditary tendency.[1] I have observed that menorrhagia, severe headaches, and migraine occur with some frequency in women of gouty parentage. The worst case of epistaxis in a young woman that I ever witnessed was in the daughter of an exceedingly gouty man. Without doubt, all these ailments in such persons yield more distinctly to anti-gouty treatment than to any other mode of medication.

Plantar gout is an irregular form of the disorder, and has not been hitherto particularly described. It is very agonizing, and may attack both soles simultaneously, hard swellings being formed gradually in the fasciæ. It begins subacutely, and is apt to linger for many weeks. Achings in the insteps are common symptoms, with a sensation as if a tight boot were worn. No redness is seen, but some painful induration may be found over a tarsal bone, which subsides very gradually, together with the associated painfulness.

Deep-seated pain in the heel has been recognized as of gouty origin. The sensation is compared to the feeling of a foreign body being implanted there, such as a bullet. And it is noteworthy that this is sometimes a symptom of a renal calculus, which may itself be the outcome of gouty taint. The pain is sometimes distinctly in the tendo Achillis. The coccyx is also the seat of gouty pain. I feel sure that a dull aching pain in the ensiform cartilage is sometimes of this nature. The tenderness may be extreme; it comes and goes somewhat suddenly, and some degree of hepatic discomfort is mostly associated with it. Pain is especially felt on stooping.

Amongst the irregular forms of gout which occur in other than articular structures are the aching and boring pains in certain muscles and muscular groups. The adductors of the thigh and the gastrocnemii are apparently especially liable to

[1] Trousseau.

suffer. Cramps have long been rightly ascribed to gouty influence in many cases. I have long believed that the cramps which accompany the cachexia associated with granular disease of the kidney were related to gouty taint in certain instances. I first learned to inquire for this symptom from reading Dr. George Johnson's lectures on the subject of granular kidney.

Wandering pains have been noted in various parts, and in the subjects of gout which does not regularly develop itself some fresh ache or disagreeable symptom crops up almost every week; and thus patients complain of gout "flying about," and they do not know where a fresh precipitation or petty outbreak may occur.

Patients thus affected commonly know by their general sensations on awaking in the morning whether they are likely to suffer from any special manifestation during the day. Such manifestations occur very frequently during the night, and are first discovered early in the morning. Sometimes there are observed fleeting pains, twinges, in one or more joints of the fingers and toes.

So-called muscular rheumatism appears to occur with marked frequency in persons of gouty tendency. In many of these cases I have observed that, as happens so commonly in gouty affections, the pains are only discovered in the night, or on the morning following an exposure to damp which occurred, perhaps, many hours before on the previous day. The patient retires to bed in perfect comfort, and awakes in the early morning to find himself racked with the characteristic torture.

Lumbar pain in the mornings, passing off in an hour or two, is occasionally noted without any obvious renal disturbance. Uratic deposits in lymph-spaces may be the exciting cause of some of the pains just described.

A frequently recurring minor trouble in some goutily disposed persons is a painful follicular inflammation in the ala of the nose. An indolent furuncle forms, which does not proceed to suppurate. It lasts for a few days and then resolves. The pain is considerable and annoying. The inflammation returns again and again in the same spot, or very near it. Allusion will subsequently be made to the flushings of the face and of the nose observed in some gouty persons. Gradual thickening of the integuments of the end of the nose is a well-recognized change in the subjects of chronic gout.

Tinglings in the hands and feet are often complained of by gouty persons, the sensation being described as "pins and needles." Women about the time of the menopause are sub-

ject to this.[1] Treatment for imperfectly developed gout is often effectual to remove these. There may be in these cases some degree of perineuritis, excited by uratic stasis.

Episcleritis is sometimes noted, and may become persistent without causing much annoyance. Other troubles affecting the eyes in gouty persons have been noted previously. Iritis may occur. To determine its significance, regard must be had to the personal and family history as to true gout, since rheumatism may be, perhaps, the excitant constitutional cause. The goutily disposed seem especially obnoxious to the poison of gonorrhœa, and are more than others apt to suffer from sclerotitis. Conjunctival hæmorrhages may occur spontaneously in the gouty.

Irregular gout, as involving the respiratory system, has been described in Chapter iv., where I have treated of bronchitis and pneumonia. Asthma, as dependent on gout, is discussed in Chapter x. p. 217. I shall have again to refer to these disorders in treating of retrocedent gout. Gouty tonsillitis, commonly one-sided, is met with, and is apt to be very painful. Catarrh of the fauces and pharynx is apt to become chronic, and to excite severe hacking cough. Stokes noted the sighing respiration of undeveloped gout.

Irregular gouty manifestations of the alimentary system have also been referred to. Deep-seated pains in the tongue are to be noted, lasting from a few hours to two or three days.

Xerostomia.—I saw on several occasions a widow lady, over sixty, in whom extreme dryness of the tongue and mouth, lasting for months, proved one amongst many plain indications of a gouty habit. True gout came out in the great-toe some years later. Dr. Hadden's patient with dry mouth, whom I saw at the Clinical Society's meeting, appeared to me to afford an example of incomplete gouty habit. She had had shingles and facial erysipelas.[2] A granular condition of the pharynx is very common, causing chronic gouty sore-throat, with difficult expectoration of tough, greyish, pearly mucus. In such cases snoring is very frequently observed during sleep, sometimes of great intensity, and aggravat-

[1] "It is probable that the decussations of the sensory nerves of the hands and feet are high up within the cranium, and not in the cord, as is the case with those of the upper arms and thighs; for, the former, being tactile and executive instruments, must have both their special motor and sensory centres within the encephalon. Thus, numbness as well as motor palsy beginning in both hands or in both feet is a sign of intracranial disease. In like manner symmetrical gout of the hands, and symmetrical skin-affections like purpura and psoriasis palmaris, are associated with *trophic nervous debility of centric origin*. The hot palms in fevers and in various neuroses belong also to this class of symptoms."—*Laycock*.

[2] Clin. Soc. Trans., vol. xxi., 1888, p. 176.

ing the congested state of the fauces. In such instances it is not due to dorsal decubitus only.

Œsophagismus.—Spasmodic condition of the œsophagus has been distinctly proved to occur as an irregular form of gout. Brinton first described this,[1] and Garrod mentions an instance of it. Dr. Moorhead, of Weymouth, recorded a well-marked example in a man over sixty years of age.[2] I have met with one or two examples. Gouty œsophagismus may prove very severe, and was so far rebellious in one case which I conceive was probably of this nature, recorded by Mr. Henry Power, as to prove fatal.[3] *Post-mortem* evidence was negative as to any structural disease in the gullet or stomach, or, indeed, in any part of the body. Painful and spasmodic dysphagia may be due to a gouty state of the root of the tongue and pharynx, which condition I once met with in a retired army-surgeon. There was severe pain and spasm in the left side of the pharynx in this case, and the attacks occurred at intervals for two months. Du Hahn related a case of this kind, in which relief came with onset of gout in the hand.[4] Hiccup has been noted by W. Gairdner.[5]

Much discussion has arisen on the subject of gout in the stomach and intestinal tract. Some authors deny the occurrence of such disorders. I am convinced of their existence, and shall discuss them under the head of visceral gout. Here, it will suffice to mention, as irregular gouty manifestations in these organs, dyspepsia with anorexia, nausea, vomiting, heartburn, flatulency, and gastrodynia. Pain is rarely met with unattended with pyrosis, acidity, or flatulency. The bowels are irregular; sometimes costiveness prevails, at others severe diarrhœa may occur.[6] By metastasis, as will be shown, the stomach may be gravely involved in gouty inflammation.

All the disorders just enumerated may be scattered by the onset of an acute articular attack.

A severe form of colic (colica arthritica) is sometimes induced as a form of irregular gout, which I shall discuss under the head of Visceral Gout. The belly is often flatulent before a gouty paroxysm, and the bowels hard to purge. Hæmorrhoids may occur in association with constipation and with portal venous con-

[1] Lancet, January 6, 1866, p. 2. [2] Lancet, July 23, 1881, p. 164.

[3] Lancet, March 10, 1866.

[4] Quoted by van Swieten in his Commentaries on Boerhaave's Aphorisms, vol. xiii. p. 60, 1765. [5] *Op. cit.*, p. 68.

[6] Todd described cases in which a considerable discharge of mucus coloured with bile occurred for two or three days as a variety of gouty exacerbation which afforded great relief.

gestion, which are frequent in gouty subjects. They are often hereditary. Pruritus ani is another allied symptom. Varix in the legs is not uncommon, and may be met with in obese gouty subjects. Enlargement of veins is noticed in connection with acute gouty attacks in any part. Severe anæmia may be caused by bleeding from piles, and in this condition acute attacks of gout may supervene, of which I have seen several examples. The urinary system suffers markedly from irregular manifestations of gout. The special implication of the kidneys has been already discussed at length, and the tendency for progressive interstitial nephritis to set in as gout becomes chronic, is now fully recognized. In the present connection I would allude to the frequent occurrence of nephritic complaints in the form of gravel, renal colic, and vesical calculi. These fits of gravel and colic may alternate with articular gout, or may appear in members of gouty families who do not present as yet, or may never present, signs of active gout in their joints. The connection is, however, very intimate between these two states, and may affect both sexes. Renal calculi may form in persons long before any articular gout supervenes. I by no means infer from this fact that all subjects of renal concretions are indisputably gouty. This is certainly not the case.

As already noted, gout may fall on the mucous membrane of the bladder, causing recurring hæmorrhage or severe cystitis.[1] By metastasis, eczema of the skin may pass to the bladder and suddenly induce cystitis. In elderly men prostatic enlargement may supervene, and add to the difficulties of the inflamed bladder. Very free hæmorrhage may occur from the bladder in irregular gout. The urethra may be the seat of gouty inflammation, simulating very closely an ordinary gonorrhœa, with scalding pain and purulent discharge. Gouty orchitis will be referred to subsequently. I suspect that ovaritis may occur as a result of irregular gout. I have no experience of it, and gynæcologists are not in accord as to its dependence on this state. If the disorders of the uterus and its appendages were studied more particularly with reference to diathetic conditions, I am of opinion that some new chapters in their pathology might be written. At the present time, this large subject is, perhaps, regarded in too mechanical and surgical an aspect, and, owing to the prevailing tendency to specialization in practice, the general physician is not brought much in contact with it. I believe that expression of the gouty habit may be sometimes met with in women in the

[1] *Vide* Clin. Lect. Urinary Diseases, R. B. Todd, 1857, cases related, p. 357.

form of uterine congestions with metrorrhagia, dysmenorrhœa, and leucorrhœa,[1] and Dr. Priestley gives me his experience to the effect, that women of gouty heritage are more apt than others to suffer from chronic metritis, chronic capsular and interstitial ovaritis, and menorrhagia. Dysmenorrhœa and amenorrhœa due to plethoric states are also recognized in this connection. In all such cases great benefit is derived from anti-gouty medication, and especially from drugs which promote a free circulation through the portal vein.[2] Elderly males, the subject of irregular gout, are sometimes much troubled by priapism, occurring in the night. In these cases there is usually found an ill-expressed gouty condition with acid urine. There may be no erotic feelings.

Glycosuria, as a frequently associated state, has already been described.

The liver presents symptoms of disturbance in irregular gout. Biliary lithiasis with colic is not uncommon, especially in women of gouty families. Catarrh of the bile-ducts has been observed. Murchison related two cases in gouty men, under forty years of age, who had vomiting, wasting, jaundice,[3] and hepatic enlargement from this cause. The cases simulated cancer, but subsided under treatment. Occasional engorgements of the liver, followed by diarrhœa, have been noted in gouty persons.

In respect of the cutaneous system, any of the various skin-diseases described in Chapter xvi. may be met with—eczema, psoriasis, urticaria, and pruritus being the most frequent manifestations.

The circulatory system is markedly involved, and, thus, may occur palpitations, cardiac and arterial, irregular pulsations, syncope, and pseudo-angina pectoris. Arterial spasm in various parts, such as "dead fingers," or flushings, may be met with.

The nervous system is variously, and sometimes profoundly, influenced by the gouty state. Many morbid conditions thus arise, and have been discussed in Chapter x.

Varieties of headache, hemicrania, neuralgia, neuritis, vertigo, tinnitus aurium, and vague, sometimes fugitive, pains may be met with.

Gouty Psycopathia.—The psychical conditions are of infinite variety. Irascibility, "touchiness" of temper, capriciousness, morbid forebodings, miserable mental introspection, hypochondriasis, neuromimesis, melancholia, and even suicidal tendency,

[1] *Vide* Art. "*Goutte*," H. Rendu, *Dict. Encyclop. des Sciences Méd.*, p. 129.

[2] *Vide* Inflam. of Uterus, Reynolds' Syst. Med., vol. v. p. 736.

[3] *Op. cit.*, p. 156.

have all been carefully noted as expressions of irregular gouty states of the system (melancholia arthritica of Musgrave). A fit of violent rage or passion may be a solitary expression of, or possibly a substitution for, an attack of regular gout; so, too, some transient mental derangement.

The mental element in persons of gouty habit is a very noteworthy factor, and a due consideration of it is of high importance for successful treatment of many cases.[1] Mental energy may never safely run in excess of other bodily forces, and many persons become gouty because the former outruns the latter. This is often exemplified in the cases of men who are placed by various callings under conditions of life in which they are ill-fitted to act and develop their peculiar energies. The man who is adapted by his muscularity and vigorous circulation for an active outdoor life is unfavourably circumstanced, if he must needs support himself by sedentary occupation amidst town-surroundings. Defect of aëration and insufficient muscular energy will tend to induce a gouty state, and no less will the tendency be maintained by undue mental energy, which now becomes the channel for his enforced activity. It is the case of "the square peg in the round hole." There ensues a battle of forces; the restrained muscular energies evoke vicious distempers, amongst which comes out urichæmia (acquired gout), and the mental phases are apt to become morbid, and, in turn, injuriously influenced by the onset of the gouty state.

Insomnia is sometimes a manifestation of irregular gout,[2] and has not received sufficient recognition. The rude interruption of sleep, which is common in an acute gouty paroxysm during the earlier hours of the morning, affords a type on which the less-marked and minor symptoms of gouty insomnia are founded.

The simplest form of sleeplessness which occurs in persons goutily disposed is due, in most instances, to acid or fermentative dyspepsia. The earliest and best account of this with which I am acquainted was given by Cullen,[3] who remarked that "persons who labour under a weakness of the stomach, as I have done for a great number of years past, know that certain foods, without their being conscious of it, prevent their sleeping. So, I have been awaked a hundred times at two o'clock in the morning, when I did not

[1] *Vide* contribution on this subject by Dr. Mortimer Granville. Lancet, vol. i. p. 676, 1881.

[2] On Insomnia and other Troubles connected with Sleep in Persons of Gouty Disposition. By Dyce Duckworth. Brain, July 1881.

[3] *Works*, edited by Thomson, vol. i. p. 127. Edin., 1827. Cullen did not himself connect this symptom with gouty tendency.

feel any particular impression; but I knew that I had been awakened by an irregular operation in that organ, and I have then recollected what I took at dinner, which was the cause of it." This sleeplessness is often caused by some particular article of diet which the sufferer digests imperfectly, or may be due to excess of wine or mixing of various liquors. Very often fatty or saccharine matters in excess, or mixture of fruit and wine, may cause this dyspepsia. There may be no overt symptoms of dyspepsia, but a simple excess in eating, or a single article of diet which is digested with difficulty, may so disturb the cerebral circulation that sleep is interrupted and suspended for a time. I described these symptoms in a paper which was published in 1873, and stated my belief that they were more common in persons who had tendency to gout.[1]

The dyspepsia probably arises from faults not only in the stomach, but in the duodenum and upper part of the small intestine.

Dr. Murchison described this form of sleeplessness, and attributed it to the hepatic derangement which induced lithæmia and other symptoms of gout.[2] A very noteworthy point about such cases is the particular time at which the insomnia begins. The sufferer retires to rest feeling quite well, and free from any discomfort. But his sleep is rudely interrupted, it may be by some unpleasant dream, and he is at once aware of uneasiness in the stomach, has heartburn or flatulence, and perhaps nausea.

If nothing be done, the patient will lie awake with throbbing head and active flow of thoughts for an hour or two, when sleep will return. On rising the next morning, he will probably experience some headache and find his appetite diminished. An attack of hemicrania may perhaps render the next day miserable for him. Such a form of dyspepsia is plainly a manifestation of a gouty tendency.

An incapacity to digest certain definite articles of food is very marked in the gouty, and is not unfrequently one of the earliest tokens of the disorder. In youth there may be vigorous digestion for all kinds of food, but as the third decade is approached, the inability declares itself.

It may be objected that there is nothing very remarkable about such symptoms, and that they are common enough. The charac-

[1] On Certain Forms of Sleeplessness. Brit. Med. Journal, December 27, 1873, and republished by Longmans, 1874.

[2] *Op. cit.*, 2nd edit., p. 590, 1877.

teristic part of the disturbance is the special digestive inadequacy at a very definite period. If we suppose that this enfeeblement is due to the taking of a full meal late in the day, and that the digestive powers would be adequate to dispose comfortably of the same if taken early—which is fairly conceivable—we have no means of knowing whether sleep, were it sought after some hours, would be interrupted under such circumstances. The fact remains that, in persons of gouty constitution, sleep is apt to be disturbed by the irritation arising from their peculiar digestive incapacity, and at a definite period of that process. An interval of four or five hours occurs between the meal and the awaking, the patient being disturbed within about two hours of retiring to bed. This is the time at which attacks of gout are especially liable to come on, and the sufferer is suddenly awakened with pain in the affected part. In this case, as usually in that of gouty dyspeptic insomnia, the patient has retired to bed feeling comfortable and in his ordinary health.

Other forms of gouty trouble manifest themselves not uncommonly in the early hours of the morning. Thus, attacks of bronchitis with asthmatic dyspnœa sometimes replace, or alternate with, regular onsets of gout, and the paroxysms of asthma are very prone to begin and to disturb the patient after midnight.

Not only is sleep thus interrupted, but other peculiar symptoms are met with in those of gouty proclivity in connection with the sleeping hours. It has been observed that in cases where a regular attack is expected, but does not supervene, sleep is abruptly broken some hours before the usual time of waking, and does not return. Some horrible dream may lead to this, and the same occurrence may take place for several mornings in succession. Scudamore relates two cases where sleep was merely disturbed by uneasy dreams, and gout was established in the joints on awaking in the morning.

Startings and shouting have been noted, associated, or not, with the dyspepsia preceding or accompanying gout. Grinding of the teeth during sleep is a symptom met with in those who are gouty. I have collected several examples of it, and Dr. Donkin likewise directed attention to some well-marked cases which occurred in the family of parents who were both gouty.[1] In this family there was also history of somnambulism. The father, a gouty man, was a habitual somnambulist in early life, and occasionally in later years walked about in his sleep. The mother ground her teeth at night for many years. She was of gouty parentage, as

[1] Brit. Med. Journal, February 21, 1880, p. 279.

already stated, but had had no overt gout herself. The whole family, of eight children, ground their teeth almost incessantly at night. Most of them were extremely "nervous," and walked in their sleep. They also talked during sleep.

I have record of one case in which there is both tooth-grinding and occasional somnambulism, the mother and maternal grandfather being distinctly gouty. Nightmare and startings of the limbs have been observed with some frequency in persons goutily disposed.

In connection with the subject of insomnia in the gouty, may be noted the fact that many of the special determinations of the malady take place during the night, whether the sleep be disturbed or not. The patient retires to bed feeling in his usual health, but on awaking in the morning, he discovers at once some new phase of his malady; it may be muscular pain or stiffness, angina of the fauces, the beginning of a hemicrania, or more or less severe pain in some joint or adjacent texture, such as a stiff neck, lumbago, or a burning phalangeal joint. These troubles, or some of them, have come on in the night, but have not been sufficient to disturb sleep. Cramps in the calves of the legs are especially prone to vex gouty persons at night, and sometimes for several nights precede a severe attack.

The fact that not only acute attacks of gout are apt to supervene during the hours allotted to sleep, but that other less severe gouty manifestations likewise occur during the night, or are found to have come on at that period, is one amongst many which may be appealed to in proof of the neurotic element in this malady, for it has this peculiarity in common with several other morbid affections which are conceded on all hands to be distinct neuroses. Thus, epilepsy, neuralgia, spasmodic asthma, gastralgia, angina pectoris, laryngismus stridulus, and hemicrania are all prone to disturb sufferers during the early hours of sleep, or immediately on awaking. In all these cases we have to seek for a cause which determines these outbreaks with such marked constancy in connection with the sleeping state.

From the nature of the case, we have but scant knowledge of most of the physiological conditions which occur during sleep. It is, however, known that the bodily temperature falls both in health, and in most, if not in all, morbid states, between the hours of midnight and six o'clock in the morning. Some observers have noted the minimum temperature to occur between eleven P.M. and three A.M., and although the fall does not amount to more than one or one and a half degree, it has nevertheless a

distinct significance as indicating some direct nervous influence on heat-production. Certainly this constant and normal reduction of temperature is independent of removal of clothing and abstraction of heat by bedclothes. Again, the subjective sensation of chilliness about three o'clock in the morning is familiar to all who sit up at night, and at that period, too, there is a maximum of weariness and exhaustion, and the greatest instinctive demand for sleep. Even the worst sleepers will commonly fall asleep at this hour, although they may have been miserably wakeful and restless previously.

There is also a greater susceptibility to cold at night-time. The "middle watch" is the most trying in all respects. This is the period of the greatest exhaustion of the whole nervous system, the automatic cerebral activity ceases, and sleep is "the diastole of the cerebral beat."[1] As the old writers put it, the "brain-power is lowered" in sleep.

Digestion is feebler during sleep than in the waking state, and so, too, is the action of the heart, and both the circulatory and respiratory acts are reduced in force and frequency. With the exception of the cutaneous functions, perhaps all others are at rest as far as possible.

Sleep is more profound in the earlier hours of night, and gradually becomes less so towards morning.

It seems impossible, in view of the foregoing considerations, not to find some reasons for the marked tendency towards irregular outbreak of nervous energy during the hours when so many cyclical processes are modified or interrupted.

In perfect health, and in persons not neurotically disposed, no irregular effects ensue; but in morbid states and in the neuroses, the hours of sleep are particularly those in which we might expect some outbreak or irregularity, and, as a matter of experience, we find such to be the case in marked degree.

The anæmia of the brain in sleep may have some influence in determining some of these disorders; but this condition is not believed to be the cause of sleep, but only an effect or concomitant of it. In the cases of dyspepsia already considered, there is a manifest source of irritation at a distance; but the peculiarity here is that it only becomes potent at a definite time to disturb sleep, either by the generation of some special morbid product in the course of digestion, which may act from a distance reflexly, or may enter the circulation and rouse the higher centres. These centres may, during the temporary depression due to sleep, be

[1] Foster, Text-Book of Physiology, p. 573, 1st edit.

more than at other periods specially irritable, and in the cases of such persons as are neurotically disposed, they are almost certainly in less stable condition than are those of the healthy.

The direct influence of an excess of uric acid circulating in the blood can hardly be lost sight of in connection with gouty insomnia. It is well-recognized that such excess is frequently present without the induction of any overt disturbance, nervous or otherwise, in persons who have no gouty proclivity; but in any case of true gouty habit we must not ignore the influence of what, when in excess, is a real poison in the circulation.

The special determinations of gout to certain parts are inseparably connected with excess of uric acid in such parts, and thus we may fairly conceive that some of the nervous symptoms which occur in the gouty owe their cause to irritation of nervous tissue by this peccant matter. Insomnia may well be one symptom due to this irritation.

It is to be noted that these troubles in connection with the sleeping state are not only met with when the subjects of them are in a very gouty condition, or as precursors of outbursts of gout in the arthritic form; they form part of the many minor affections to which persons goutily disposed are sometimes liable. Gout, like other maladies, has varying significance in the particular individual affected, and the fact of goutiness so far modifies the constitution or bodily habit of the patient. Thus, many sufferers have no troubles connected with the sleeping state, just as many have no urinary difficulties, no hemicrania, and no tophi.

The fact that attacks of gout set in violently by day, and not by night, in no degree minimizes the value and importance of accurate observations on the symptoms presented during sleep in persons disposed to gout. We must not fail to recognize their special significance when we meet with them.

Many sufferers are good sleepers in the intervals between severe attacks, and many others can secure good nights with due precautions as to diet and other habits. The particular insomnia described is rather the indication of the gouty habit than a particular phase of either acute or chronic gout, as commonly understood, and as such its importance has not hitherto been clearly signalized. Its recognition is necessary for the employment of the only line of treatment that can truly avail to avert it, and to break the persistence of the habit on which it depends.

Nightmare.—Sometimes, nightmare is a troublesome symptom in persons of gouty habit. Mr. Thomas Godfrey, of Mansfield,

has recorded a noteworthy instance in a woman of gouty inheritance, aged seventy, who suffered from gouty glycosuria and eczema, and who endured terrible nightmares at intervals all her life. They had increased greatly of late, so as to embitter her existence.

Retrocedent or Metastatic Gout.

When an acute attack of articular gout suddenly subsides, and symptoms of disturbance appear in other and, it may be, distant parts of the body, there is said to be retrocedence or metastasis of the process.[1] This is a form or variety of irregular gout. Sometimes, these phenomena recur rapidly and shift their positions; this is termed "flying" gout.

Inasmuch as no part of the body is exempt, or likely to be exempt, from the occurrence of gouty processes, the situations in which metastatic transference may be witnessed are very various. Hence, some forms of visceral gout. The most frequent forms of retrocedent gout are those in which, after subsidence in a joint, the disease alights on the heart, the brain, the stomach and intestinal canal, and the urinary bladder. Asthenic gout is most liable to prove metastatic, but sthenic gout will sometimes, under depressing treatment, shift to the viscera.

Retrocedent quality pertains to asthenic varieties of gout, and is always significant of atony and debility. The fact that the disease is anywhere manifested regularly or frankly in the joints indicates unlikelihood of metastasis. The subjects of flying gout are commonly of feeble constitution and with unstable nervous system. They may have been living on too low a diet, or have undergone too prolonged exertion.

Cardiac Gout—Retrocedence to the Heart.—The most marked instances of this particular form of flying gout have followed on some grave imprudence during an attack in a joint. Plunging a gouty foot in cold water or in snow has been known to determine severe cardiac pain within a few minutes, together with syncopal tendency more or less serious. The pulse may fall in frequency and volume, or may, with absolute cardiac failure, cease, and a fatal issue ensue. Intense spasm is thus induced. If the patient is rallied promptly, violent palpitation may ensue, with severe cardiac pain and orthopnœa. Cough and expectoration follow, and many days may elapse before the balance of health is restored.

In place of an arthritic attack, after premonitions of an impend-

[1] This peculiarity was noted by Galen and by Aretæus.

ing paroxysm, there may suddenly supervene syncope with great cardiac depression, ashy pallor of body, facial anxiety, and pulse of extreme feebleness. These symptoms may last for some hours, and only yield to active stimulation, while several days may elapse before ordinary health is recovered.

Nocturnal attacks of cardiac pain may come on after imperfect development of a joint-attack, and dyspnœa and collapse may be associated with this.

The same effects have been known to follow early and imprudent exposure to cold east winds when recovering from arthritis, as in a case related by Garrod in a man æt. fifty years. The pain was violent across the chest, and radiated into both arms. The pulse was feeble and intermittent. There was no overt cardiac disease and no pyrexia. The noxious practice, carried out by the illustrious Harvey in his own case, of plunging a gouty foot or part into cold water has often been followed by alarming symptoms.

It is probable that attacks of pseudo-angina pectoris are of this nature. Some of the cases of retrocedence to the heart described by early writers would now be explained differently, and be referred to true angina with structural disease of the aorta and its valves, or to fatty degeneration of the cardiac walls; while others were plainly examples of embolism of the pulmonary artery, due, sometimes, to unrecognized distal gouty phlebitis.

Minor attacks of flying gout affecting the heart may present no further symptom than feeble action, with or without intermittency.

It is probable that retrocedent gout alighting on the heart is never directly fatal, unless there is a decided degree of organic disease present, valvular or parietal, or both.[1]

This subject will again engage attention under the head of Visceral Gout. The danger is greater when parietal degeneration exists, than when valvular disease is present without damaged cardiac muscle. It is sometimes difficult to determine whether the heart or the stomach is more affected by retrocedency. Flatulent distension of the latter may interfere with the heart's action, and so simulate true cardiac spasm. Disengagement of the flatus commonly affords relief in these cases. A feeble heart may thus suffer severely and give rise to alarming symptoms.

Cerebral Gout—Retrocedence to the Brain.—Under influences similar to those just described, metastasis may occur to the encephalon.

[1] *Vide* Stokes on Diseases of Heart and Aorta, p. 359, 1854.

The symptoms vary in degree, from mental confusion to mania, and are sometimes apoplectiform with coma. Temporary insanity is well-recognized as sometimes dependent on gouty retrocedence.

With relief to joint-symptoms, indications of mental failure supervene, with wandering delirium. Vertigo, somnolence, photophobia, and disturbed vision may be noted in some cases. Apoplectic strokes may suddenly be induced with every classical symptom, including loss of consciousness, coma, stertorous respiration, relaxation of limbs, and failure of the sphincters. Hemiplegic weakness may be noted. All these pass off under suitable treatment or on the supervention of arthritis. Such examples resemble in all points the gravest case of cerebral hæmorrhage, and sometimes only reveal their true nature by a frank outburst of gout elsewhere, and by the complete removal of encephalic symptoms which thereupon ensues.

Apoplexia Arthritica.—Many noteworthy examples of apoplectic seizure, due to retrocedence to the brain, are given by Scudamore. They occurred chiefly in plethoric men at the age of sixty. Free venæsection proved of great value, and paralytic sequelæ were thus, he believed, often averted.

I do not believe that these cases are now so frequently met with as at the beginning of this century. Improvement in diet, greater temperance in alcoholic drinks, the use of diluents, and less tight clothing, probably account for fewer instances at the present time. In no such case can it be believed that the symptoms depend on cerebral hæmorrhage; the complete subsequent recovery negatives this idea. The diagnosis as to this may, however, be impossible in the first instance, and any undue tension of the pulse will in any such case justify more or less venæsection according to circumstances.

Gouty Encephalopathy.—Less grave attacks of gouty encephalopathy may be met with in which consciousness is not lost, but there appear signs of hemiplegic paresis in the face or limbs, with aphasia, thick speech, or amnesia. Severe mental emotions or overwork may determine such accidents in the presence of actively gouty states of the system. The essential lesion is probably that of vascular congestion.

According to W. Gairdner, "metastasis to the head is the most frequent form witnessed, indicated chiefly by a kind of stupor, in which sight and hearing are preserved, but loss of appreciation of surroundings, of recognition of persons, or place, or time prevails. The patient does not recognize members of his own family. The

utterance is imperfect or lost. He seems like a person entranced; his eyes are vacant and staring. The intelligence is impaired; actions are automatically carried out. The pulse is full and hard. This condition comes on gradually, being preceded by headache, somnolence after meals, deficient mental alacrity and loss of interest in matters which formerly occupied the attention. The patient realizes his mental failure and regrets it."[1] Such symptoms may sometimes betoken a degree of uræmia, and be rather of this nature than true expressions of metastatic gout.

Charcot has recorded an example of complete aphasia without loss of consciousness or any paralysis in a gouty man, which passed off on supervention of access of gout in the joints. Subsequently, attacks of epilepsy came on, preceded by a sensory aura in the little finger, limited to certain groups of muscles of one arm, with aphasia, indications of a cortical lesion. The aphasia may be intermittent, and has been known to persist for several months.

An exact diagnosis of the conditions prevailing in any case of gouty encephalopathy is only to be made after careful examination of the various organs and textures of the patient. Regard must be had to the presence of a gouty habit, or to history of remote or recent attacks. If there be indications of interstitial nephritis, or of feeble heart and atheromatous arteries, the possibility of uræmia or of meningeal or cerebral hæmorrhage must be borne in mind.

The discrimination is sometimes very nice, and at times not immediately possible. The age of the patient and the general tissue-state aid much in the determination. The pulse may be tense in both gouty and uræmic encephalopathy. It may be very difficult to pronounce at once for epilepsy or for uræmic eclampsia in a goutily-disposed person. Both sexes may thus suffer, but chiefly males after middle life.

Allusion has already been made to gouty mania, melancholy, and hypochondriasis, also to hemicrania, as indications of cerebral gout.

The following case, which I saw in consultation with Dr. Longhurst, exemplifies some of the features of cerebral gout:—

Mrs. T., æt. sixty, a lady of large frame, obese, and generally of robust constitution, was seen, first, two weeks before I was summoned, suffering from "confusion in the head" and flushing of the face. There had been two attacks of vertigo. No headache. The heart's action was rather feeble, no murmurs detectible. The pulse was regular, of moderate volume and full. Arteries not hard. An apoplectic fit being feared, the patient was purged, and benefit followed. An attack of sciatica

[1] *Op. cit.*, p. 77.

now supervened, which was treated with quinine. The day before I saw this lady, she had had many visitors, and subsequently fell into the same state of hebetude and mental oppression as at first. She lay prostrate, with her eyelids closed, intolerant of light, and was confused in her statements. The pupils were equal; no arcus senilis; conjunctivæ suffused; but no strabismus existed, nor was paresis detectible anywhere. There was no pain in any part. The cardiac sounds were clear and feeble, but the pulse was rather firm. The urine was natural, but had contained lithates some days previously. Some years ago the patient had suffered from similar attacks. There was history of occasional "bilious" disorders. An encouraging prognosis was given.

The treatment consisted in calomel with acetous extract of colchicum, and a mixture with bromide of ammonium, aromatic spirit of ammonia, and compound tincture of lavender. Sinapisms were applied to the nape, and hot pediluvia employed. Light dietary. Improvement gradually set in; but complete recovery was not attained for about four months. Before the last attack this patient had been much busied setting her house in order on her return from the country.

I am indebted to Sir Andrew Clark for the particulars of the following cases which have occurred in his practice, illustrating the points now under consideration.

He was called to see a gentleman, æt. fifty, who had suffered for two days from incessant and irregular cardiac palpitation, some dyspnœa and recurrent vertigo. No objective signs of disease could be discovered; but in the history of his habits, in his aspect, and in the disordered condition of his functions, it was considered that there was conclusive evidence of a gouty state. He was put on a meagre diet, ordered to take a dose of calomel at night, and to place his feet in a hot mustard foot-bath. Between two and three o'clock next morning the patient was awakened with an acute attack of gout in both feet, when the cardiac irregularity, dyspnœa, and vertigo all disappeared. Within the following ten years this gentleman had several similar attacks, and they were always ended by an attack of gout brought on by immersion of the feet in water as hot as could be borne.

A. B., æt. fifty-seven, medical practitioner, a tall, well-built man with a large head, burst into tears on entering the consulting-room. Recovering, he said, "I have come here merely to satisfy my friends; you can do nothing for me; I have a tumour under the dura mater over the left eye." In reply to the question how he knew that, he said that several "specialists" had given that opinion. He was fully examined, and no conclusive evidence of the existence of a tumour was discovered. There was, furthermore, no sign of organic disease anywhere else. Inquiring as to probable cause of his symptoms, which were persistent headache, transitory disturbance of vision, and recurring numbness and formication on left side of the body, it was elicited that he took a leading part in the affairs of his neighbourhood, that he lived generously, that he did not fret himself with denials about the quantity of wine consumed, and that he took no exercise whatever. Beyond a few scattered patches of eczema, tinglings in the feet, and indications of imperfect excretion, no other signs of grave disorder were detected.

To a suggestion of gout he replied with indignation, said that it had never existed in his family, and that he had never done anything to beget it in himself. When the patient asked what should be done on the supposition that he was gouty, and was told that he should live a careful life, eat meat only once a day, take regular exercise, and substitute a little spirit and water for wine, he replied that he did not believe he was gouty, and that if he was, such a regimen would destroy him. Nothing more was heard of the patient for six months, when Sir Andrew Clark was summoned to the country to see a patient at X——. On arriving at the station, he was met by his *quondam* patient with the cerebral tumour. "Ah!" said Sir Andrew, "why have you

not fulfilled your promise to die of your brain-disease?" "Hush," said the patient, "not a word. You must not mention the matter here; I have had a famous attack of gout in the feet, and it has dissolved the tumour!"

The prevailing condition of the blood under these conditions is almost certainly that of diminished alkalinity due to urichæmia. Antecedents of cerebral troubles are commonly on the side of the digestive system, whence a diminished formation of uric acid and urea (according to Dr. Haig), owing to defective absorption and nutritive changes, and a lessened acidity of the blood. The latter state entails the outcome of the uric acid pent up in the liver and spleen, which induces urichæmia, and so provokes some gouty manifestation. Dr. Haig has shown that the amount of urea falls from 561 to 363 grains in the four days preceding an uric acid headache, owing to defective absorption and metabolism.

Retrocedence to the Stomach and Intestinal Canal.

Gastro-Enteric Gout.—The symptoms induced by gout alighting in the stomach and intestines are multiform. In the simplest and most unequivocal cases, sudden pain in the stomach is the first and prominent feature. With this is associated great depression, bodily and mental. Onset of articular gout affords sudden and complete relief. Vomiting sometimes occurs, also pyrosis and retching. Attacks of gastralgia with vomiting may occur from time to time in those subject to occasional fits of frank gout. Both sexes may suffer, perhaps males more frequently. Gouty concomitants are usually detectible in such cases, forming manifestations of irregular attacks. The gastric attacks do not always terminate by articular outbursts, but in a series of such ailments an attack of frank gout may occur, giving the clue to their true nature. Professor Ball and Dr. Buzzard have surmised that in some of the cases in which gouty affection of the stomach has been diagnosticated, the symptoms are properly referable to the gastric crises which form part of the malady known as tabes dorsalis or locomotor ataxia. It is well to be mindful of the possibility of such an error. Some writers are sceptical as to the occurrence of gout in the stomach. Sir Thomas Watson remarked that "gout (so-called) in the stomach was, under the test of an emetic, sometimes nothing more than pork in the stomach." Without doubt, erroneous diagnosis has often been made of this condition when other explanations would have been more in accordance with the facts presented. But that gout may seize upon the mucous membrane of the

stomach and intestines, causing acute gastro-enteritis, and prove fatal, admits of no doubt, since autopsies have attested the fact.[1]

Severe chronic dyspepsia may form part of a gouty habit where for a long time no joint-symptoms appear, or where these are very trifling, although highly significant.

Application of cold to gouty joints has often been followed by gastric spasm; even getting the feet wet while they were gouty has been effective in inducing this condition. Two forms of gastric trouble are met with as a result of metastasis: (*a.*) the spasmodic, and (*b.*) the inflammatory.

In Moxon's remarkable case, already referred to (p. 89), the latter form occurred. The patient was a man, æt. thirty-nine, who had suffered much from "rheumatic gouty" symptoms, and later became the subject of albuminuria and diarrhœa, which proved fatal. The *post-mortem* changes were so noteworthy that I give them here somewhat fully.

In the lungs was found early-spreading phthisis, with a few clusters of recent tubercles. The heart was hypertrophied, weighing 15½ ounces. The stomach was thick-walled and rigid from inflammatory infiltration; the mucous surface patched unevenly with a layer of buff-coloured fibrin, which, on removal, brought away part of the mucous membrane. The colon showed boil-like phlegmons in the submucous tissue in various stages, some ready to burst, some already small abscesses, others as ulcers, some of the latter healing and others cicatrized. The kidneys were small and granular, with sodium urate in grains in the pyramids. The cartilages of the great toe and knee-joints were encrusted, and rendered uniformly opaque white by urates. There was no history of sudden retrocedence of gout to the stomach in this case. As remarked by Moxon, it may be questioned if the gastritis and phlegmonous colitis were due in this case to gout. Such changes are uncommon under any circumstances, and form no part, at all events, of concomitant lesions, as ordinarily met with, in chronic interstitial nephritis. Tuberculosis will not induce such conditions. Gout sometimes may do so.[2] A noteworthy case of retrocedence to the stomach and intestines is recorded by Landré-Beauvais, with an account of the appearances after death. I have referred to this case in the section relating to gout in paralysed limbs, p. 234.

Dr. Sutton informs me that he has met with two or three cases in which he made a diagnosis of gout in the stomach. They occurred in men who suffered from atonic gout; the symptoms were severe, and accompanied by great depression and signs of collapse. The patients complained most of distressing pain in the epigastric region; this was associated with very anxious expression of countenance, cold sweats, small pulse, and extreme restlessness. The symptoms yielded on supervention of gout elsewhere. In one case he made an autopsy where the symptoms of rapid collapse led to the belief that there was cardiac rupture, death being sudden. He found no such rupture nor other organic

[1] Professor Ball of Paris thus expresses his view of the relationship of a gouty habit to disorders of the stomach:—"La goutte est pour l'estomac ce que le rhumatisme est au cœur."

[2] *Vide* Musgrave, *De Abscessu Intestinorum Arthritico*, *op. cit.*, p. 173.

changes to account for the rapid death, save early granular contraction of the kidneys and much sodium urate deposit in and around the great toe-joints.

The following case was kindly related to me by Sir Andrew Clark. J. J., æt. fifty, came home from the Colonies with supposed disease of the stomach. At first his chief symptom was merely pain after food, but latterly the pain had become constant, and was aggravated by everything ingested. He had lost flesh, strength, and colour. The circulation was hurried, and he was nervous and sleepless. No regimen and no medicine gave permanent relief; but whilst using liquid food very freely, and taking nightly a hot pediluvium, he was suddenly seized with acute gout in a hand and foot, and from that time all gastric symptoms disappeared.

This was an example of larval gastric gout.

Gastrectasia.—Sometimes, extreme dilatation of the stomach occurs in cases of this nature.

Gouty processes implicating the intestinal tract express themselves by pain, spasmodic colic, vomiting, and diarrhœa. Spasmodic and inflammatory forms are met with. Constipation is sometimes a consequence. Such enterodynia was long since recognized and termed "arthritic colic."[1] There may be much intestinal flatulent distension. Diarrhœa may be the solitary expression of gouty enteritis, lasting for a few days, with much mucous discharge. These attacks are apt to recur at intervals of a few weeks, and may alternate with regular fits in the joints. Indiscretions in diet, strong emotions, or exposure to cold may determine them. Warnings may appear in the form of slight pains, hemicrania, or lateritious sediment in the urine. James Begbie has recorded a marked example which occurred in his practice.[2] A clergyman, æt. sixty, of abstemious habits, a great sufferer from gout, which was strongly inherited, after a longer interval of freedom than usual, was suddenly seized with abdominal pain, vomiting, feverishness, and great pain over the belly, increased by pressure. There was a small, rapid pulse. The first diagnosis was peritonitis. The symptoms did not respond to ordinary treatment. Surmising that gout might be the cause of the attack, colchicum, which had always relieved the articular fits in a marked manner, was given. Copious diuresis followed, constipation, which was severe, yielded, and speedy relief was obtained to all the symptoms.

These attacks may be sometimes unattended by pyrexia.

[1] It is important to note, in connection with arthritic colic as described by Musgrave, who long practised in Exeter, and, perhaps, by other observers, that much of the gout seen by them was due to, or associated with, lead-impregnation. Much of the colic described may have been lead-colic from cider-drinking. It was Sir George Baker, another Devonshire man, who discovered the true cause of the colic named after his county.

[2] *Op. cit.*, p. 17.

The following case affords a well-marked example. A clergyman, æt. thirty-four, of robust figure, large head, hair dark, thinning on vertex, inheriting gout from his maternal grandfather, his father also having suffered from "gravel," consulted me on November 19, 1887. He complained of periodical attacks of pain, which came on gradually in the belly between the navel and pubes, becoming more and more intense, and lasting for about eighteen hours, complete recovery not taking place for three or four days. The pain radiated over the abdomen, especially to the left side. His last attack, a typical one, began at 4 P.M. on the 12th inst. with sharp twinges. At 3 A.M. on the following day vomiting occurred, pulpy undigested food being rejected. After attacks of this, the pain yielded somewhat, and some heavy sleep followed. By 9 A.M. the intense pain had ceased. The urine was thick for two days, and not till the 16th was he entirely free from discomfort.

These attacks began at the age of seventeen, and recurred at first at intervals of six months, becoming more frequent. For a week before each he felt "bilious" and mentally depressed, and his sleep was unrefreshing and attended with dreams. At these periods he became giddy while reading. He had always been a bilious subject. He had suffered from several attacks of gout in the heels, coming on suddenly in the night after the spasmodic seizures. The latter now recurred at intervals of five weeks. He took beer freely when at college. Certain "gout pills" had been very beneficial. He was a large eater, and took meat three times a day, sometimes drinking beer at breakfast.

There were no signs of organic disease anywhere detectible.

The urine (*post jentaculum*) was alkaline; sp. gr. 1.020, void of albumen and glucose. The tongue was fairly clean.

He was rigidly dieted and ordered more exercise. Blue pill and colocynth with henbane pill were given, and an alkaline draught twice daily after food.

A month later there was improvement. In his last attack there was no vomiting, but the pain was very severe and lasted longer than formerly, being concentrated in the left iliac region. He was now ordered to take each night a drachm of compound rhubarb-powder with some dried sodium carbonate. I saw no more of this patient, but a year later he wrote that he was "still a cripple in the stomach, but got through last winter wonderfully well. The pills were necessary as well as the powder to secure relief from the bowels. A bad attack occurred on February 28; since then several approaches to one. The attacks have been prevented from their final issue by the pills. Smoking after meals causes pain in the affected region."

This patient has probably been imprudent in diet, and not benefited accordingly.

I will add a second example which is characteristic of gouty gastro-enteritis with colic.

X. Y., æt. thirty-eight, a wealthy merchant, residing in Glasgow, consulted me, in November 1888, for periodical attacks of abdominal pain. He was a tall, spare man, with red hair, thinning on vertex, and fairly robust. His parents were living, the father having suffered from "rheumatic" pains in the knees, the mother being crippled by "chronic rheumatism," which had lasted seventeen years, and suffering from bronchitis. He had only one brother, who was subject to bilious attacks, followed by vomiting. Ten years previously this patient had suffered from "erysipelas," and again last year after an injury. The disorder was termed "rheumatic erysipelas," and spread all over the body. In 1863 he had pemphigus, and three years ago a bad attack of eczema. For four years past he had suffered once or twice in the month from attacks of griping pain in the abdomen, especially violent at the epigastrium, which came on usually about 7 P.M. He was unable to connect them with any habit of life or any article of diet. They would follow a day of work or of

pleasure. The pain began about his dinner-time, and waxed steadily worse for twelve hours, preventing sleep, and inducing retching and vomiting of frothy and watery matters, not acid and not bilious. No jaundice followed the attacks, but the belly remained tender for several days afterwards. Slight chilliness was felt at beginning of the paroxysm. The pain tended to pass into the right iliac region in the course of three or four hours, when a distinct swelling could always be felt, which his attendants had described as due to a "swollen valve." This disappeared within the next twenty-four hours, and left the parts in a natural state.

The bowels were fairly regular. Before the attacks there was either constipation, or one or two loose motions were passed on the day they occurred. The effect of each seizure was to cause weakness and prostration, and to confine the bowels for a day or for thirty-six hours, the subsequent motion being dark and in detached lumps. The urine was scanty and high-coloured during the attacks, becoming turbid on standing. No feverishness was experienced.

There was no history of frank gout, but the effect of a few glasses of champagne or port wine was to cause pains in the knees and the soles of the feet. He was generally temperate, ate a good breakfast, a light luncheon, and a hearty dinner with sherry and claret. His habits were active, as he walked and rode several miles daily.

He had been to Bath on two occasions, and had derived benefit from the waters there, together with a restricted dietary.

I could detect nothing wrong in any of the organs. The tongue was clean, and slightly indented. The urine was natural.

During the attacks nothing was found to afford relief, and the stomach became so irritable that no food or medicine was tolerated. The belly was somewhat distended.

I prescribed calomel in pills in occasional courses, with Friedrichshall water and a nightly dose of compound rhubarb powder, and gave directions for a very rigid diet.

In this case there were plain indications of the gouty habit. It was clear that these attacks of gastro-enteritis with spasm were not due to indiscretions of diet at any particular time. Their recurrence I believe to have been due to a "growing up" of a gouty state, induced by continual use of unsuitable diet. It is probable that gout may sooner or later supervene in more frank form in some joint.

Dr. George Budd described the two varieties of gastric gout which are to be met with.[1] (1.) The form met with in chronic gout, when there is a feeling of weakness and sinking, with griping pain and cramp. Vomiting is rare, and there is no pyrexia or sign of inflammatory action. Pressure relieves the pain. (2.) The form resulting from retrocedence of active gout from a joint. Here there is much pain in the stomach, pyrexia, frequent retching or vomiting, and often profuse diarrhœa. The symptoms are alarming, and the disorder often fatal from collapse within two days. The treatment of the two forms is very different.

Vesical Gout—Retrocedence to the Bladder.—Many well-marked examples of gouty cystitis have been recorded, in which this trouble has ensued on metastasis from arthritis. Exposure to wet, cold, and fatigue are common determinants in this as in other examples of retrocedency. Urethritis is sometimes thus induced

[1] On the Stomach, p. 103.

with puriform discharge and scalding on micturition, and gonorrhœa is often suspected in such cases.

The attack may come on suddenly, with pain, frequent micturition, hæmaturia, ardor urinæ, and restlessness. The urine is scanty, high-coloured, and contains mucus, blood, or pus. The articular symptoms are in abeyance, and the cystitis may persist acutely for some days, and not yield completely for many weeks.

In this case, as in other instances of metastasis, there may be symptoms indicating that the disorder has also alighted on other parts. Thus, the heart may be affected after the bladder, and not be restored to its natural condition till articular inflammation is again established. Gouty cystitis is analogous to the lumbago, pneumonia, bronchitis, or gastro-enteritis, which sometimes suddenly vex subjects of retrocedent gout.

Elderly people are most frequently sufferers from this form of cystitis, and prostatic enlargement is often associated with it. Bronchitis, after being extremely rebellious to ordinary treatment, may quickly yield to an attack of gout in the foot.

Gout of the hollow viscera excites the same symptoms, great irritability of mucous surfaces and intolerance of all matters brought in contact with them; hence, vomiting, diarrhœa, cough, and frequent micturition, according to the organs involved.

Eczema has been known to disappear from the integument, and severe cystitis to set in forthwith in gouty subjects. This may be regarded as an eczema of the bladder or a visceral enanthem.

Gouty Orchitis, Parotitis, and Tonsillar Angina.—Sudden metastasis to the testis is well-recognized, also to the parotid gland. I have already mentioned the transference of gouty (tonsillar) angina to the great toe-joint.

Urticaria and Fugitive Cutaneous Congestions.—Gastric disturbances in the gouty are sometimes quickly relieved by the occurrence of urticaria, or of other fugitive congestions of the integument.

Acute articular gout may sometimes pass off without any metastasis under the influence of strong mental emotion, such as shock or fright, or from the profound effect of sudden and overwhelming joy.

CHAPTER XIII.

VISCERAL GOUT AND GOUT OF SPECIAL ORGANS AND TEXTURES.

Visceral Gout.

It has, I believe, been sufficiently shown in the preceding sections that gout attacks the viscera as certainly as it involves the joints. Hence, no apology is needed for the term visceral gout. I have described the several affections which are thus induced, and need not refer to them further. The chapter on the morbid anatomy of gout affords ample proof of their existence. We have seen that both functional and organic disease of the viscera and of abarticular parts may be due to gouty dyscrasia.

The causation of visceral attacks is probably exactly that of articular paroxysms. Retention or precipitation of uratic salts in any locality is apt to induce a gouty fit, and hence hyperæmia, irritation, pain, and inflammatory symptoms of varying degree in such parts, with special indications of perverted function, according to the organ involved.

Illustrative examples are furnished by bronchitis, cystitis, orchitis, parotitis, neuritis, phlebitis, gastro-enteritis, and the condition of the liver in respect of glycosuria in the acute form, or in exacerbations, as met with in persons while suffering from a more than usually gouty state of the system. Personal proclivity and tissue-state probably have to do with the peculiar determination, while the dominating condition of the nervous system acts no less forcibly in each case.

Retrocedency of gouty process to any organ affords an apt illustration of visceral gout admitting of no dubiety.

It has already been shown that, in cases of gouty heredity, various organs may, even in early life, be the seat of disturbances due to that dyscrasia, and, further, that such manifestations may

be substitutions for more obvious articular symptoms, which, however, may supervene later in life. To use the words of Charcot, "When the visceral affection precedes the articular gout, and constitutes for a longer or shorter time the only manifestation of the diathesis, it is called *larval* gout; when, on the contrary, it follows the articular symptoms, it is called *retrocedent*, provided, at least, that the metastasis has been excited by the intervention of an external cause—cold, for example."[1]

The viscera may be primarily attacked, or become secondarily involved by retrocedence of gouty process from joints or other parts. It would be strange if, in a disorder of the whole body, any part were spared, and it has been shown that, with the possible exception of the lymphatic glands, none of the bodily textures are exempt from overt manifestations of the disease.

The liver, as the largest viscus, is perhaps more concerned than any other organ. The kidneys suffer as severely as any joint from textural changes. The heart and vascular system generally, and the lungs bear the full stress of perverted nutrition in consequence. The skin, the alimentary tract in all its length, the bladder, testes, penis, parotid glands, and, without doubt, the brain and nerves, may all be involved.

By inheritance, as has been shown, the gouty habit impresses its peculiar features on the as yet uninfluenced tissues of the body, implanting a veritable gouty physiognomy, capable of recognition in early adult life.

Certain gouty persons are more apt to present visceral than articular phases of the dyscrasia, others the reverse of this.

Many disorders of the viscera may be recognized as of gouty nature, when as yet no plain signs of articular gout have supervened. A sudden and fugitive type commonly attaches to these. Thus, in some individuals a periodical recurrence of bronchial catarrh, of functional disorder of the liver, of headaches, and various other troubles, is sometimes a truly gouty expression, and demands treatment in accordance with this view of their intimate causation. Certain fluxes, bloody or otherwise, are of this nature, as severe epistaxis in the daughters of gouty fathers, which may recur occasionally through a long life, and prove of no moment, possibly being rather salutary, and calling for no such interference as plugging of the nostrils. The blood is soon made up, and its loss may avert worse consequences. A diagnosis of the true significance of cases of this nature is hardly possible without careful inquiry into family history and proclivity, and

[1] *Leçons sur les Maladies des Vieillards et les Maladies Chroniques*, Paris, 1868.

in ignorance of this, errors both in treatment and prognosis may readily be fallen into.

Hæmorrhage from the bladder in elderly men of gouty habit is another case in point. No treatment is called for, and no harm results.

Several grave diseases of the eye are illustrative in this relation. The gouty habit, or taint, is sufficient to induce these. It is an error to expect always a history of articular gout in such cases. It may, or may not, be forthcoming. The life- and family history of the patient commonly afford the clue to ætiology in these cases. Herein, as I am well aware, lies the great danger of pronouncing too readily for gouty influence. This snare will not entrap those who are duly cautious, and only anxious to seek the truth, and, in any doubtful case, an open mind must be kept. A large and varied field of work is the best school wherein to chasten hasty and crude opinions in any class of ailments. Specialism is notoriously warping to the mind, and inducive of error.

Visceral gout is commonly a phase in the chronic form of the disorder, and in its acutest forms is seen in the several metastases, which are duly recognized. The storms (as they have been termed) of glycosuria met with in the gouty have been suggestively considered by Dr. Ord as due to hepatic gout, the hyperæmia and undue activity of the liver being sometimes a substitution for articular troubles. In the chronic interstitial nephritis of gout there are sometimes active phases of disturbance in the kidney, leading to more than the wonted polyuria or to increased albuminuria. A fugitive and a sudden character pertains to these phases, very suggestive of the like manifestations in gouty joints. So, with respect to bronchitis, gastric catarrh, neuralgia, hemicrania, and many other ailments attaching to the dyscrasia. The local afflux subsides, and the parts return to their normal trophic equilibrium, at whatever level that may be.

In this chapter I propose to describe more in detail some of the characters of gout as affecting certain organs and other abarticular parts.

Gout as Affecting the Heart.—The morbid anatomy of the heart, as met with in fatal cases of gout, has been already described. The gross changes discovered form part of the wide-spread degenerations associated with, if not actually dependent on, chronic gout. Thus, they are commonly related to the degree of sclerosing nephritis present in such cases, and are also more or less dependent on the condition of the coronary arteries in respect of indu-

ration and atheroma. Chronic valvulitis may be induced, with consecutive hypertrophy. Dilatation may follow on this as a result of general failure of nutrition, aggravated, commonly, by imperfect blood-supply through the coronary arteries.

The occurrence of painful cardiac neuroses, such as true and pseudo-angina pectoris, has been previously discussed. In chronic gout, cardiac action may be preternaturally violent and rapid, or, again, remarkably infrequent. Intermittency of the pulse has long been recognized in certain cases as dependent on gout, and is more often met with in practice than the reverse condition of tachycardia. Either state may excite much concern, and demands careful analytical study.

It has been axiomatically laid down that an irregular, intermittent pulse in persons past the prime of life, and especially if associated with derangement of the functions of the stomach, and unaccompanied by any clear indication of disease of the heart, will frequently be found to be due to gout.

Professor Burdon Sanderson has directed attention to irregularity of pulse in persons of gouty constitution where no cardiac valvular disease is present, and shown that the cardiac rhythm may be regular during an attack, and become irregular at other times.[1]

"During the period of inspiration, the frequency of the heart's contractions is increased, the pulse becomes dicrotic, its form being entirely different from that which it assumes during the respiratory pause, when it is relatively retarded. During the respiratory pause, on the other hand, the contractions are less frequent, the diastolic intervals are longer, so that the heart has time to fill completely before it contracts. Hence, the quantity of the blood delivered into the aorta is much larger in proportion to the quantity which can be transmitted by the capillaries. The duration of the ventricular systole is greater, and the arteries remain a much longer time distended. The pulse is no longer dicrotic. Thus, in one and the same individuals you have dicrotism during inspiration, absence of dicrotism during the respiratory pause; the only difference in the state of the circulation being that, in the one case, the diastolic pause is shortened, and consequently the ventricle contracts upon an insufficient supply of blood; whereas, in the other, its expansion is complete and its systole effectual."

As pointed out by G. W. Balfour, cases of palpitation in the gouty are apt to be subdued by emotional excitement or exertion,

[1] On the Sphygmograph, p. 76.

the reverse condition occurring when organic valvular disease is present. The discomfort of palpitation is relieved although the cardiac beats are more frequent. Another point of distinction between functional disorder of the heart arising from gout, and symptoms due to organic disease, lies in the subjective character of the palpitation in the former, there being sometimes thumping, or tumbling, sensations with little or no dyspnœa, while in the latter dyspnœa is both subjective and objective, and generally well-marked, palpitation not being a constant or, when present, a very distressing symptom.

Gout alighting on the heart by retrocedence from other parts has been referred to. In such cases there is, so far as my knowledge goes, no clinical or other proof of any inflammatory process involving the muscular walls, or of endo- or peri-carditis. The latter is only known as a complication in associated chronic nephritis.

In cases of gouty irregularity of the pulse, special regard must be had to the volume, which is good so long as the heart is organically sound. If the cardiac walls are softened and dilated as the result of degeneration, there may be small volume and low tension associated with the irregularity. The intermittent character may pass off after a short time, or may remain for many years without prominent symptoms.

Extreme infrequency may be observed where the heart is feeble and dilated, the pulsations falling as low as twenty in the minute, as recognized at the wrist, but occurring with greater frequency at the heart, many of the beats not being forcible enough to reach the distal arteries.[1] Sometimes intermittency depends on purely nervous causes, apart from gout, in gouty subjects; but there is probably in some of these cases, as part of the gouty neurosis, an unstable condition of the cardiac centres in the medulla oblongata.

Here, as always, the gravity in any particular instance is to be gauged by the nutritional state of the cardiac walls, and the general condition of the patient in respect of gouty cachexia.

Where the gouty habit is plainly established, the prognosis is, so far, not unfavourable if the heart be fairly sound and the renal functions unimpaired. Intermittency *per se* is not a grave symptom, but with associated degenerative changes the case is very different, and the outlook unfavourable.

[1] A noteworthy instance in point is recorded by G. W. Balfour, where this was observed in an old lady, "long gouty, though without regular attacks," with feeble and dilated heart, who suffered from epileptiform attacks, associated with flatulent dyspepsia. These passed off, and life was prolonged for some years without any return of the severe symptoms. *Diseases of the Heart*, p. 258.

A similar prognosis attaches to the like symptom as sometimes permanently induced by malarial fevers, in which case old age may be reached.

The sudden deaths which occasionally overtake the gouty are probably always due to aortic disease or to fatty heart, and depend on syncope, or on rupture of the left ventricle. The fatal event may be determined by an attack of gout, which disturbs the cardiac action. Where this is not present, the case may be erroneously supposed to be one of retrocedency.

A pulse of high tension is very commonly met with in the gouty, but is by no means always to be found. It may occur long antecedent to cirrhosing change in the kidneys, but is probably often in relation to that change, and may be expected in association with it as part of the cardio-vascular degeneration. Continued high arterial pressure tells in its wonted manner more especially on the aortic valves, leading to sclerosis, and inducing thereby either stenosis, or permanent patency, or both. The mitral valves also partake of this change in consequence of strain, the same sclerosing endocarditis here leading to reflux, and sometimes to stenosis. Without question, mitral valvular lesions are most often due to rheumatic disease, but gout sometimes plays a part, albeit a small one, in inducing sclerosing change in the manner just indicated. Hence, elderly men may present symptoms of this disease in association with obvious gouty dyscrasia, in whom no previous history of rheumatism is to be met with. The more ordinary rheumatic form is an appanage of early life, and very specially of the female sex. In the gouty form, as seen in elderly persons, there are not present, unless degeneration of the cardiac walls has taken place, the well-recognized symptoms associated with mitral stenosis in the young. The heart may be vigorous and acting forcibly, although the pulse is irregular as to rhythm and volume. If severe bronchitis occurs, the right side of the heart may yield under stress of this, and add to the gravity of the case.

Where high arterial tension prevails in any case, it is not an abiding condition. It may vary from day to day and from hour to hour. It is met with in acute and in chronic gouty states, also in incomplete or, so-called, suppressed gout.

The contamination of the blood by imperfectly metamorphosed products insufficiently oxydized is the primary cause of it, the capillary circulation throughout the body being thus impeded. Hence lithæmia, pregnancy, anæmia, and lead-impregnation are well-recognized causes of increased blood-pressure in the arteries.

In the latter case, as suggested by Broadbent, there may probably be formed albuminates of lead, which are too stable for dissociation and oxydation, and it is proved that lead checks elimination of uric acid from the system. Lime-salts in excess probably act similarly, and both tend to induce gout.

Continued dyspepsia, common as an antecedent in gout, and more common as an irregular phase of it, may in time set up arterio-capillary fibrosis and high pressure of the blood-column as a result. Causes of high tension, acting temporarily, may on passing away leave the pulse of natural firmness. In the gouty habit the tendency is very apt to remain in greater or lesser degree.

It may not always be recognized by the finger, and may only be demonstrated by the sphygmograph when skilfully applied. The specific connection of high pulse-tension with urichæmia has within the last few months been proved by Dr. Haig.[1]

In gouty, as in other states, high blood-pressure may be reduced by appropriate medicinal and dietetic treatment, and efforts in this direction are desirable in any case where obviously untoward symptoms are present in association with it, regard being always had to the well-being of the patient, whatever his ailment.

The sphygmographic tracings depicted on page 223, figs. 17–20, illustrate some of the features of the pulse-wave in cases of gouty habit.

Renal Calculi in Relation to Gout.

There is an unquestionable relation between the gouty habit and the formation of uratic calculi in the kidney. It is rare for gravel and gout to co-exist. Passage of gravel most often precedes gout, and ceases on the supervention of it. The two conditions may alternate. Calculi may occur without any overt gouty manifestations, but cases are met with in which, after distinct attacks of gout, calculous renal symptoms, such as pain and hæmaturia, come on, and ordinary gouty symptoms subside. This may occur even in the third decade of life, of which an instance has been related to me by my colleague, Mr. Langton.

In another case, a man of fifty-seven years of age suffered from several severe attacks of gout in the great toes, and in ten years began to suffer from hæmaturia, which lasted, with short and incomplete remissions, for eighteen months. There was no very marked lumbar pain. There were no signs of disease of the bladder, and although no calculous particles were

[1] Brit. Med. Journ., February 9, 1889.

passed, I entertained no doubt as to the presence of stone in the kidney. There were no indications of new growth in this case.

When the calculi are large, they have little or no tendency to move, and, hence, symptoms of renal colic are not met with in such cases. The worst cases of nephralgia and colic due to calculous formation appear to be connected with the presence of small, rough, and readily removable stones—in the earlier stages, therefore, of the disorder. In some cases of calculous kidney pain is absent, or but little marked, and may be indifferently referred to the lumbar region, and hardly to one side rather than the other.[1] Mr. Knowsley Thornton has affirmed that the symptoms of stone in one kidney may be caused by the presence of stone in the opposite organ; but it would not be safe to reckon on this, if operative procedures were contemplated. The calculi usually consist of uric acid, but may contain ammonium urate or calcium oxalate. They may be formed of alternate layers of these. Uric acid calculi are perhaps the most brittle of all forms met with. They sometimes break up spontaneously in the bladder.

A case is recorded by Dr. Ord,[2] in a gouty man, aged eighty-four, of great bulk and weight, in which spontaneous disruption occurred in uric acid and ammonium urate calculi. Many spherical calculi were passed, their fragments, like segments of exploded shells, indicating that they had broken some time before emission. Dr. Ord believed the fracture to be due to an expansion of the central portion, acting like the exploding powder in a shell, caused by the action of alkaline urine, which led to swelling of mucoid matter in their composition. He refers to several other examples which support his view as to the cause of disruption. Dr. Debout d'Estrées, of Contrexéville, showed me some calculi of uric acid which had thus broken up, and he attributed the fracture to powerful detrusive action of the bladder, which crushed the stones against each other. In my opinion, Dr. Ord's explanation is, probably, the most rational one.

It is well known that hæmaturia may long and severely persist in consequence of very small renal calculi. The experience of large numbers of cases successfully treated at Contrexéville and elsewhere incontestably proves this. The personal and family history in many such cases justifies the opinion that the uric acid

[1] One of the most remarkable instances of renal calculi on record occurred in a man, æt. thirty-eight, who had been gouty for eleven years, and had tophi. His father was gouty. There were more than a thousand stones in the two kidneys, and one weighed 36¼ and another 9¾ ounces. (Dr. Gee's Case, St. Barth. Hosp. Museum, Series xxviii., No. 2349.) *Vide* Med. Chir. Trans., vol. lvii. p. 77, 1874.

[2] *Op. cit.*, p. 93.

disturbance constitutes for them a variety of abarticular gout. In many others no such opinion is warrantable. It is important to make the diagnosis of renal calculus in many cases where no fixed lumbar pain or history of ureteric spasm exists. Recurring hæmorrhage is often the sole symptom. Where cachectic states are present or indications of granular kidney prevail, hæmorrhage may occur, occasionally to a considerable amount, independently of calculous irritation. I have noted the occurrence of vesical bleedings in the gouty, and feel sure that these may occur in the kidney, as elsewhere,—so as, *e.g.*, to induce epistaxis or hæmatemesis,—because of a bad state of the blood-vessels, which, from senile or other forms of decay, become brittle. These may all, or singly, be the precursors of cerebral hæmorrhage. The latter is apt to occur in the gouty, with granular kidneys, without any bad state of the arteries generally. I have known, for example, a strong man, aged forty-nine, whose arteries were very good, struck down with fulgurant apoplexy, due to large hæmorrhage. There may have been a small aneurysm, but it was not detected. The kidneys were granular, with uratic streaks in the pyramids, and the cartilages of the great toe-joints were plastered with urates. There was hypertrophy of the cardiac left ventricle.[1]

Gouty Hæmaturia.—In persons of gouty habit, over seventy years of age, sudden and severe vesical hæmorrhage may occur, and clots form in the bladder. No harm may result from this somewhat alarming symptom, which is better left untreated, attention being directed solely to the state of the bladder, from which the clots may have to be withdrawn by suction.

Prostatic Gout is occasionally met with. It affects elderly men who may have previously had no articular attacks. Severe pain may come on suddenly in the night, with pain and spasmodic dysuria. There may be associated pain in the groin, perineum, and testes. The urine is scanty and charged with urates. The prostate is found enlarged and very sensitive when examined by the rectum. The bladder is imperfectly emptied. The attack may subside and leave the prostate enlarged for some time subsequently with a condition of cystitis.[2] Exposure to cold and wet is often the determinant.

Gouty Dyspepsia.—It has been pointed out that some form of catarrhal dyspepsia is commonly associated with gout in its paroxysmal form. It may be acute, and be relieved by the onset of the attack, or it may persist after this is established. Retro-

[1] Under the care of Dr. Church. *Vide* Hosp. P.M. Book, February 16, 1889.

[2] Reginald Harrison on Prostatic Gout. Lancet, November 24, 1883, p. 896.

cedence of gouty symptoms from a joint to the stomach has also been discussed.

I refer now to the occurrence of gastric catarrh as a somewhat frequent phase of a gouty habit of body. The tendency is met with at an early age, and may persist from time to time throughout life in persons who do not develop any marked articular disturbance.

Pain, acidity, and flatulence are leading symptoms. The tongue is furred more or less, and the appetite may be impaired, or unchanged. In acute forms, cardialgia and vomiting may occur, and relief may follow the latter. Some dietetic indiscretion may induce this. In other cases, the catarrhal state appears as an irritative dyspepsia from time to time, and may, or may not, be dependent on habitual over-eating. The liver is probably in fault, and by periodic congestive states leads to portal venous plethora, and catarrh of the mucous surfaces drained by it. Such patients speak of their liver being "sluggish," meaning by this that they suffer from constipation.

Sometimes, craving for food is a symptom, and one which must not be gratified. Sense of sinking at the epigastrium may occur. Periodical attacks of gastrodynia with headache and vomiting of mucus have been known to yield to attacks of lumbago and sciatica.

The urine in cases of gouty dyspepsia is commonly loaded with lithates, and many of the symptoms of lithæmia may be met with. The fæces are apt to be pale, knotty, and scanty during the attack.

Patients thus affected are readily made worse by errors of diet, excess of any kind of food being harmful, even of simple articles, such as farinaceous matters. They are commonly sensitive to chill, and exposure to keen winds may determine a gastric attack in several ways. It may check the action of the skin, and, so, cause congestion of the internal organs at the same time that the appetite for food is heightened. Portal venous plethora and catarrh are thus readily provoked, and a so-called "bilious attack" is set up.

Gouty dyspepsia may, thus, be primarily of gastric or of hepatic origin, and the catarrhal variety is the most common. I have already discussed the spasmodic and the inflammatory forms of gastric and intestinal dyspepsia occasionally met with.

Gouty Neuritis.

The occurrence of gouty neuritis as a definite ailment has not been long recognized. It is a most troublesome and painful disorder. The symptoms have, no doubt, been commonly misinterpreted, and classed with vague gouty or rheumatic pains, or with neuralgia. In all cases there is a plain history of gouty ailments or heredity. The subjects are usually in middle life, and may, or may not, have had classical attacks of articular gout. The evidence of the gouty nature of this disorder is clinical and not pathological.

Mr. Hutchinson has noted cases of optic neuritis which he believed to be of gouty origin.

The symptoms are chiefly sensory in most cases, but motor affection has been also observed. Thus, numbness and tingling, "pins and needles" in an extremity, are the commonest symptoms, but the pain may be sometimes agonizing. Loss of power in the affected limb may also occur, and some muscular atrophy may result. I think it is not unlikely that some cases of neuralgia in the gouty are due to minor degrees of neuritis or perineuritis, since, with more or less constant pain, there are often severe paroxysms.

The most severe form constitutes a variety of sciatica. It is probable that the perineurium is affected by gouty inflammation, which leads to thickening and compression of the nerve-bundles. This can be plainly felt in some superficially placed nerves. Thus, I have met with it in the ulnar nerve above the elbow, a distinct tumour being felt, exquisitely painful on slight pressure, extending for a fourth of an inch or more along the nerve-trunk.

To determine the diagnosis in any case, there must be unequivocal evidence of gouty habit or concomitants, and the blood may be appealed to for evidence of increased amount of uric acid in it.

The occurrence of gouty neuritis is determined, as it appears to me, very much as are attacks of gouty phlebitis, and I have met with both affections in the same individual at different times.

The early peripheral tinglings much resemble those met with in alcoholic neuritis, but are usually less severe, and a complete study of peripheral neuritis must include this class of cases.

With the exception of the great sciatic nerve, the trouble is more prone to occur in branches of the brachial plexus. The

worst cases I have met with have involved this plexus, or some of its roots at their emergence in the lower cervical region. A degree of neuralgic character naturally pertains to neuritis, paroxysms of pain being apt to occur from time to time, readily induced by such movements, often very delicate, as disturb the affected branches mechanically. Pressure is badly borne, and will excite agonizing pain. A prominent feature of this trouble is its persistency and rebelliousness to treatment. I believe that a localized patch of gouty inflammation is the starting-point, specially affecting the perineurium, and leading to effusion into the nerve-sheaths. The adjacent lymph-spaces are probably involved, and these are possible sites of deposition of acid uratic salts. Some long period must elapse before these deposits can be removed.

With the nerve-changes come altered electrical reactions of the efferent fibres and the muscles supplied by them. Dr. Buzzard has directed attention to these in several cases.[1] Thus, in a lady, æt. fifty-two, the left hand would close during the night, and could not be opened without severe pain in the wrist and fingers. It would be found icy cold. Previously, there had been tinglings in both arms, and the toes of both feet would "go to sleep." Sometimes darting pains occurred down the arm and one finger. This patient had acid dyspepsia and flushings, bilious vomiting, and thick urine. The intrinsic muscles of the left thumb were less excitable than those of the right to induced electrical currents, and less so than those of a healthy person. This lady's father suffered badly from gout, and she had partaken rather freely of sherry, and, occasionally, of whisky.

In a man, æt. forty-seven, there was loss of power in the thumb and first two fingers of the left hand. There was inability to flex the phalanges of the thumb, index and middle fingers (except the first). The forearm had wasted. "Pins and needles" were constantly felt in the fingers, and the parts were hyperæsthetic to touch, pressure, heat, cold, and pain. There was defective reaction to faradic currents in the thumb-muscles. To the galvanic current applied to the musculo-spiral nerve above the elbow A.C.C. was = to K.C.C., and the opening contraction with the kathode was equal to that with the anode. The supinator longus muscle was unaffected. The nerves implicated were the branches of the median supplied to the palm and first three fingers, as well as to the opponens, abductor, and flexor brevis pollicis, the cutaneous branches of the radial distributed to the

[1] Paralysis from Peripheral Neuritis, p. 25, 1886.

dorsal surface of the thumb and two outer fingers, and the internal cutaneous branch of the musculo-spiral. There was no history of exposure to cold or pressure. The man's habits were believed to be conducive to gout.

The amyotrophy in these cases is probably of the reflex character peculiar to arthritic muscular atrophy.

Dr. Buzzard relates other cases, and in some there was clear history of gout, and relief from treatment directed accordingly.[1] In one case puffy swelling occurred in the arm, and some herpes.

In some of them the involvement of vaso-motor branches was plainly manifested, there being coldness and discoloration of the skin. The character of the disorder, and especially the electrical changes, point clearly to neuritis as the exact cause of the special symptoms.

Difficulty in diagnosis may occur in cases with history of alcoholic habits, where the prevailing conditions are such as to induce neuritis of either the gouty or the alcoholic variety. In my experience the latter cases occur more frequently in women under forty years of age who are not gouty, than in men.

I have known puffiness of the hand and glossy fingers to occur in these cases, together with marked lameness and clumsiness of the involved digits. Pye-Smith records an example which came on soon after an attack of gout involving the hands and feet, and which subsided without treatment.[2]

Saturnine neuritis, which is sometimes one-sided, may occur in connection with gout.

Gouty Phlebitis.

The characters of this disorder have been fully described, first by Sir James Paget[3] in 1866, afterwards by Sir Prescott Hewett[4] and Dr. Tuckwell.[5] Its pathological position is amongst the many forms of imperfect or incompletely developed gout.

The disorder very markedly occurs in persons of gouty heritage, or whose habits of life have led to the acquirement of the gouty state.

The veins of the lower extremities are most frequently involved, but those of the upper extremities, as high as the subclavian, may sometimes, though rarely, be affected. The saphena and its tri-

[1] Brit. Med. Journal, December 2, 1876.
[2] Fagge's Prin. and Prac. of Medicine, 2nd edit., vol. i. p. 432, 1888.
[3] St. Barth. Hosp. Reports, vol. ii., 1866, p. 82.
[4] Clin. Soc. Trans., vol. vi., 1873, p. xxxvii.
[5] St. Barth. Hosp. Reports, vol. x., 1874, p. 23.

butaries in the calves have, so far as I have seen examples, been more often implicated than any other branches. The trouble may begin quietly, or sometimes with pain in the part, which may continue more or less severe for weeks or months, or a little itching or uneasiness may be all that is experienced, and, on examination, a cord-like hardness is found already established.

There is a tendency for the inflammatory process to spread along the branch first affected, also for other veins to be affected at some distance, so that there is sometimes present what may be termed patchy phlebitis. In one case of this kind under my care, there was excruciating pain with each fresh attack. I regard the trouble as a localized form of specific gouty inflammation, affecting the coats of the vein, leading to roughening of the internal lining, and so favouring thrombosis. The condition of the system at the time is essentially gouty, and with this there is hyperinosis or ready tendency for the blood to clot. There may be assumed to be a determination of acid urates to the part as the directly exciting cause. Other gouty manifestations may be associated with the local change, and acute gout may have already occurred, or may follow at later periods.

Traumatism plays a part in some cases in bringing on this trouble, and trifling provocation may suffice to determine the part affected. Blows, continued friction, or undue muscular exertion may thus be causes. Exposure to cold after being heated has been alleged as an excitant, but I have no experience of this.

The most serious cases are those in which a large vein is involved, such as the femoral or the subclavian.

With ordinary care the involvement of small vessels is of little moment. Superficial veins are more apt to suffer than those situated deeply, probably because more exposed to injuries, and no appreciable dropsy is observed in these cases. In the event of the deeper and larger veins being involved, considerable œdema (œdema durum) of the parts below ensues on the establishment of thrombosis, the limb being pale, with dilatation of the superficial veins.

Renewed attacks in other veins may follow, and relapses are unfortunately frequent on trifling provocation. One of the commonest causes of relapse is a too early return to use of the affected limb.

The gravest risk in any case, but especially in those implicating the deep and larger veins, is that of detachment of the thrombus or part of it, and its carriage into the pulmonary artery

or one of the lungs. Several fatal cases from this accident are on record; but they, happily, do not often occur if strict precautions are maintained throughout the progress of the disorder. The clot itself is apt to be very firm, and also intimately attached to the venous wall.

On inspection, there may be seen, at first, slight redness or blush over the affected vein. Deeper veins, if blocked, may not be palpable, but pain or œdema, or both, declare the nature of the disturbance. The ordinary termination is by resolution, with, presumably, tunnelling of the clot, a process occupying from two to three months, or much longer, or by occlusion of the venous branch. In some other forms of phlebitis, such as are due to traumatism, for instance, an earlier restoration to a healthy state may be expected. Suppuration does not occur, this result being infinitely rare as the outcome of gouty inflammatory process in any part. If occlusion of an important branch occurs, the limb thereafter remains slightly puffy and heavy, with compensatory enlargement of superficial veins.

Recurrent Phlebitis.—When superficial veins are involved, they are very apt to be attacked again, and often remain tender and sensitive. I have known the same branch, a tributary of the saphena, to be affected five or six times at varying intervals, friction from stirrup-leathers while riding, and hard walking, being the direct excitants of the attacks. Recurrence of phlebitis is very common.

Women are rarely the subjects of gouty phlebitis, though in cases of atonic and incomplete gout they may suffer from a recurrent form of it. Paget quotes the experience of Sir Charles Locock in respect of four sisters who had phlegmasia dolens, and whose father had crural phlebitis.

This manifestation of gout is believed by Paget to be of comparatively recent occurrence. Sir Henry Halford described cases of what he called phlegmasia dolens in the male, due to inflammation of veins of the pelvis.[1] Paget believes that a disorder so obvious as phlebitis could hardly have escaped recognition and description, had it been as commonly met with as now. "So we may believe," he states, "that the disease has become more frequent in the last fifty years, and may suspect that not long before Sir Henry Halford's time it may have been a really new disease." He conceives that this disease is "amongst the instances of the results of morbid conditions changing and combining in

[1] Paper read before Roy. Coll. of Physicians, April 1832. Essays and Orations, 3rd edit., Lond., 1842. Essay viii., p. 121.

transmission from parents to offspring."[1] He regards it, therefore, as a new modification, outcome, or transformation of gout.

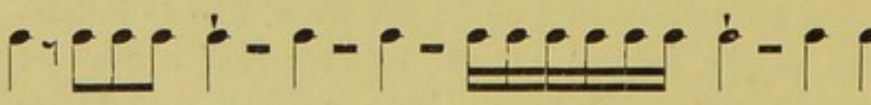
Dr. Edward Liveing has kindly given me the following notes of two important cases, which well-illustrate this form of phlebitis, and afford convincing evidence, if such were needed, of the truly gouty nature of it.

CASE I.—A. C. was the rector of a country parish, a middle-aged bachelor, living a very retired, routine life. He was of stout build, tending to corpulency; was accustomed to much standing about out of doors, but little active exercise; was moderate, but not abstemious in his habits. This gentleman belonged to a gouty stock, his mother and maternal uncles developing some of the many forms of the malady, though of a mild kind. One of his brothers suffered from granular kidney with great cardiac hypertrophy, and ultimately fatal cerebral hæmorrhage; several of his nephews and great-nephews from mild arthritic attacks, gouty nodules, severe migraines, and other forms of irregular gout. One of them is the subject of the second case.

A. C. enjoyed fairly good health until past fifty years of age, having rarely missed his duty. He had been in the habit of taking pretty frequent doses of blue pill and colocynth "for his liver," as he said. He never had a typical attack of acute gout in foot or hand, but when about fifty-five began to suffer from subacute attacks in his heels and ankles and the tendons about them, leading in the end to much thickening and enlargement, and reducing his exercise still further. He was also greatly troubled by a general gouty pruritus, without visible eruption, and only relieved by brushing.

When about sixty-five he was suddenly attacked with phlebitis or phlegmasia dolens in the right leg. The deeper veins were involved, and there was great enlargement of the whole limb, of india-rubber-like consistence, and in every way characteristic of the gouty form. While he was still confined to bed, the left limb was similarly attacked. The whole character and course of the malady were quite typical, and recalled the then recent, but now classical, description in Sir James Paget's Essays. Recovery was slow, and the limbs never regained their proper size, and walking was more difficult than ever. It was nearly six months before he could resume his duty.

At or about the time of this attack attention was drawn to the state of the pulse, which had become extremely irregular. Before that it had been slow, soft, full; it may have been occasionally intermittent, but nothing more. Tension was low, and no rigidity of vessels. But from this time the derangement of rhythm was most extraordinary, and may perhaps be rudely represented thus:—

 and so on.

This continued with some variation to the end of his life, some ten years later. Meanwhile he had no particular illnesses, and, except for occasional attacks of faintness and one or two severe nose-bleedings, which alarmed him, and feebleness, he was fairly well. There was never a trace of albumen in the urine nor any renal trouble. The cardiac impulse was feeble, diffused, and often difficult to fix, and the sounds were feeble and very much alike. There was never any bruit audible. We suspected a flabby thin left ventricle.

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[1] "On Some Rare and New Diseases," Bradshaw Lecture, Roy. Coll. of Surg., 1882, p. 13.

He did his duty to the end, though with considerable difficulty, owing to the unwieldy state of his legs and the tendency of the skin to inflame.

Without warning, the circulation was suddenly arrested in the left leg, to such an extent that it became quickly gangrenous up to the knee. About a week later he had a slight cerebral seizure—some twitching of the face, followed by a coma-like sleep, from which he awoke with embarrassed speech and weakness of the right side. The following week he died without fresh symptoms.

CASE II. is that of B. D., a nephew of the former, and also a country clergyman. He was of a very different build and temperament from his uncle, being tall and spare, and soured by ill-health. From his college-days he had suffered from persistent dyspepsia, with exacerbations from time to time so severe as to make feeding difficult. He was consequently compelled to, and did, live for thirty-five years a very abstemious life. Alcohol in any form and fruit and vegetables were literally poisonous to him, and so were all made dishes and sweets, and indeed everything but the plainest food. His diet was one of bread and some form of animal food, chiefly mutton, with milk and rice and similar puddings. His malady was very much what has been described, with doubtful propriety, as "eczema of the stomach." He became a martyr to physic, orthodox and otherwise, in the hope of relief, which he never obtained.

When about the age of thirty-eight, he gave up his living on inheriting a country estate, and, hoping the change might do him good, he lived for nearly thirty years an outdoor agricultural life, but, unfortunately, with little benefit.

I remember his consulting me some years later, during one of the longest and severest of his bouts of illness. He then presented very much the aspect of a patient with a malignant disease of the stomach—greatly emaciated, features nipped, and complexion sallow, with much gastric pain and distress. I had my suspicions, but kept them to myself. In time he got better, and recovered his usual amount of health.

When about the age of fifty-six, he developed eczema of both palms, and to a less extent of the groins, which continued to trouble him for many years. But from this time the gastric troubles greatly abated, and for the last ten years of his life he was able to take wine and other stimulants, and a much greater variety of food, including fruit and vegetables.

Among the occurrences of this period, I remember a temporary goitre, which lasted a year or more, and transient prostatic troubles. There was never any albumen or renal affection.

When about the age of sixty-six, severe inflammation was set up in the second toe of one foot, apparently from so trivial a cause as cutting a corn. For a time the toe threatened to slough, but ultimately healed, though not very soundly.

He was now suddenly attacked with gouty phlebitis, and the characteristic enlargement of the legs, very much as his uncle had been, though less severely. He was in bed many weeks and recovered; but though the swelling of the legs went down, they remained almost useless to him, and he had to be carried up and down stairs, and lifted in and out of his chaise.

During this illness my attention was drawn to the state of the pulse, which had become very nearly as irregular and intermittent as his uncle's, and without any indications of heart-disease except feebleness. A troublesome ulcer now formed over one ankle, which his medical attendant called gouty, and which would not heal.

The following winter, while driving out as usual, he was supposed to have had one foot chilled, for the great-toe became gangrenous; and in the course of the next two months the gangrene extended to the leg, and brought him to his end.

Gangrene.—In cases of chronic gout, with much debility and feeble circulation, gangrene of the extremities may occur. It is

a rare condition, and the direct association with gout is hardly demonstrable. The foot is commonly the part affected, but both feet may suffer simultaneously. The form of gangrene is dry. Carmichael, of Dublin, recorded an example in a gentleman between sixty and seventy years of age, subject to gout.[1] The gangrene spread up from the left second toe to below the knee. Gasping respiration set in and delirium. The pulse was irregular, and none was felt in the iliac artery of the affected side. Later, none was detected in that of the opposite side. At the autopsy the heart was found softened and fatty, aortic valves slightly ossified. The left iliac artery was plugged with a fibrinous clot, which was breaking down. Both femoral arteries were also sealed by fibrin. The saphena, iliac, and renal veins were blocked by coagula. The order of events in cases of this nature is as follows:—A chronic gouty state leading to cachexia, with degenerative changes in the heart and arteries, induces at last such a feeble state of circulation that arterial and venous thrombosis sets in; or, from valvular degenerations, fibrinous emboli may be shed into distant arteries. In such cases the kidneys are presumably unsound from chronic interstitial nephritis. In other cases there may have long been glycosuria connected with the gouty habit, under which circumstances gangrene is somewhat prone to occur. Arterial atheroma is not always present in marked degree in these cases.

Orbital Cellulitis and Suppuration of the Eyeball in Persons of Gouty Habit.

Two cases of relapsing double orbital cellulitis of probable gouty origin have been recorded by Mr. Nettleship.[2] In one the patient was a man aged forty-four years. He was very gouty, and had suffered from two attacks annually for twelve years. Seven years previously he had had chemosis and acute orbital periostitis. In the other case, also in a man aged thirty-five years, there was double orbital cellulitis, temporary iridoplegia, and amblyopia. His father was a painter, and never free from gout. There was no proof of syphilis.

The symptoms were symmetrical, including proptosis, redness and swelling of ocular conjunctivæ and eyelids. The right side was the more involved. The affection had lasted for three weeks.

[1] Dublin Med. Journal, vol. ii., 1846, p. 233.

[2] St. Thomas's Hospital Reports, vol. xi. p. 9, 1882.

The roof of each orbit was thickened. Vision was impaired, whether from failure of accommodation or from disease of the optic nerves was not determined. Mr. Nettleship believed that the ciliary nerves were involved. There was perfect recovery. Mr. Critchett described an instance of suppuration of the eyeball, which occurred in a case of chronic gout in a man.[1]

Gouty Amyotrophy.

Amongst the local disorders due to gouty arthritis when of long duration is some degree of muscular atrophy. This implication of the muscles is not peculiar to gout, but is well-recognized as a result of chronic arthritis from any cause.

Minor degrees of wasting occur in connection with paroxysmal gout of short duration, but are hardly recognized till several attacks have involved a joint. They are naturally most marked in the case of the knee, wrist, and elbow. The causation is the same in all cases, a reflex atrophy due to changes in the nerve-centres, which are irritated by the painful state of the nerves supplying the affected joint. The painful sensory, centripetal, impressions modify the trophic conditions of the centre, and lead, reflexly, to impaired nutrition of the associated muscles of the part.

Mere disease of the joints is insufficient of itself to induce the degree of muscular wasting which occurs. The gravity and permanence of the atrophy is accurately determined by that of the articular trouble, and if the latter is amenable to treatment, the wasting may be recovered from, sometimes completely.

This condition of arthritic amyotrophy may follow on earlier muscular involvement, manifesting itself in disturbance of motor influences, which, by inducing reflex spastic states, may lead to deflections of digits and other parts, as already explained in an earlier chapter.

A well-marked example of muscular wasting consecutive to acute articular gout is recorded by M. Cornillon, of Vichy.[2] The patient was a male, æt. fifty-five, who suffered from several attacks of biliary colic. He had had acute gout in the right shoulder and wrist two winters in succession, suffering extreme pain, and consequent immobility of the limb. The feet were not affected. There was a tophus in the right ear, atrophy of the deltoid, extensors of the fore-arm, thenar and hypothenar eminences,

[1] Medical Times, vol. i. p. 62, 1858.

[2] *Progrès Médical*, Mai 26, 1883, p. 105.

and interossei muscles. The hand was griffon-like. There was diminished contractility to galvanism in many of the affected muscles. Pain was felt at the level of the seventh cervical vertebra.

The late Dr. Theophilus Thompson regarded as a distinctly gouty manifestation a case of progressive muscular atrophy in one son of a family in which the mother was severely affected with chronic rheumatic arthritis, and seven brothers were typically gouty. I have no knowledge of any similar case, and no systematic author has noted this connexion.

CHAPTER XIV.

ON THE PROPRIETY OF SURGICAL OPERATIONS ON THE GOUTY.

The fitness of a gouty subject for operative interference is to be determined by consideration of the degree in which his textures suffer from the degenerative changes induced by the dyscrasia. Where the heart and kidneys are unsound, it would be unwise to urge any operation that was not imperatively called for. Fits of gout may be brought on by operations as by other forms of traumatism, and they exercise a temporary malign influence on the healing process, but hardly more than this. Fractures of bone may be delayed in uniting, the integuments may ulcerate and the bones be laid bare, but all these troubles subside with the subsidence of the local gout, and the after-progress is commonly satisfactory. The operation for cataract is apt to fail or do badly in the gouty. Glycosuria is an unfavourable condition for surgical interference, especially if pronounced or of long duration. Incoercible hæmorrhage may result in such cases. On the whole, it may be laid down that, as a rule, none but operations of necessity should be performed on the subjects of marked gouty habit. This caution applies to minor operations, such as removal of wens and fatty tumours, to ligature of veins and piles, and even to puncturation; and it is necessary, too, in spite of the practice of all antiseptic precautions, which may avail little or nothing in such cases.

There is increased vulnerability in the subjects of chronic gout, and my own experience leads me to state that even trifling injuries may, sometimes, induce erysipelas, destructive cellulitis, and gangrene. A caution against *nimia diligentia* may be sometimes as necessary for *chirurgus* as for *medicus*, and truly, in many phases of disease, abstention from interference is more important than any line of action.

CHAPTER XV.

ON SOME DISORDERS SIMULATING ACUTE GOUT.

Of these, I will refer to three.

Pyæmic Arthritis.—Pyæmic arthritis has before now been mistaken for uratic arthritis. The error is quite pardonable in the first instance, especially if the patient has already suffered from gout. The diagnosis is not long undisturbed in such a case. Suppuration, which is the rarest event in gout, rapidly follows tumefaction, and, if care be taken, the original site of infectivity may not be far to seek. Any recent operation, as for piles, or the existence of otitis, may lead to detection of the source of the arthritis.

Acute Necrosis of Bone.—Acute necrosis of bone may set in suddenly sometimes in elderly people, and there may be a history of gout in the case. The tibia, fibula, or humerus may be the site of redness, swelling, and pain, and, so, closely simulate an acute attack of gout. Œdema may ensue, but fluctuation becomes detectible, and sub-periosteal abscess is to be diagnosticated. In the more frequently occurring cases of acute necrosis and suppurative epiphysitis in growing boys, the diagnosis of acute gout is hardly likely to be made.

Gonorrhœal Rheumatism.—Gonorrhœal rheumatism, involving one joint, not unfrequently simulates gouty arthritis. The history and course of the disorder guide to a correct diagnosis. The likeness here is the more interesting because there is commonly a history of gouty tendency, and patients with this proclivity are specially vulnerable to gonorrhœal poison. There may, therefore, be a gouty element in the case demanding recognition. The inadequacy of sodium salicylate to afford relief, and the benefit derivable from potassium iodide, quinine, and colchicum in some cases, after treating the urethritis, are also significant and noteworthy.

CHAPTER XVI.

SKIN-DISEASES IN CONNECTION WITH GOUT.

SEVERAL varieties of skin-disease are now well-recognized as dependent on a gouty habit. Galen observed this connection.

Pruritus—P. Hyemalis—P. Ani—P. Vulvæ.—Pruritus is noted with some frequency. There may be no visible lesion associated with it. The form known as pruritus hyemalis is, I believe, sometimes connected with gouty predisposition, and anal itching is often thus associated. The latter may, or may not, be connected with a hæmorrhoidal state. Alternation of pruritus with articular gout has been noted. Vulvar pruritus is common in women suffering from gouty glycosuria, and may be one of the leading symptoms calling attention to this state.

I have known chronic general pruritus yield completely on the supervention of gouty cystitis. This occurred in an elderly gentleman, who was fond of strong soups, and drank a good deal of sherry.

Prurigo.—Prurigo is sometimes the outcome of pruritus. In this case papular lesions are induced by long-continued scratching. In the old, care must be taken to exclude phthiriasis. The pruritus is primarily neurotic, and due, probably, to urichæmia. It gives rise at times to intolerable suffering, and local means are commonly powerless to afford relief.

The papules of prurigo are sometimes, and not unfairly, regarded as truly lichenous. Uncovered parts of the body are not, as a rule, affected.

Acne.—Acne has been alleged by some authors to be sometimes associated with a gouty state, but I have no experience of such cases. Garrod records a case where alternation with articular gout occurred.

Furunculi—Anthrax.—Furuncles and carbuncles sometimes occur in gouty persons, the former in association with glycosuria, but

not, in my experience, very often. The nape and lower lip may be seats of carbuncle.

Pemphigus.—Pemphigus is probably never, or most rarely, a manifestation of gout. Uric acid has, however, been found in the serum drawn from the bullæ in several instances.

Psoriasis.—Psoriasis is sometimes met with in gouty persons, and may alternate with articular attacks. Garrod, as the result of special study of the subject, declares that psoriasis is not connected with gout, as is eczema. Cases, however, do occur in which alternations of articular gout, psoriasis, and bronchitis are manifest.[1] Garrod found that psoriasis was more frequently associated with chronic rheumatic arthritis, and that successful treatment of the joints relieved the skin-disorder. I am prepared to affirm that the majority of cases of psoriasis seen in practice manifest no direct connection with a gouty habit, but some are distinctly so related.[2]

I believe that, sometimes, patches of dry eczema have been mistaken for those of psoriasis, and, hence, the latter may have been unwarrantably credited with a dependence on gout.

Dermatitis Exfoliativa.—This disorder, also known as pityriasis rubra, is apt to occur in members of arthritically disposed families. It may be developed from psoriasis, and cases of the latter may become examples of general pityriasis rubra. True gout figures in the ætiology of this disorder, perhaps in one-fifth of the cases, and it may alternate with attacks of it.

Eczema.—Eczema is, without doubt, the type of skin-disease most directly connected with the gouty habit. This is true of all forms of the disorder, and to find the true relationship here, it is necessary to be familiar with the many phases of eczema. Garrod's researches affirm most pointedly the intimate connection between gout and eczema.[3] He found that the latter was prone to affect the following parts, in the order of frequency mentioned: the ears externally, within the meatus and behind the auricle, the nape of the neck, eyelids and face, groins, and flexures of other joints, scrotum, glans penis, and prepuce, backs of hands and feet, interdigital surfaces, arms and legs, and various portions of the trunk. It is commonly symmetrical on both sides of the body.

Garrod found that eczema may precede overt gouty attacks by

[1] Greenhow has reported several examples. *Op. cit.*, p. 148.

[2] I have notes of two cases in which gout with tophi in the ears occurred in men long subject to typical psoriasis, and of one in a woman with tophaceous gout, who died of carcinoma of the liver, following the same disease in the mamma. She had psoriasis guttata.

[3] Trans. Internat. Med. Congress, London, 1881, p. 102.

many years, and attack patients whose whole history was gouty, but who had never suffered from arthritis; also, that it may occur late in a gouty life, even in extreme old age, when fits of ordinary gout have become much less frequent and less severe.

Acute eczema may replace an articular attack. In gouty families, a parent may have arthritis, one son ordinary gout, and another eczema without arthritis. It may attack the females in such families about the menopause, true gout supervening after that period.

Garrod believes that eczema occurs in about thirty per cent. of cases of gout of long duration.

The disorder in its acute stage is apt to cause much distress and suffering from extreme itching and burning sensations. In the chronic, dry form, the itching is sometimes very severe, indiscretions in diet quickly arousing great irritability in the patches. Urticaria may be occasionally associated with it, adding much to the torment.

Dry patches of eczema in goutily disposed persons may remain present for weeks in a quiescent state. I have notes of a case of incomplete gout where this condition occurred, and irregular action of the heart supervened. When the eczema became active and recommenced to itch, the heart became regular in its action. Beyond the irregular cardiac rhythm there were no other troubles of the circulation, and while it lasted active exercise was possible without any inconvenience. This alternation was observed on several occasions. Severe attacks are apt to occur in spring-time, and exposure to cold north-east winds may provoke them.

Metastasis of gouty eczema has been suspected with good reason in cases where asthma or acute cystitis have supervened on its disappearance, constituting, in the language of the French school, a veritable enanthem.[1]

There is a great tendency for gouty eczema to recur. I have known it come out annually in symmetrical fashion on both fore-arms. In another case it returned five times at intervals of two years, lasting about two months on each occasion, gouty attacks occurring in the intervals.

[1] "I saw once a gentleman of an active, stout habit of body, who, having applied a piece of ice cut to the shape of his gouty foot, rid himself indeed very soon of the pain, but in a little after, a filthy herpes broke out all over his face, and taking possession of his very eyelids, occasioned the greatest uneasiness, from which I had scarcely got him free, when a sharp fit of the gout quickly succeeded."—*van Swieten's Comment. on Boerhaave's Aphorisms*, vol. xiii. p. 160.

I think this was probably an attack of eczema induced by gouty retrocedence from the foot.

In saturnine gout cutaneous manifestations are rarely met with, possibly because of the associated spanæmia and diminished nervous energy in such cases.

Urticaria.—Urticaria is sometimes markedly dependent on the gouty habit. It may precede, by shorter or longer periods, a paroxysmal attack of gout. In the same patient either gout or urticaria may supervene after errors of diet. Uric acid is probably the direct excitant, acting as bile and other irritants are apt to do in inducing this disorder. Urticaria and eczema may co-exist at the same time in gouty persons.

There is nothing specific in the form of urticaria associated with gout. The fugitive and persistent varieties of it may occur, and so may the metastases recognized as peculiar to it.

The relation of erysipelas to the gouty habit has been already discussed in Chapter ix., p. 209.

Herpes—H. Zoster.—Herpetic attacks, in all varieties, are common. Shingles has been noted in cases of gouty glycosuria. Labial herpes is especially frequent in the simple catarrhal states of those goutily disposed. Parts the site of former injuries may become subject to recurring herpetic attacks, and these may occur as a form of retrocedent gout on exposure to cold and damp before an articular attack has completely subsided. Shingles may co-exist with paroxysmal gout. The severest form of herpes—H. ophthalmicus or H. frontalis—I have not met with in association with gout. Cases are recorded where it occurred in "rheumatic" subjects.

Fugitive Gouty Inflammation.—Graves records a remarkable case of fugitive inflammation which occurred in a man who had had various phases of gout, including gastric attacks. On the cessation of the latter the face began to swell at various points, beginning at the forehead, spreading to the cheek and eyelid, so as to close up the latter, and to the lips, the nose never being affected. These tumours appeared on other parts of the body. The left side was chiefly involved, and dryness occurred in the nostril of that side. In a few hours these tumours subsided, no trace of them being found the following day. The patient believed that they sometimes occurred in the stomach; and the mouth, palate, and uvula were occasionally attacked. The symptoms described would lead to the belief that the nature of the disorder was very closely allied to urticaria. There were sensory symptoms, expressed by a feeling as if a current of air was directed on the face, then a sense as of a fillip of the finger, or the bite of a gnat, on the part, which soon assumed the character of a bump.

Subcutaneous Nodules.—These are certainly met with most commonly in association with rheumatic manifestations. I have recorded several instances of these.[1]

In a case of saturnine gout in a woman, I witnessed the formation of many small nodules over the tibiæ,[2] and in a case of chronic gout, also in a woman,[3] small nodules appeared over the left tibia, movable, unattached to the periosteum, and grating when rubbed against the bone.

It is noteworthy that while in the rheumatic cases the occurrence of these nodules uniformly betokens a slowly progressive cardiac valvulitis, in neither of these (gouty) cases was there any such disease.

Æstus volaticus.—Flushing of the face in paroxysms is recorded by Graves in the case of an elderly gouty lady. The attacks came on daily at three o'clock, the nose becoming hot, bright red, and, later, purple, the redness spreading to the cheeks, accompanied with uneasiness but not with pain. This always passed off about the same hour in the evening. Minor attacks of this disorder, which represent a vaso-motor neurosis of the skin, are termed *æstus volaticus*.

Xanthoma.—Mr. Hutchinson has recorded a case of xanthoma which occurred in a Hebrew, æt. forty-four, a man of dark complexion, who lived freely. He inherited gout, and had been subject to attacks of it for twenty years. His father and maternal grandfather had suffered, like himself, from xanthoma of the eyelids.

In this case, the patches followed an attack of jaundice produced eighteen years previously by severe fright. There were numerous painless and symmetrical enlargements of many tendons, also bursal enlargements. Over a swelling on the right olecranon were streaks of xanthoma. Exostoses (lipping) were also present on the ulnæ and tibiæ. No uratic tophi were detected.[4]

Xerodermia.—I have notes of a case of xerodermia which occurred in a boy aged ten years, whose maternal grandfather had "chalky" gout. The mother presented several plain indications of the arthritic diathesis. I am not aware that any connexion has ever been made out between gouty inheritance and this form of skin-disorder, and simply record the case as one of interest to future observers. I have related another example on

[1] Clin. Soc. Trans., vol. xvi., 1883.
[2] Clin. Soc. Trans., vol. xx., 1887, and p. 170 of this treatise.
[3] *Vide* Fig. 13, p. 81, depicting the fingers of this woman.
[4] Lancet, April 20, 1889.

p. 400, where xerodermia occurred in a case of diabetes in the son of a gouty man. A brother of the patient also suffered from the same cutaneous disease.

The implication of the skin in cases of gouty habit is, I conceive, determined by some inherent weakness or special predisposition in the part. Were this not so, skin-affections would probably be even more common than they are in the gouty.

Some persons appear to enjoy immunity from any form of dermatitis, whatever be the constitutional provocation, while others are only too prone to be thus affected.

CHAPTER XVII.

GOUT IN WOMEN, IN EARLY AND IN ADVANCED LIFE.

"THE arthritic diathesis and cachexia, as manifested in woman, have never received the attention they deserve." This was declared five-and-twenty years ago by Laycock, and he believed the omission arose from a fundamental mistake in pathology, viz., that they occurred but seldom in the sex. It is questionable whether gouty manifestations are now more common in women than was formerly the case. The late Sir Robert Christison told me he thought women manifested more gout in his time than in that of Gregory, and he believed the cause to be that women lived more highly than formerly. In the height of luxury in Rome under the Empire, women suffered from gout.

It is certain that women occasionally present all the symptoms of classical and paroxysmal gout, but such cases are rare. Young women may suffer in this way. I have known of cases occurring under twenty years of age, but they are infinitely rare before that period. W. Gairdner declared that he had met repeated instances of paroxysmal gout in very young girls. Hippocrates noted the immunity from gout in respect of this sex:—*Γυνὴ ου ποδαγριᾶ, ἢν μὴ τὰ καταμήνια αὐτέη ἐκλίπη*;[1] but facts do not support the doctrine thus laid down.

The following case was well-marked:—

A. M. W., æt. twenty-four, single, never very robust. Dark-haired, somewhat sallow and lean. Father healthy and active. Mother, a Swiss, under my care some years previously with Graves' disease, of which she died. Both parents and patient strictly temperate. Paternal aunt crippled with alleged "rheumatic gout." Came on February 23, 1887, with pain in left metacarpo-phalangeal and phalangeal joints of left forefinger. Had suffered much during the winter from chilblains on hands and ears, also, occasionally, from other erythematous eruptions.

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[1] *Αφορισμοι, τμήμα ἕκτον*, 29. Cullen referred to this, and denied the truth of it.

Ctma. regular. The feet have been tender and weak, and the right great toe-joint aching. On the 26th, after taking some iodide of potassium and iron, pain set in in right great toe-joint of tight, bursting character. The hand was relieved. Rapid improvement under colchicum and magnesium carbonate. Subsequently, quinine and iodide of potassium were given, and occasional doses of blue and compound colocynth and henbane pill.

This was a case of acute but atonic gout. The pulse was soft and the tongue clean and flabby. It is not unlikely that the iron given at first helped to determine the attack.

Tophaceous gout in women is exceedingly rare. I do not remember to have seen more than four or five well-marked cases. Tophi in the ears of women are also very seldom met with. The following case was one of the worst I have seen :—

A. H., æt. thirty-eight, admitted under my care in Elizabeth Ward, May to August, 1888. Fair-haired, slender woman. Married, and has a family. No serious illnesses. Not exposed to lead influence. No history of gout in her family is obtainable. At the age of twenty years had first attack of gout in right great toe-joint, and has had many subsequent attacks, generally three in each year. Was subject to "bilious sick headache" for three years before onset of gout, the attacks having occurred monthly. There is much uratic deposit in the hands, one mass being as large as a walnut, and some in the helices of the ears. There has been some in the toes, which has, she states, been discharged. There is a history of whisky- and rum-drinking.

Dr. Haig has reported this case in respect of uric acid excretion under the influence of certain drugs.[1]

The influence of the catamenia as a measure of systemic depuration has, no doubt, much to do with the differences met with in gout in the two sexes. It is certainly the case that the disorder appears in women soon after the menopause, and menorrhagia has been noted as not an infrequent occurrence before the cessation of the menses in goutily disposed habits. A hæmorrhagic tendency pertains to the daughters of gouty men (not reaching the grade of hæmophilia), whence epistaxis, occasional menorrhagia, and hæmorrhoidal flux. Such overflow, when not so excessive as to induce debility and severe anæmia, may prove depurative, and thus avert gouty paroxysms.

The manifestations of gout in women are commonly of the incomplete, asthenic, or irregular varieties. Women suffer rather from goutiness than from gout. Many of these cases are roughly included in the category of "rheumatic gout," so called, and are sometimes mistaken for chronic rheumatic arthritis. Many joints may be involved. The hands display the changes most markedly.

[1] St. Barth. Hosp. Reports, vol. xxiv., 1888, p. 217.

As I have already remarked, some of the varieties of Heberden's nodes are the result of chronic gout in women. The digits are distorted variously, knotty at the joints, with axial displacements, especially affecting the terminal phalanges, which may be everted or inverted in respect of an imaginary mesial line. The nails are usually fluted or coarsely striated. The whole hand thus becomes enlarged, and larger gloves are required. Burning pains occur in the phalanges, and the small crab's-eye cysts already described may form over the terminal nodes. (*Vide* Fig. 13, p. 81.)

Pains in the feet are often felt, achings and burnings, especially of the soles, and this is apt to be troublesome at night. Dietetic indiscretions readily aggravate these.

These signs may occur in the third decade, especially if there be strongly marked heredity; but they are more frequent in the fifth and sixth decades.

Such women are sometimes of robust constitution, but the general health may be feeble in other cases, and many anomalous and wearying pains in the limbs and trunk may be associated with this phase of goutiness. Such troubles are often called by the lower orders "rheumatics." The amount of real crippling is small as compared with that induced by chronic rheumatic arthritis. Glycosuria may be also associated with this state; so may varieties of eczema, vertigo, cramps in the legs, various dyæsthesiæ, deafness, tinnitus, mental irritability, heats, flushings, acid dyspepsia and hepatalgia. All these troubles are recognized as manifestations of goutiness generally.[1] Anti-gouty medication and regimen mostly relieve them, and removal to a dry mild climate in winter is very beneficial.

Failing to find evidence of uratic deposit or history of overt arthritis, the clinical purist may hesitate to make a diagnosis of gout in such cases. Family history and the clinical phenomena must, however, suffice to decide the line of treatment. Pregnancy is sometimes determinant of more or less widely spread subacute arthritis in women of gouty heritage. After delivery, the articular symptoms may completely subside, but other incomplete gouty ailments are apt to arise from time to time, such as bronchitis with noisy laryngeal cough, teasing eczematous eruptions, glycosuria, joint-pains, and plantar causalgia. Any of these ailments plainly proclaim the dominant diathetic state, and as plainly indicate the treatment proper for it.

[1] "The morbid localizations are nothing more than manifestations of one general dominant tendency."—*Trousseau.*

Gout in Infancy and Early Life.

W. Gairdner reported the occurrence of fits of painful gout in infants at the breast, and believed that the pain alone in one case was the cause of death. He quotes observations of Morgagni to the same effect, ancestral gout forming the essential factor in the history. I have never met with such cases, but have witnessed some about the first climacteric period.

Gout occurring in young persons may be fairly set down to hereditariness, and, according to Laycock, is significant of a feeble constitution.

The prognosis of gout is much more grave when it comes out early in life with history of heredity. For purposes of life-assurance, such cases, if accepted, should be heavily weighted. The onset of gout after the fourth or fifth decade of life is of much less importance in this respect. Laycock noted that gouty male children were often fond of meat, while girls thus impressed were apt to loathe it, and suffered from what he termed kreatic nausea.

Sir Henry Pitman has given me the following particulars of a well-marked case of acute gout in the great toe-joint, which occurred in a boy at the age of eleven years. Heredity was traced to a grandfather. There was present at the time of the attack an enlarged scrofulous gland in the neck. The patient is still living, about fifty years of age, and has been subject to attacks of gout from time to time. At Cambridge he drank, not immoderately, of college ale, which is a sufficiently gout-provoking beverage.

Tonsillitis (quinsy), enlarged tonsils, granular states of the pharynx, and catarrhal conditions of the throat and respiratory mucous membranes are not infrequent expressions of gouty inheritance in children. Tendency to hepatic congestion with "biliousness" and pale stools are also thus recognized, and such attacks recur from time to time, reminding one of the like "growing up" of regular gout in adults. Gastro-enteric catarrh may occur, and loaded states of the urine. There is also tendency to various skin-disorders and to herpetic outbreaks, as has been already mentioned.

In one case, within my knowledge, a very temperate gentleman had minor gouty troubles all his life, but never had an acute attack till the age of eighty-six, when he was on his death-bed.

In persons of vigorous constitution, attacks may continue to

come on at intervals in advanced life with but little general disturbance to the system, and not calling for any very active treatment.

Gout in Advanced Life.

Instances are not uncommon where the first overt attack of paroxysmal gout has occurred at or after the assigned limit of human life. I have knowledge of several instances where the patient had reached eighty years of age before a first acute attack appeared. Scudamore had no experience of such a case after sixty-six years of age. Garrod records a first attack in a lady of ninety-one, and relates the case of a Bishop of Durham who thus suffered at the age of ninety, having been lithotomized when twelve years old. He lived to ninety-two.

Although in these cases the attack of frank gout has been remarkably delayed, it is certainly true, for some of them, that many minor tokens of the gouty habit had occurred in previous years, so that a paroxysm might sooner or later have been predicted.

In the aged it has been observed that any unusual shock, mental or traumatic, sufficient to disturb the balance of healthy nutrition, has been determinant of a gouty paroxysm. Passage of uratic gravel and calculi sometimes occurs in elderly gouty men, with relief to articular attacks; and temporary glycosuria may occasionally replace gouty fits.

Incidence of Gout upon Particular Members of Families.

It has been observed that the younger children of gouty parents are sometimes more prone to develop the disease than the elder ones. This is explained by the fact that the parents are apt to be more gouty with advancing years, and, so, more liable to pass on the disorder to their later offspring. The influence of atavism sometimes only comes out with later procreation. A parent with gouty proclivity may die before overt gout appears, who would, had he lived, have perhaps manifested it later in life.

The habits of individual members of families must, of course, be taken into account in considering the incidence of the disease on particular sons or daughters. The variety and degree of goutiness, also, in any individual member may depend much on

the condition of health of the parent, especially of the father, at the time of procreation.

W. Gairdner believed that some of the worst forms of atonic gout are probably to be met with in the offspring of profligate and debauched fathers. Such men can but transmit a frail nervous system, possessing evil potentiality and instability.

Likeness in features may naturally be supposed to pass on together with likeness in tissue-organization and propensity.

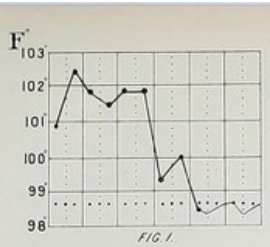


FIG. 1.
Acute Gout.

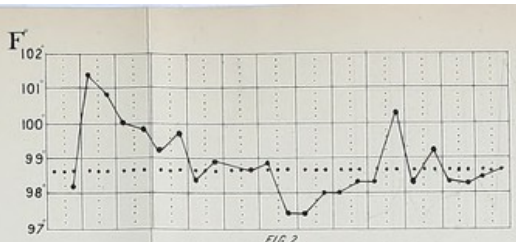


FIG. 2
Acute Gout. Several joints involved.

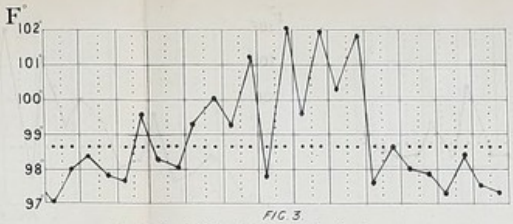


FIG. 3.
Acute Gout. Occasional attacks during six years.

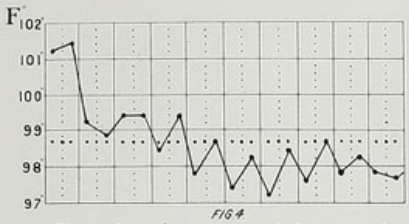


FIG 4
Chronic Gout. (forty years) end of acute attack.

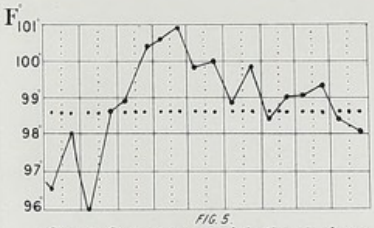


FIG. 5.
Chronic Gout. Acute attack lasting six days.

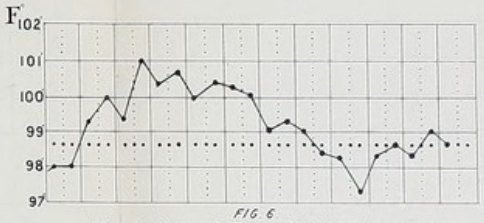


FIG. 6.
Chronic Gout and Chronic Nephritis Acute attack.
Death one month later from pleuro-pneumonia.

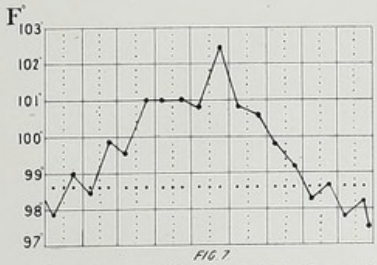


FIG. 7
Second acute attack of Gout within three months.

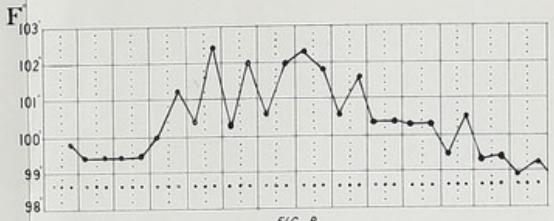


FIG. 8
Gouty (eighteen years) Hydatid tumour of Liver. Acute attack.

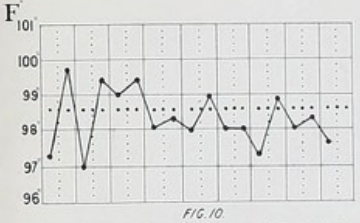


FIG. 10.
Acute Gout. Ankle joint newly seized on fifth day.

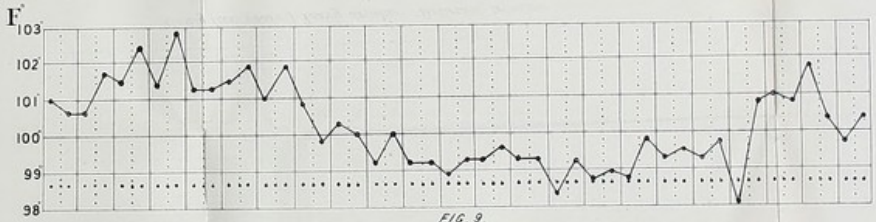


FIG. 9
Gouty (six years) Heavy drinker. Delirium tremens. Acute attack. Relapse.

CHAPTER XVIII.

PYREXIA IN GOUT.

1. Pyrexia in Acute Gout.—I have endeavoured to illustrate the type of pyretic movement in acute gout by a study of a number of cases which have occurred in the Hospital during the last few years, and I am indebted to my late research clerk, Mr. Scott, for securing the facts for me from the clinical temperature charts in sixty-five cases. As it would be impossible to set these out in full, I give the results in figures and append a few charts. (*Vide* Plate 2.) The temperatures are usually taken at 8 A.M. and 8 P.M. These were all male cases, the eldest being sixty-eight, the youngest twenty-seven. The greater number were between forty and sixty years of age.

In some cases I have had them taken day and night every four hours. The pyrexia is best observed in cases which occur while the patients are in hospital, no early record being attainable in patients admitted with acute gout. Such cases are rarely admitted. I find that the temperature seldom exceeds 102° in the sharpest attacks, if there is no complication. A preliminary rise is commonly noted for one, two, three, or four days before a joint is actively involved, the evening temperatures always being highest. With onset of overt symptoms there is a fairly sharp rise to 100° or 100.4° in the evening, with a remission the following morning to normal or 98.8°. On the second day there is an evening rise to 101° or 102.2°, followed by a morning remission to 99.6°, or even subnormal as low as 97°. The evening rise thereafter is usually irregular, reaching 100° or 101°, and remitting on subsequent mornings to normal or a little below this. The pyrexia lasts from two to seven, eight, or ten days, and subsides, often, with a subnormal range for a few days. In not a few cases there is but little pyrexia, the temperature hardly reaching 100° even at night.

Where temperatures are frequently taken, the highest pyrexia is observed at six in the evening, and the lowest at eight or ten o'clock in the morning. The rise begins in the forenoon, and the fall begins towards midnight, continuing gradually till 8 A.M.

In relapses, the temperature may rise suddenly, with an evening exacerbation. In one case there was a rise from normal to 101.2°; next morning 100°; next evening 102.2°; next morning 100.6°; same in evening; next morning 98.4°; and thereafter subnormal. This was in a man, æt. twenty-nine, whose first attack occurred eight years previously. The relapse occurred three weeks after he had been in hospital for the former attack.

The highest temperatures occur in sthenic gout in young men, when there is plenty of vital power and reaction in the system. My cases show that age has not much influence in regulating the degree of pyrexia attained, for some of the highest records occurred in men over fifty. In the oldest man, æt. sixty-eight, 100.8° was reached on the fifth night, the highest point noted. In a man æt. sixty, 101° was noted on the fourth evening, and 101.4° on the fifth evening. The attack commonly subsides in two days after the acme is reached, but may linger on for a week. In a man aged fifty-four, who had a hydatid tumour, presumably of the liver, an acute attack began on May 7, with an evening rise to 102.4°, but the temperature had begun to rise two days before, reaching 101.2° the previous night. This attack lasted twelve days, pyrexia ruling from 100.2° to 102.2° for five days.

2. Pyrexia in Chronic Gout.—In most cases of chronic gout and gouty cachexia, there is no noteworthy type of temperature. My cases show that pyrexia only occurs with such exacerbations as practically constitute instances of acute gout. In such cases there may be a rise to 100° or 101° at night for two or three nights, but the pain and symptoms of disturbance may last long after the temperature has become normal. In many instances there is a slight continuous pyrexia while pain lasts in various joints, and this may persist for several weeks.

These cases are commonly intractable, little influenced for good by any drugs or treatment, and slight exacerbations are frequent. They are not seldom mistaken in practice for cases of chronic rheumatism, and as there are polyarthritic pains, the likeness to the latter is more noteworthy. They are most common in men past middle life, already broken down by repeated gouty attacks, and often by intemperance. Their pains abate slowly, and there is prolonged convalescence.

In noting these temperatures, care has been taken to exclude

all complications such as pneumonia, adenitis (which occurred in one case), and delirium tremens.

The charts (Plate 2) have been selected from a large series, and exemplify the main characters of the pyrexia accompanying acute gouty paroxysms in joints.

Figs. 1 and 2 illustrate a common type of febrile movement. In 3, 5, 6, 7, 8, and 9, it is shown, as already pointed out, that before the acute features of an attack the temperature rises, sometimes one, two, or even four days previously. In 9 there was a serious complication with an attack of delirium tremens, and with the unstable condition of the thermic centre, thus associated, there is to be observed a higher degree of pyrexia than is usual.

Where many joints are involved, there is commonly only moderate pyrexia. This clinical feature aids in the diagnosis of these cases which often closely simulate acute rheumatism, even in respect of severe sweating. In one case of this kind lately under my care, in a man aged forty, of gouty heritage, who had had previous attacks of gout in his great-toes, the sweating was profuse for some days, but there was no sour rheumatic odour given off with it. The temperature never exceeded 101°, and was mostly below this. There was much effusion into the right knee-joint in this case, and severe pain in the toes, ankles, and some of the digital joints of the right hand.

In 10, taken from a woman with cancer of the liver, a very slight pyretic reaction was present during acute attacks. The influence of cancer and the cachectic condition of the patient appeared to modify the thermometric ranges. If any pyrexia occurs in connection with cancer, it is usually slight or moderate.

The local temperature of joints involved in acute gouty attacks is commonly lower than that of the axilla or mouth, sometimes by five or six degrees, although the part feels decidedly hotter to the hand than unaffected areas. The latter sensation is therefore due, on the principle of *ubi stimulus, ibi fluxus*, to the greater quantity of blood in the vicinity of the inflammatory focus.

CHAPTER XIX.

GOUT IN RELATION TO THE VARIOUS CLASSES AND AVOCATIONS OF SOCIETY.

I HAVE already stated my belief that gout spares no class in any population. Hence, may be met rich and poor man's gout. Without paradox, too, it may be added that the rich man may be the subject of "poor," that is, atonic or imperfect, gout, and the humble artisan the victim of severe and frank gout.

Regarding the disorder as one primarily due to dietetic errors and excess, it has come to be popularly considered as the appanage of those who are exposed to luxury.

Its incidence in greater frequency on the upper classes is, doubtless, thus largely explicable. In families of ancient lineage, which are often markedly gouty, there has been an intensifying influence at work by reason of intermarriage amongst individuals similarly circumstanced in respect of the good things of this world. Marriages of consanguinity between members of gouty families will naturally tend to aggravation of the habit in succeeding generations. The ill effects of these are seen especially amongst wealthy members of the Hebrew race where they congregate in large cities, and whose families are, strangely enough, more prone to gout and phases of goutiness than to tuberculosis and strumous disease.

In considering the incidence of gout upon classes, regard must be had to the influences of country- and town-life upon the individuals, and many other factors come into play in each of these. Thus, the nature of the occupation, habits, diet, the characters of the soil and water, must all be taken into account.

A country squire, whose time is divided between his duties and varieties of sport, is commonly regarded as a likely subject of classical gout, because, hypothetically, he comes of long lineage, his ancestors lived freely, and he himself tends to follow in many

of their steps. He may nowadays do more than this, and take an active part in the strain of town-life for a considerable part of the year. It would be hard to determine the share taken by each of the dual parts of such a man's life in generating or evoking gout.

Political life is notoriously conducive to gout. The tension and strife involved by it, together with the high living which hard thinking is apt to induce, and the insufficient bodily exercise and sleep thus entailed, are all provocative of this disorder.

When to these are added the responsibility and anxiety attaching to high political office, hardly a factor is wanting to evoke gout in the holders of it.

It is difficult to affirm dogmatically as to the incidence of gout in the several learned professions. It is certain that there is now less frank gout amongst the members of all of these than was prevalent half a century ago.

Respecting the Clergy, inasmuch as there is little or no gout in Scotland and Ireland, the question is practically limited to that profession in England, and amongst these it may now be declared that the disease in its frank form is not often met with. The manner of life of the modern parish priest is one little likely to rouse into activity even a lurking taint of gout, and perhaps sufficient evidence of the immunity of the English clergy from gouty cachexia is afforded by the highly satisfactory life-statistics relating to that profession. These demonstrate that activity of body and mind, with the regularity, for the most part, of their duties, is conducive to a long lease of health. The abstention from alcoholic beverages now so commonly practised by the clergy will doubtless avert much frank gout; but, where gouty heritage exists, incomplete phases of it will not improbably be manifested in the descendants for several generations.

The incidence of gout on Lawyers is considerable, and greater by far than on the Clergy. The causes which commonly determine the disorder come into full operation in practising lawyers. Brain-work, pressure of business, sedentary life in a vitiated atmosphere, the high-living that mental exercise necessitates in men of affairs, all tend to induce a gouty state. Those who succeed in any profession which entails brain-work, together with the strain of practice, are naturally men of originally robust constitution, who have led in their earlier years active out-door lives; and when, by reason of their success, they are compelled to alter their habits, and live in confined air in large cities, they become particularly prone to develop, or to acquire, gout.

These conditions especially prevail in the Medical profession, in which, however, gout figures to a less extent than amongst Lawyers. Medical practice commonly entails a good deal of out-door life, and greater activity of body than that of Law.

I am indebted to my friend Deputy Inspector-General William H. Lloyd, of the Medical Department of the Admiralty, for the subjoined statistics relating to the occurrence of gout for the last twenty years amongst seamen and marines serving afloat in the Royal Navy. In respect of this return, it must be noted that it includes observations made by many naval surgeons in various parts of the world. Dr. Lloyd remarks that the force on which these ratios are calculated has been, on an average, about 45,000 yearly, and that the number of cases is not likely to be over-stated. Practically, the ages were from 15½ to 45 years, only about 3 per cent of the force being above 45 years old. The incidence of the disease in the various ages cannot be given.

STATEMENT *showing the Number of Cases of Gout, with the Ratio per* 1000 *of Force, n the Royal Navy for the years* 1868–1887.

Year.	Cases.		Year.	Cases.	
	No.	Ratio per 1000.		No.	Ratio per 1000.
1868	98	1.9	1878	102	2.1
1869	87	1.7	1879	79	1.7
1870	82	1.7	1880	89	1.9
1871	105	2.2	1881	62	1.3
1872	94	2.	1882	80	1.8
1873	93	2.	1883	100	2.3
1874	102	2.2	1884	86	2.
1875	99	2.2	1885	79	1.6
1876	118	2.6	1886	78	1.6
1877	98	2.1	1887	90	1.8

For the following statistical Table, relating to the incidence of gout on British troops serving in all parts of the world, I am under obligation to my friend Surgeon-Major William Nash, who has obtained permission for its publication from the Director-General of the Army Medical Department. The Table indicates the marked infrequency of gout amongst soldiers serving with the colours. The admissions were more numerous in the last three years than they were twenty-five years since, but were very few in either period.

RETURN *showing the Average Annual Strength, and the Number of Cases of Gout admitted into Hospital, Deaths, and Average Number constantly Sick, among Her Majesty's British Troops during the years* 1862–1864 *and* 1885–1887, *with the ratio per* 1000 *of strength.*

Years.	Average Annual Strength.	Admissions into Hospital.	Deaths.	Average Number constantly Sick.	Ratio per 1000.		
					Admissions into Hospital.	Deaths.	Average Number constantly Sick.
1862	197,550	18	...	No information.	.09	...	...
1863	199,007	41	...		.20	...	...
1864	192,147	46	...		.24	...	...
Total	588,704	105	...	...	.18	...	...
1885	177,928	78	...	4.07	.44	...	.02
1886	188,739	59	...	3.11	.31	...	.02
1887	193,975	70	...	3.29	.36	...	.02
Total	560,642	207	...	10.47	.37	...	.02

Sailors are practically free from gout so long as they actively follow their calling, and the same may be affirmed of soldiers. In both these vocations, especially in the army, the men are young, and, therefore, not of an age to manifest the disorder. Their active lives in the open air tend, further, to check its development. Retired naval and military men are not infrequently the subjects of gout.

The following cases were reported for me by Dr. Sidney Davies:—

I.—*Gout in a Sailor.*—Alfred B., æt. 63, came to the out-patient department of St. Bartholomew's Hospital on May 8th, suffering from a severe attack of gout in several of his joints. He had been for forty-five years at sea, and was a master-mariner when he left his ship, having become incapacitated by the gout. Being, in consequence, reduced in circumstances, he went into the Union infirmary for a time. No history whatever was obtainable as to gout or rheumatism in any of his ancestors or relations. One brother, older, has never suffered. He used to drink bottled beer and Hollands gin in moderation when at sea. His first attack of gout occurred fourteen years ago, while he was sailing on the coast of Africa, within four degrees of the equator. The pain seized him about midnight in the great toe. Two days after it went to the ankle. After the attack had lasted a few days, it left him for a space of two years. Subsequently, the attacks came on about every year, and, latterly, three or four times a year. The hands were first affected six years ago, the elbows and knees four or five years ago, and the shoulders and hips since that time.

When he presented himself, the patient had a pallid anæmic appearance. His

hair was grey, his teeth strong, and well-enamelled. There were no tophi in the ears, or anywhere else. The mucous membranes were pale, uvula large; tongue pale, coated with a thin yellow fur. Hands much deformed; carpus deflected to the ulnar side. Fingers of "parsnip" type. Extremities of the ulnæ much enlarged. Knuckles everywhere enlarged. Right hand œdematous. He was not troubled with cramp in the legs. There was visible pulsation in the brachial arteries. The heart-sounds were clear. There was thickening of the bursa over the right olecranon. Urine 1015, acid, contained a trace of albumen, but no sugar. The patient has had no previous illness but "fever." He stated that he slept well, but had to get up three or four times in the night to make water. He was ordered the following prescription:—Pot. iod., gr.iij.; syr. ferri iod., fʒss.; tr. nuc. vom., ɱx.; aquæ, fʒj. ter die sum., and lin. sinapis co. to apply to the affected joints.

When he presented himself at the Hospital, he had been in continual suffering for six months.

On May 28th he said he felt much better. The medicine was discontinued, and he was ordered lin. saponis co., fʒiv., tr. iodi, fʒss., to be mixed and applied to the joints instead of lin. sinapis co.

On June 4th he was better. Ankylosis existed in many of the phalangeal joints of both hands; only the forefingers and thumbs could be opposed, so that the grasp was very imperfect.

August 3rd.—Continues free from pain.

II.—*Gout in a Sailor.*—Peter W., æt. seventy-three, came to the Hospital on April 6, to seek relief from an attack of gout, chiefly in the left hand. He had lived at Padstow and at Plymouth, and was formerly a sailor. He was married, had six children, and the eldest son was living, aged fifty-three, was formerly a sailor, and suffered from gout.

The patient was a man of healthy appearance, his head was bald and shiny, he had no arcus senilis, and was nearly edentulous, the teeth which remained were yellowish; eyes glistening; the nails were striated. There was a well-marked tophus on the left ear, and a doubtful one on the right. The uvula was long and glossy, the skin of his hands smooth and shining.

The first attack of gout seized the left great toe-joint eighteen years ago. On the occasion of the second attack his left wrist, instep, and right middle finger were involved in the disease. The last joint of the right middle finger was much enlarged. The fingers were straight. The bursa over the right olecranon was full of, presumably, uratic deposit.

The patient did not suffer from cramps, nor from headaches. His urine was acid, sp. gr. 1024, and contained a trace of albumen.

Three years ago he began to have attacks of "deadness" at the end of the ring-finger, passing to the other fingers and to the wrist, accompanied by burning pains, alleged to be constant. He was rendered miserable by them, and the discomfort kept him awake at night. The heart-sounds were clear. The arteries had the hardness of senility.

Merchants and men of business are certainly prone to gout, the more so if they take insufficient exercise, and are exposed to the responsibilities and anxieties inseparable from large monetary transactions and precarious speculations. It is, doubtless, in respect of the strain and excitement attendant on the latter that stockbrokers are frequently sufferers.

Farmers, in spite of their wholesome calling, are as a class somewhat prone to gout; but in their case, as in that of others,

a well-marked ratio is found to exist between the occurrence and frequency of the disease, and periods of prosperity or the reverse.

Artisans suffer more than labourers. The particular occupation followed has much to do with the incidence of the disease in these classes, as has also the important matter of habits in respect of strong liquors.

Workers in lead and painters stand apart from all others in their special proclivity to become gouty.

In all the instances just referred to, my remarks apply, of course, to the class as a whole. In every case, regard must be had to the influence of heredity, and to the individual tendencies and habits.

CHAPTER XX.

GEOGRAPHICAL DISTRIBUTION OF, AND INFLUENCE OF CLIMATE, SOIL, WATER, AND SEASONS ON, GOUT.

THIS subject has received a good deal of attention. The field of experience is naturally much enlarged at the present time, when locomotion is rapid and easily accomplished.

I can hardly doubt that errors have crept into some of the accounts available for a study of the geographical distribution of gout, so that many forms of arthritis have been improperly reckoned as gout which have no claim to be so regarded.

For many years past I have sought information from practitioners coming from various parts of the world respecting their experience of gout, and in most instances there has been little or none forthcoming, save where Europeans have formed part of the community, and some amongst these have either carried hereditary taint with them, or have led such lives as favour the onset of gout.

There can, I believe, be no doubt that there is more gout in the British Isles than in any other part of the world, and the greater number of examples are to be met with in England. The disease is chiefly spread over the temperate zone. During extensive travels in many parts of the world, I have been on the outlook for gout, and am compelled to affirm that I have seen very little out of England. I believe that the next largest field is presented by France, where, however, the upper classes present the majority of cases. There would appear to be little gout in Germany, Austria, and Italy. In Holland there is practically no gout, and but little is met with in Belgium or Spain. In Scandinavia the populations are free from gout, and the same is the case in Russia. Yet, in the capitals and large cities of all these countries cases of gout may be met with, chiefly amongst the well-

to-do or luxuriously living of the community. Some cities show greater preponderance than others. Thus, Hamburg and Bremen furnish more cases, probably, than Berlin. Communities of rich merchants, including many Jews, are likely to be centres of gouty disease.[1]

The peculiarity of the greater frequency in (geographical) England is the occurrence of the disease amongst the lower orders, especially the artisans. This is not the case in any other country. The greatest prevalence, too, is certainly in London. Some of the larger cities and towns in England furnish cases of gout amongst the luxurious and well-fed of the population, but very little gout is met with amongst the labouring classes. There is less gout in the North than in the South of England.

In Scotland and Ireland gout is practically confined to the limited class of luxurious livers. Glasgow, though a busy and wealthy commercial centre, furnishes but rarely cases of gout in its upper classes. The fact is that, with the "high thinking and plain living" of Caledonia, there is practically but little beer, and much less wine, consumed than amongst similar classes in England, whisky taking the place of both to a large extent.

Cases of gout are hardly ever seen in the Scottish and Irish hospitals, and are rare in the northern parts of England. Such cases as occur there are commonly in the persons of over-fed and bibulous men-servants.

It is alleged that fewer gouty ailments are met with in the area of the Moselle than in that of the Rhine, and this is attributed to the respective qualities of the wine produced in each area, the Rhenish wines being more acid.

It has, however, long been shown that it is a fallacy to connect directly the occurrence of gout in any district with the dietetic and drinking habits, or with the special liquors consumed in that district. Yet, such habits, and the peculiarities of the drinks taken, must be regarded as factors in the onset and evolution of gout anywhere. These are not, however, the *sole* factors in inducing the disease.

In Russia, save in the capitals, little is known of gout. In Greece, Turkey, and the Levant, generally speaking, there is no gout. Strict Mahometans are no subjects for the disease, but

[1] To explain the occurrence of gout in Italy and Greece in the days of their early greatness, of which there is trustworthy record, we must believe that the habits of the people in respect of indolence, luxury, and diet were such as to induce the disease, notwithstanding the good climate they enjoyed. With change of habits the disease has disappeared from the present populations of these countries.

where the habits of the "infidel" are followed by some of them, gout is found to supervene. The same is the case in India. Only such natives as indulge in animal food and European liquors are the victims of gout. Temperate Europeans may fairly expect to escape gout anywhere in the tropics, but there is a good deal of gout in India. Twenty-three years' experience amongst the European *employés* of several large Indian Railway Companies has convinced me on this point. Good living, brandy and beer in inordinate quantities, with limited amount of exercise, together with anxiety and head-work, would appear to be the inducing factors. Similar experiences hold good for Europeans in Ceylon and China. Hirsch, however, mentions the occurrence of gout amongst the indigenous population of Amoy. New Zealand, in common with most British colonies, appears to be free from gout.

There is no record of the disease anywhere on the African continent, nor is it known in any of the adjacent islands, including Madeira and the Canaries. Europeans may develop gout anywhere, either from hereditary tendency or such habits as lead up to the disease; and hence cases are met with occasionally in persons who have resided in the tropics or in colonies where the disorder is unknown among the native races or the mass of colonists who own no gouty heritage.

In the United States of America gout is practically unknown. A few cases are met with in the large cities, but, according to Dr. Da Costa, of Philadelphia,[1] the disease has not yet developed amongst the increasing population. Cases of lithiasis are met with, he tells me, which are regarded as early indications of the disorder. Hence, I cannot agree with Hirsch's statement to the effect that gout would appear to be as common in the large and populous cities of the New World as it is under the same circumstances in those of the Old.[2]

The ancestry, diet, and habits of the citizens of the United States for the most part are such as will long prevent the onset of gout in that country. The immigration of Irish, Scottish, Scandinavian, and German people into that country peoples it mainly with a stock void of gouty heritage. The good climate and the active open-air lives of the people will tend to avert goutiness. But little wine is used, the beer is light, and much water and weak tea are taken. The alcohol chiefly used, or abused, is rye-whisky.

In Canada the same conditions prevail.

[1] The Nervous Symptoms of Lithæmia, Amer. Journ. Med. Sciences, October 1881.

[2] *Op. cit.*, vol. ii. p. 657.

With respect to colonists, it must be borne in mind that they begin their new life at an early age, when gout is little likely to supervene, and the frugal and active lives led by the majority of those who succeed and live their full term naturally prevent the onset of the disorder; and to these the words of Rousseau may be fairly applied, "Temperance and labour are the best physicians of man."

In Australia gout is practically unknown, except amongst immigrants already affected. It is noteworthy that the colonists there are large meat-eaters, and in the towns take beer freely. They also consume much tea.

Nothing is known of gout in Central America or California. In the West Indies it is practically unknown. The only case I have met with was in a gentleman from St. Kitts, who led a temperate life, but had suffered severely from lead-impregnation, owing to improper water-storage. He had severe gout, with tophi in his ears, and chronic interstitial nephritis.

Little is known of gout in South America. Hirsch directs attention to Dr. Dundas's observations on the immunity from gout enjoyed in Brazil, which the latter deemed remarkable, inasmuch as the prevalent habits of the higher classes and well-to-do foreigners were such as to induce the disorder. Indolent lives are led and much animal food is taken, but not much wine. It is probable that not many Englishmen are included amongst the foreigners, and of these, it may be assumed, that they proceed early in life to that country, and stay no longer than they are compelled to do.

With respect to tropical residence and its influence generally on the onset and course of gout, it must be borne in mind that the skin is kept in very free action, that much diluent liquid is taken, and that sun-influence is everywhere a potent factor in checking gouty processes. Cases of confirmed gout are not favourably influenced by extreme heat or by tropical residence, which is unduly enervating.

Cold and dry climates appear to confer immunity from gout, and no less from rheumatic disease in all its varieties.

The warmer and more moist climates favour the latter, as is shown by the frequency of chronic rheumatic arthritis in Ireland and the western portions of Scotland.

Influence of Soil.—Inasmuch as all arthritically disposed persons are very "barometric" (*Trousseau*[1]) and sensitive to damp and "shifty climate," it is of importance to have regard to the

[1] A gouty patient once told me his joints were "barometers."

nature of the soil and other telluric influences to which gouty persons may be submitted. They certainly do best on dry, gravelly soil, and do less well on water-logged or clay strata. Chalky soils are favourable, provided that hard water be not drunk. Sandy soils are apt to be somewhat cold and damp, but sandy hills with soft water are on the whole not unfavourable. Dry sites on hilly slopes sheltered from the north and east, with a southern aspect, are the best for the gouty. My experience fully confirms the opinion of Laycock that arthritically disposed persons are favoured by an inland and hilly residence, and respond less to the influences of marine climate, even when not injuriously affected by it.[1]

Such persons as become "bilious" and uncomfortable at the seaside are commonly goutily disposed. An inland residence that is at once high and dry usually suits this class of sufferers better. East winds are generally noxious to, and ill-borne by, gouty persons. Extreme heat is also very trying for them,[2] and they enjoy most comfort in temperate, dry, and equable climates. Barometric extremes are equally unfavourable, and the condition of atmosphere during thunderstorms is especially disagreeable, and apt to induce in the gouty, more than in other persons, headache and depression.

Influence of Water.—The gouty are very dependent on the nature of their water-supply. Waters much impregnated with lime or with iron are distinctly unsuitable or apt to provoke gout. Sulphate of lime in water is especially noxious to the gouty.[3] Both calcic and ferruginous salts tend to check elimination of uric acid, and so are badly borne by the gouty.

A question arises as to the influence of climatic and geographical influence on the outcome and development of gout. It appears to be certain that the conditions of life in tropical and subtropical climates tend to check the onset of gout even when gouty heritage is a factor. The causes at work here probably relate to the free action of the skin, less animalized diet, more simple and abundant diluent drinks, and the generous influence of the sun.

[1] This point is well-discussed by Dr. Robertson, of Buxton, in his book on "Gout," published in London, 1844, p. 33. He is of opinion that continual residence at the seaside aggravates the gouty habit.

[2] Scudamore noted the bad effects on the gouty of the long-continued heat of the summer of 1818.

[3] Hence, the special harmfulness of a cheap and factitious wine, "plastered" with gypsum, called "sherry."

In colder but bright climates void of moisture, the prevailing conditions necessitate active muscular exercise and free aëration with oxydation, abundant nitrogenous food, with equally abundant ingestion of hydrocarbons, being, thus, readily disposed of.

Under the influence of these causes persons with gouty heritage may, even if strongly impressed thereby, entirely fail to develop the disease, or do so only in mild or incomplete forms. Dr. Robertson thinks there is good reason to believe that the stimulating air of the sea-coast diminishes the development of a gouty habit, though intensifying it when established.

Seasons.—It is commonly believed that gout is apt to occur with greatest frequency in the spring and autumn.[1] This appears to be the case in respect of attacks of frank gout, yet there are so many exceptions that it is not possible to be dogmatic on this point. It is certain that paroxysms occur at all times of the year, and any frequency observable in spring and autumn is probably attributable to the climatic peculiarities prevalent at those seasons.[2] Warm, genial, and sunny weather best suits all subjects of gout, provided the temperature is not excessive and exhausting. East winds and "shifty" weather are ill-borne. Hence, the violent changes of spring, and the loss of sun-influence together with the damp mists of autumn, are likely to check the action of the skin, and throw burdens on internal organs which are conducive to the generation of gout.

The same views prevail in regard to many skin-disorders, and probably own a similar explanation. A sunless or wet summer may prove as gout-provoking as an ordinary spring or autumn. A cold and bright winter, if there be little rain or snow, may prove exhilarating and little harmful to the gouty. The fact is that equability in all surroundings is best for such patients, that all sudden changes are harmful, and that the highest standard of health attainable by them is only secured by care and watchfulness, *de die in diem*, as to habits of exercise, food, and clothing.

This is especially true for subjects of the gouty cachexia, and is naturally of less importance in younger and robust subjects,

[1] Gout follows the laws of other nervous diseases in respect of its frequency and intensity at certain seasons of the year. So does rheumatism. Thus, the curve of intensity begins in February, rises during March and April, and falls at the end of that month, disappearing in summer and autumn, reappearing in December. Delirium tremens follows the law of dietetic ailments, and its curve rises in the summer solstice. Were gout entirely a dietetic disease, it should follow the laws of such ailments. Thus, winter and spring are the worst seasons for the goutily disposed, and a great physiological law influences gout together with other nervous disorders.

[2] "Towards rain and frost gout appears."—*Lord Verulam, Nat. Hist.*, Cent. ix. p. 8.

who can better withstand vicissitudes, both of climate and season.[1]

As the seasons in our country are notoriously uncertain in character, we must follow the advice of Sydenham, and note the constitution of each as it comes round. With modern advances in meteorology, this becomes a more exact and fruitful study than was formerly the case.

The influences of British winter climate on the production of gouty states have not merely to do with cold and negation of sun-influence. Dampness of atmosphere and darkness are each provocative by their directly depressing qualities. In the absence of light and genial influences externally, a suitable amount of exercise and aëration is apt to be neglected, and pleasures of mind and body are consequently sought indoors. Hence, over-action of the former, and under-action of the latter. Social joys are sought to replace what cannot be found out of doors, and over-indulgence in food and stimulants is too commonly the set-off to the wholesome influences which are often hard to find out of doors. Small wonder, then, that gouty tendency is aggravated or the habit directly induced. Hence, the value of some outdoor pursuits or bodily recreation, when such can be obtained. These are, unfortunately, often far to seek in winter in large cities.

Attacks of gout are believed to be infrequent at sea. The disease is extremely rare amongst sailors, but I have known severe attacks to occur in gouty subjects even in tropical waters. Insufficient exercise and over-eating, with, possibly, too free indulgence in alcoholic liquors, afford sufficient explanation of such cases. Constipation of the bowels, common at sea, is a predisposing cause. Sudden changes from the tropics into colder areas are also intelligible causes for attacks.

[1] Laycock taught in his lectures that winter was as dangerous to the gouty as to the tubercular.

CHAPTER XXI.

TREATMENT OF THE SEVERAL VARIETIES OF GOUT, MEDICINAL, REGIMINAL, AND PREVENTIVE.

THE literature and lore of the therapeutics of gout and of gouty states are, like that relating to the nature and phases of the disease itself, of enormous extent. In discussing them, I am reminded of a saying of my former preceptor, the late Professor Hughes Bennett, of Edinburgh, who was wont to affirm of any disorder or ailment for which long lists of remedies were recommended, that, in such cases, we were probably very ignorant of the true nature of the disease which we sought to control or combat.

In respect of gout, as of other maladies, it may, however, be confidently stated that modern advances in the study both of morbid anatomy and of morbid processes, in the dead-house and laboratory, and also at the bedside, have rendered the therapeutic art at once more simple and less uncertain.

The curability of gout has been often questioned, and great names have been quoted denying such possibility. In particular, Cullen and Trousseau have stated their belief that nothing could be safely done to cure acute attacks; and the prescription of "patience and flannel" of the former physician has come to be proverbial in obstinate cases, both of gout and rheumatism.

It may be fairly stated that for many years no difficulty has been experienced in successfully treating paroxysms of acute gout, and that such are truly, as affirmed by Garrod, as controllable and amenable to proper remedies as any other inflammatory affection.

There is no doubt that, either with the inherited or acquired gouty habit, patients are liable all their lives to more or less manifestation of the dyscrasia which will dominate and modify many of their general nutritional processes. Drugs alone cannot be trusted, once and for all, to remove a gouty habit of body. It is of the essence of this malady to recur and grow up in the

system from time to time, often in spite of all regiminal and other precautions. As has been shown, the degree of impressibility by this or by any other diathesis varies infinitely in different individuals. Much may be done by diet and by remedies to check the tendency to recur, but it is too commonly the case that,—once gouty, always gouty.

The natural history of a fit of the gout, which is well-known, leads to the belief that gout in this sense cures itself, and is Nature's way out of the trouble.[1] This is, at all events, so far true, that no countenance can be given to any therapeutic efforts which would tend to abort a fit once established. For all this, the physician cannot consent to look on and do nothing in such a case, any more than he can stand idly by a patient suffering from pneumonia or one ill with enteric fever.[2] There is much to be done in such cases, though no educated physician would now allow that he treated the *diseases* in any one of them. He, of course, treats the *patient*, and helps him through his diseases. This is the rational practice of physic of the last quarter of this century.

Again, in chronic forms of gout there is much to be done for the patient to render life tolerable, and to prevent the mischievous spread of the morbid processes.

Much discredit has been brought on the treatment of gout and gouty states because of the impatience and credulity of sufferers. They will not submit to the necessary regimen and rest, and they are too ready to employ any nostrum or application foisted on them by ignorant persons, or advertised in the newspapers. Having damaged their health thereby, they come under rational treatment at a great disadvantage, both to themselves and the practitioner.

In the treatment of gout it is essential to have in view the whole malady, and not the mere accidents of it. The constitutional state demands unceasing attention in every case. The patient's attention is mainly directed to the painful or disabling episodes of his case, and he is little disposed to have regard to the necessary *régime* called for in his daily life when these gouty outbursts have passed away.

Treatment relates, therefore, to the active phases of the malady, and to the patient's condition in the intervals between such attacks. The management of the cachexia induced by gout in its later stages relates more to the general state of the patient

[1] "The gout is the only cure of the gout."—*Mead.*

[2] "Nature seeks a relief *quâ detur porta*, and the physician must not arrive only to forbid it and to lock up the mischief."—*W. Gairdner.*

than to anything specifically due to the dyscrasia which has induced such a condition.

I propose to describe: (1.) The treatment proper for a paroxysm of regular acute gout; (2.) The medicinal and other treatment in the intervals between the paroxysms; (3.) The treatment of chronic and irregular gout; (4.) The local treatment of the joints in chronic gouty arthritis, and the treatment of tophi; (5.) Treatment of retrocedent and incomplete gout; (6.) The treatment of special disorders dependent on the gouty habit; (7.) Treatment of gouty cachexia and of gout in elderly persons; (8.) Preventive treatment of gout.

As pointed out by Sir Thomas Watson, Heberden looked forward to the time when a specific for gout, as certain as those discovered for ague and scabies, would be found, and the former writer conceived that the time had come when colchicum was proved to be of its known utility in easing the pain and other troubles attaching to gout. The "inert expectancy," as he terms it, of Sydenham and of Cullen, has now given place to more active, if still empirical, measures, and with decided benefit to the sufferer.

It is remarkable how long the knowledge of the value of this drug lay dormant. It was well known in the sixth century as a remedy for gout under the name of Hermodactyls.[1] It appears probable that these consisted of the corms of a variety of colchicum, though not of the *C. autumnale*. Only within the last century has the latter been employed in practice, and no other variety is now made use of, although parts of other plants of the same natural order (Melanthaceæ) have some repute in the treatment of gout.

The bitter Hermodactyls are regarded by Planchon, Dymock, and others as being the corms of a Colchicum (*Colchicum Variegatum*). By the courtesy of Mr. Carteighe, President of the Pharmaceutical Society, and of Mr. Jackson, of Kew, three authentic specimens of this form of Hermodactyl were procured from the Kew Museum and examined by Professor Dunstan in the Research Laboratory of the Pharmaceutical Society. (*Vide* fig. 22.) They were found to contain a minute quantity of an alkaloid, which, however, possessed none of the properties of colchicina, the alkaloid of *Colchicum autumnale*. A sufficient number of these Hermodactyls could not be obtained to investigate further the nature of the alkaloid.

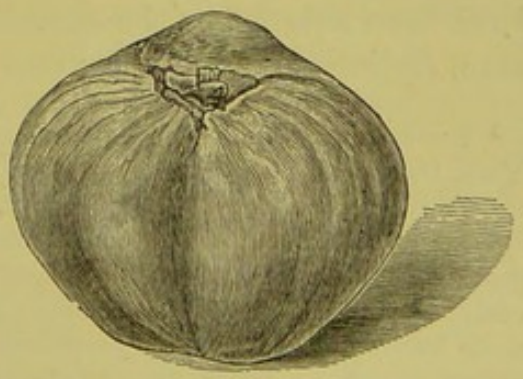


FIG. 22.

The Hermodactyls of Pereira and Guibourt are not bitter, and are yielded most probably by a species of Fritellaria. In fact, they are *not* the corms of a Colchicum.

[1] Recommended by Alexander of Tralles (a city of Lydia), circ. 580 A.D. (often quoted by van Swieten).

No other drug has effectually displaced colchicum from its pre-eminence as a remedy for gouty pain. Within recent years the claims of sodium salicylate have been pressed in various quarters, and this most valuable addition to our therapeutic armoury in the case of rheumatic fever has sometimes displaced the older practice of exhibiting colchicum in gout. Of this more anon.

1.—Treatment of Acute Gout.

An attack of frank, paroxysmal, gout constitutes a crisis in an aggravated gouty state of the system, and, as such, is a severe symptomatic expression of that state.

Abortive Treatment.—The idea of treating gout with a view of aborting it could not occur to those who regard it from a proper pathological stand-point. Several methods have been proposed for this purpose. Thus, firm pressure, as by strapping with diachylon plaster, applied at once to the affected part, is alleged to give relief and prevent further trouble. Various vaunted specifics are in popular use, such as Laville's liquor, and remedies of like kind, supposed to contain colchicum, veratrina, and other anodynes. Hypodermic injection of morphine in the neighbourhood of the affected part is another such remedy.[1] In desperation, patients have themselves applied snow and cold applications to banish the pain and local disturbances.[2]

Its Undesirability.—Practical acquaintance with the disease affords no countenance to any of these methods of treatment. The risks of suppression of local symptoms are so great, and the benefit, if there be any, so dearly bought in most cases, that an ectrotic treatment can seldom be advisable.[3]

Cases are on record in which threatening articular attacks have been averted by the exercise of strong will. Thus, a strong man, feeling an acute paroxysm impending, determines to "fight

[1] Recommended by Dr. D. B. Simmons, of Tokio, Japan. Medical Record, New York, October 8, 1887, p. 485.

[2] The illustrious Harvey, and Gregory, of Edinburgh, resorted to this.

[3] Mr. Teale has kindly given me the following particulars of a case of acute gout, which was remarkably and successfully aborted by morphine given subcutaneously:—"A lady, a cripple in her hands from 'rheumatic gout'—unable, I believe, to write letters for two or three years—was in great suffering from a more acute paroxysm, affecting hands, arms, and shoulders. She was moaning, almost screaming, with pain, and utterly helpless. To ease the pain, I injected a small dose of morphia—an $\frac{1}{8}$th of a grain, I think. Next morning her gout seemed to be gone; she could move her hands and arms. In three days she packed up her clothes herself, and took a long journey; and for a year or two sent messages to me to say how she had regained her power of writing and had lost her pain."

down" his gout, and forthwith steps out briskly for a few miles, and "walks off" the attack. In such instances we may take note of the remarkable influence exerted by a vigorous nervous system on the peculiar phenomena of a gouty fit. They are not frequently to be met with, and may only be looked for in persons of robust habit, who offer strong resistance to gouty dyscrasia.

The measures to be adopted relate to an illness which will last from one to two weeks. The natural history of an acute gouty access is well-ascertained.

Treatment must be local and palliative, and also constitutional and general. As in all diseases, our efforts are directed to the conduct of the case. We do not treat a disease, but we treat a patient with a disease. The individual is to be studied in each instance, and the questions to be answered at the bedside are: —What will the disease do to the patient, and how will he bear it?

A paroxysm of gout must be treated somewhat differently, according to the age, state of constitution, and vital powers of the individual attacked. Where the attack is regular and sthenic, the degree of intensity of the pain and inflammatory symptoms, even if great, never warrants severe, or what are termed antiphlogistic, measures. Practice of this kind is proved to be bad, to retard recovery, and render relapse or early recurrence of fresh attacks, probably less regular, certain. Hence, general bloodletting, or local depletion by leeching, is to be avoided. The former is rarely necessary, and the latter, according to Garrod and others who have had experience of it, is harmful. I have several times seen patients whose gouty joints had been leeched. In one case six leeches did no good. In another, four leeches were believed to bring relief in twenty-four or thirty hours.

The patient must remain recumbent, though not necessarily in bed, for several days. This is of great importance. The affected limb should be raised, supported on a firm pillow, kept warm, though lightly covered, and protected by a cradle. A thin layer of carded cotton wool, invested with a domet bandage, is a grateful application for most cases. Experienced sufferers tell of relief afforded by warm spirituous lotions on lint covered with oiled silk—whisky and water being a favourite application. A lotion consisting of a drachm of sulphuric æther in six ounces of water is sometimes found soothing (*Pye-Smith*). The intensity of the pain often suggests the employment of some anodyne lotion, and, hence, laudanum and water in various proportions, or the combination of belladonna liniment with morphia, as employed

by Garrod,[1] and which I have found useful, may be prescribed. As moist applications are sometimes uncomfortable and inconvenient, I have occasionally used anodynes dissolved in oleic acid with decided relief.[2]

Iodoform has been recommended for external use in acute gout, employed as ointment, or with flexile collodion. Nitrate of silver, rubbed over the affected part, is well-spoken of by some practitioners. I have no experience of either of these local methods of treatment.

The unguentum atropinæ (gr.i. ad ℥i.) of the Pharmacopœia is available, but somewhat less manageable than the oleate.

The application of oil of peppermint has been recommended. I have no experience of this practice, but I have known menthol in a spirituous solution to be employed as a lotion, and to succeed when other anodynes have failed.[3] Cocaine, in the form of liniment, ointment, or lotion, may be used.[4] Flexile collodium and French chalk in fine powder are amongst many extolled local anodynes. I believe that any application which chokes the sweat-ducts is undesirable in local gouty inflammation, but the truth is, that relief in one case is sometimes gained by a remedy which will quite fail to afford ease in another. Occasionally, I have known the pain of gout to be so prolonged and atrocious, and so rebellious to all local measures, including strong belladonna and laudanum lotions, that little relief was reached till the disease had well-nigh run its course. It is advisable to have a variety of local applications at our disposal, so as to find some one that may prove effectual.

Poultices are sometimes soothing, and warm fomentations likewise. I prefer the latter, but only at the outset of an attack, as a preliminary measure to those just mentioned. Blisters are not advisable in early and regular articular paroxysms, but find a place, if skilfully used, in some phases of subacute or chronic

[1] ℞ Lin. Belladonnæ f℥iij., Morphinæ Hydrochloratis, gr.x. M. ut ft. Linimentum. Sig. A teaspoonful to be mixed with a tablespoonful of hot water, and applied on lint under oiled silk every four hours.

[2] ℞ Atropinæ gr.iij., Morphinæ Hydrochloratis gr.xv., Acidi Oleici f℥i. Solve ut ft. Linimentum. Sig. To be painted over the painful joint with a large camel's hair brush, and carded cotton to be superimposed with a domet bandage.

[3] Three parts of menthol and two of camphor may be rubbed up together to form an anodyne application, or ℥ss. of menthol may be dissolved in f℥vi. of spirit of chloroform for a lotion.

[4] ℞ Cocainæ gr.v., Paraffini Mollis ℥ij. Solve, calore leni, ut ft. Unguentum.

℞ Cocainæ gr.iv., Ol. Amygdalæ Dulcis f℥ss. Solve, calore leni, ut ft. Linimentum.

℞ Cocainæ gr.iv., Acidi Oleici f℥ss. Solve, calore leni, ut ft. Linimentum.

gouty arthritis. They should be applied for three or four hours nightly as "flying" blisters.

For internal remedies, it is well to begin in most cases with an aperient dose of calomel and colocynth and henbane pill. Strong purgation is improper, and may aggravate the attack. If the belly be hard, this may be taken as an indication that the patient will bear purging. A reverse condition should determine against it. This is an old and very true observation. One or two grains of calomel, with six or eight of the above-mentioned pill, should suffice over-night, to be followed by a Seidlitz powder, or four ounces of Püllna or of other saline aperient water, taken warm, early the following morning.

It is well to wait till the attack is fully manifested in the part before beginning treatment by colchicum. This was Sir Henry Halford's rule, and it may be followed implicitly. The wine of the corm is sufficiently trustworthy, though the tinctures of the seeds and of the flowers have their advocates, and the first dose should be larger than subsequent ones. Thirty to forty-five minims may be given at night, and half the dose in the morning. Some practitioners affirm that colchicum itself is sufficiently aperient and requires no combination with other purgatives, but general experience is not in favour of this practice. Hence, it is well to add twenty grains of carbonate of magnesium or a drachm of the sulphate of that base to each dose, and syrup of white poppy, one drachm, and camphor mixture, or some aromatic water, one ounce and a half, may be the vehicle of it.[1]

Great benefit may be sometimes secured from a dose of the mistura sennæ composita, to which half a drachm of the wine of colchicum is added. This draught should be given early in the morning during an acute attack. This may replace colchicum during the day.

After three or four days of this treatment, two pills containing two or three grains of the acetous extract of colchicum, with as much compound ipecacuanha powder and compound colocynth pill, may be given for two or three nights, followed up, if necessary, by a mild purgative. The condition of the tongue is commonly a safe guide to the progress of the case. Marked relief to the gouty fit will rarely accrue till the tongue begins to lose its coating.

Treatment of acute gout by colchicum is that most commonly practised. The works of most authors on gout tell of numerous

[1] The *Haustus Colchici* of the St. Bartholomew's Hospital Pharmacopœia is a well-proved formula:—℞ Magnesii Carbonatis gr.x., Tinct. Sem. Colchici ℥xx., Aq. Menth. Virid. ad f℥i.

untoward effects produced by this remedy, and contain grave warnings against over, or too prolonged, dosing with it. It is probable that the drug has been little abused, certainly of late years, by professional hands, and many of the evils referred to have doubtless ensued when it has been taken on the patient's own responsibility, either overtly or, rudely, in the form of some nostrum. I have never myself had any experience of undesirable effects from the use of colchicum. Sir George Burrows was wont to employ it sometimes freely, but I never saw more severe results than purging with the characteristic green stools.[1] Both practitioners and patients appear sometimes to expect too much from this drug, and there is temptation to press it in certain cases, especially when it is desirable to render a patient fit for some duty or appointment by a definite day.

Gouty persons should be warned of the imprudence of conducting their own cases with this or with any other drug, and be especially put on their guard against resorting to reputed specifics, most of which contain colchicum or its congener veratrina.

The wine of the corm of colchicum is that most frequently employed. It is the least satisfactory in appearance of all the liquid preparations of the drug, precipitating, as does ipecacuanha wine, a sediment, and seldom being bright. The sediment has been found inert as a remedy in gout.

Preparations of the seeds are more purgative than those of the corm. Hence, according to Dr. Robertson, the dose of the former should be smaller—twenty as against thirty minims. A grain of dried corm is contained in five and a half minims of the wine, and nine minims of the tincture contain one grain of the seeds.

Battley's liquor colchici recentis contains one grain of solid extract of the corm in eight minims, or the equivalent of fourteen minims of the wine. The extract and acetous extract may be given in doses of half a grain to two grains.

There is a compound tincture of colchicum which is preferred by some practitioners. It should rather be called an ammoniated tincture, since it is made by macerating the seeds of colchicum in aromatic spirit of ammonia. I have no experience of its value.

Veratrina and colchicina have both been employed internally and locally in acute gout. The former was employed by Dr. Turnbull as an external application. Twenty to forty grains of either may be mixed with an ounce of spermaceti ointment, or

[1] In appearance these have been likened to green pea-soup.

the same quantity may be dissolved in an ounce of rectified spirit and applied with a camel's hair brush. Oleic acid may also be used instead of ointment or spirit.

These two alkaloids are generically allied. Veratrina causes violent sneezing, while colchicina does not. I have employed the former with benefit as a local application in gout, but have never administered it internally.

In a case of painful gout of the knee-joint, I applied a lotion of equal parts of tincture of colchicum seeds and water on lint under gutta-percha tissue. No relief was obtained, and a red papular rash came out on the skin in consequence. In this case, an arnica lotion, one part of the tincture to eight of water, applied for several days, induced no rash on other parts of the skin, as commonly met with in gouty persons.

In a case of painful gouty finger-joints with a "crab's-eye" (inflamed) bursa, a colchicum lotion was found very soothing.

The explanation of the specific effects of colchicum on gout and gouty pain is not yet forthcoming. There are several theories as to its action. Much discrepancy exists in the accounts given by different observers as to its influence on excretion both of urea and uric acid, due, as suggested by Lauder Brunton, to experimentation being carried out while different diets were employed. A small dose increases gastro-intestinal secretion, while a larger one causes nausea and diarrhœa, lessens muscular irritability, and paralyses the central nervous system. The blood-pressure sinks gradually in consequence of lowering of the irritability of the vaso-motor centres.[1]

Professor Rutherford, of Edinburgh, has conclusively proved that colchicum in large doses is one of the most powerful true cholagogues known, the bile being also rendered more watery by it.[2] Its aperient properties are very marked, and, hence, it is often a valuable addition to other aperients for ordinary purposes.

Its active principle, colchicina, has been employed in gout with alleged benefit. It is very powerful, and can only be prescribed in doses varying from a sixtieth to a fifteenth of a grain, dissolved in water, hypodermically, or by the mouth. A solution of one-twentieth of a grain in twelve minims of distilled water may be used for subcutaneous injection, four to eight minims being

[1] Schmiedeberg.

[2] *Post-mortem* the mucous membrane of dogs' intestines was found intensely vascular after injections of drachm doses of extract of the corm. The intestinal glands were stimulated as well as the liver (*Rutherford*).

thus employed. I have never had occasion to try this method, and being strongly averse from all forms of hypodermic medication, save when indispensable, I am not likely to employ it.

In many gouty patients it is far from advisable to injure the integuments, even by a needle, and as the best results of the whole drug can be obtained in the usual fashion, I see no advantage in resorting to this inconvenient and painful plan of giving it.

It is certain that all the benefits sought from colchicum can be obtained without producing in the patient any very noteworthy physiological effects. In other words, the specific power of the drug over gouty arthritis may be secured without the induction of depression, nausea, or untoward purging. Sweating is sometimes caused, but this is not undesirable. Colchicum is a vascular depressant, the frequency and force of the pulse being reduced by it, and with the lowering of arterial tension comes relief to the circulation in the inflamed joint. As a remedy, the drug is depressing, in proportion to the dose and length of time it is given. The indication, therefore, is to employ as much as, and no more than, will control the urgent symptoms, and not to continue its use, unless in small doses, longer than is necessary.

The anodyne effects are those especially to be sought, and with these are associated the other specific and beneficial actions of the remedy. Relief is usually forthcoming before the occurrence of "colchicum stools." As already stated, some practitioners trust to colchicum for such aperient effects as may be desirable in any case of acute gout. The objection to this is that, to ensure sufficient purgation in many cases, it is necessary to employ far larger doses of the drug than are needed only to control the pain, and colchicum purging is especially lowering and depressant. As a result of such depression, the system is little able to resist fresh onsets of gout, which are therefore more apt to recur after a short interval.[1]

The experience of most practitioners is that, given with prudence, no evil effects are apt to follow, and no special proneness to renewed attacks is thereby established. Occasional, but moderate, purgation, with mercurials given at bedtime, is of great value in acute gout, especially in sthenic cases, and before the age of sixty. The good effects of colchicum are not exclusively due to its action as a vascular depressant, since other agents which lower blood-pressure will temporarily relieve the pain of gout.

Garrod made some very careful observations in gouty patients

[1] "Drachm doses of the wine may be given with no effect upon the disease, but with sad disturbance to the patient's system."—*W. Gairdner.*

on the action of colchicum in respect of its power over excretion of uric acid and urea. As a result, he affirms that: (1.) It does not appear that the drug produces any of its effects on the system by causing the kidneys to eliminate an increased quantity of uric acid. When it is continued for any length of time, it appears to exert a contrary effect. (2.) It cannot be shown that the drug has any influence on the excretion of urea or other solid ingredients of the urine. (3.) It does not act as a diuretic in all cases, but, on the contrary, diminishes the amount of urine when it induces purging.

I am of opinion that a large part of the beneficial effect of colchicum in gout is due to its decided action on the liver. Powerful cholagogue action necessitates active hepatic metabolism, and with this is secured a more complete disposal of uric acid and other products, which are believed with good reason to be retained in the liver in cases of gout. Dr. Lauder Brunton suggests that colchicum affects the ferments by whose action uric acid is formed, and so lessens the production of that acid. He believes that it also paralyses the sensory nerves, but has no action on motor nerves or muscles. Dr. Latham thereupon suggests that if, in gouty fits, uric acid is irritating the sensory nerves, and through them the more active portion of the vasomotor centre, we may paralyse the sensory nerves with colchicum, so that the uric acid no longer produces its effect, and the paroxysm ceases. The drug, therefore, probably acts in more ways than one, possessing not only specific anodyne properties, due, perhaps, largely to its action as a vascular depressant, but also the power of hastening and modifying hepatic and other tissue-metabolism, together with an eliminant property.

According to Dr. Robertson,[1] of Buxton, whose experience of gouty patients and their treatment has been very large, the action of colchicum is greater and more decided on the local manifestation of gout and the inflammatory nature of the paroxysm, than on the constitutional condition on which gout depends, and of which the local ailment is only a form and development. He regards the drug as a specific for gouty inflammation or gouty localization, and not as influencing the condition of body which is the proximate cause of gout. He, like Halford and Holland, is strongly in favour of waiting till an early attack is well-established before exhibiting the drug. When the fits have been frequent and severe, and the parts are already the seat of chronic gout,

[1] The Nature and Treatment of Gout. Lond., 1845.

the earlier in the paroxysm colchicum is prescribed, the more usefully it acts.

Its influence on the sequelæ of the fit and in diminishing the liability to its return may be best secured by small doses of the drug. These may be continued for a long period, if necessary, without any harmful effects. Sir Henry Holland was in the habit of employing small doses for months at a time—in one case it was given for two years—with entire exemption from gout and benefit to the general health of the patient, who was formerly seldom free for more than two months at a time from an attack. He generally combined small doses of quinine with colchicum, when given over a long period. Sir Thomas Watson was convinced of the value of continued small doses of the drug in chronic gout.

For fifteen years I treated numerous cases of acute gout amongst the out-patients of the Hospital with little else than the colchicum draught of our Pharmacopœia, and I had good reason to be well-satisfied with the practice. In the majority of cases no other plan of treatment answered so well.[1] I have found benefit from long-continued dosage in chronic gout, *e.g.*, five or six minims of the wine or tincture twice a day, or a grain of the acetous extract in pill at night.

Many cases of acute gout may, however, be efficiently treated without colchicum. Bicarbonate and nitrate of potassium sometimes prove serviceable, and, on subsidence of the fit, iodide of ammonium may be given. Mercury in alterative doses may be advantageously prescribed, both during and after the attack in many cases.

The Salts of Salicylic Acid as Anti-Gouty Remedies.—Within the last twelve years the salicylates have been much employed in the treatment of acute and chronic gout. M. Germain Sée was one of the first to publish his experience, and he is a strong advocate of their value, believing that they favour the elimination of uric acid.[2] He became convinced of their superiority to colchicum, which he thought disposed to chronic gout, and regarded salicylate of sodium as of equal value in acute gout as in acute rheumatism. Jaccoud is also convinced of the good effects procurable by this drug.[3]

About the same time, the late Dr. Barclay made trial of sali-

[1] Of this, as, indeed, of many other therapeutic methods, I am often disposed to say with Bianca:—"Old fashions please me best; I am not so nice, to change true rules for new inventions."—*Taming of the Shrew*, Act iii. scene 1.

[2] *Progrès Médical*, 1877, p. 745.

[3] Ibid., p. 528.

cylic acid and salicylates in cases of gout in St. George's Hospital,[1] and expressed the opinion that they had nothing like the same prompt and decided action as seen in acute rheumatism when thus treated. The effects were not so satisfactory as to permit colchicum to be dispensed with. Dr. Barclay found that when a patient was susceptible to the influence of colchicum, the latter had certainly a more definite action in eradicating the disease, but he thought that salicylate of sodium might be of value when the attacks grew more repeated, and colchicum lost its good effect. He pointed out that gout was sometimes grafted later in life on true rheumatism, and that thus salicylates might prove of especial value in such instances.

The experience of Dr. Ralfe is in favour of salicylates in relieving minor gouty manifestations after the subsidence of violent paroxysms, and he prefers them to colchicum.

I have tried sodium salicylate in a considerable number of cases of acute gout, and my experience is that it is in most instances very inferior to colchicum as a drug to relieve the urgent symptoms. I have made inquiry from other physicians, and find their general experience agrees with my own. In a few cases I have certainly met with marked benefit from sodium salicylate when colchicum had completely failed; but I could not predicate the particular case in which one should fail and the other succeed. Dr. Haig is of opinion that if salicylates be used as freely for gout as for acute rheumatism, very satisfactory results will be secured, and he believes that in most cases where it has failed, the drug has not been sufficiently pushed.

The sodium salt is the best to employ. Salicylate of lithium and salicylate of quinine have been employed with alleged benefit. In each of the latter the amount of salicylic acid is probably too small to be the really beneficial agent, and any good effects are presumably due to the lithium or the quinine.

The careful researches of Dr. Haig on the action of salicylate of sodium in promptly removing headache due to urichæmia, and in reducing the tension of the pulse which is common in gouty conditions of the system, are very significant of the general therapeutical value of the drug in gout.

Many practitioners prescribe alkalies, together with salicylate of sodium, and often combine ammonia to counteract its depressing effect on the heart. In such cases it is not possible to judge of the true value of the drug.

It should not be given if the kidneys are unsound, and this is

[1] St. George's Hospital Reports, vol. ix., 1877–78.

a contra-indication which must often be present in dealing with acute paroxysms in elderly and other patients. Albuminuria is sometimes induced by salicylate of sodium in healthy persons, and is aggravated if it already exists in others.

According to M. Bouloumié,[1] salicylate treatment is well-adapted for gout in the young, where there is no debility nor tendency to nervous depression. M. Sée employed three drachms daily for the first three days, two drachms daily for the next three days, and alternated these doses in courses each of three days for three weeks. This treatment must be considered somewhat heroic if it is to be followed as a matter of routine. As with colchicum, it is well to observe the tolerance in each case for the drug, and, in particular, to ascertain the measure of renal adequacy. I have already mentioned the case of a man under my care suffering from acute gout in several joints, with much sweating, who derived no relief from colchicum, but was at once relieved by sodium salicylate given as for acute rheumatism. Another case of chronic tophaceous gout in a woman under my care, in whom acute attacks supervened from time to time, was also markedly benefited by this treatment. Dr. Haig has recorded this case in the St. Bartholomew's Hospital Reports for 1888, vol. xxiv. p. 217. His view is, that salicylate seizes upon the uric acid, and carries it off from the system as a soluble salicylurate by the urine, and he has shown that, while the blood is impregnated with the drug, neither food nor acids taken are potential to induce uric acid disturbances as evinced by headache or overt goutiness. He admits that it sometimes has no prompt action in acute gout. Ordinary acids lessen the solubility of uric acid. Salicylic acid increases urinary acidity, but does not diminish the excretion of uric acid.[2] Salicyluric acid is much more soluble in water than uric acid, and, probably, in dilute acids.

Dr. Haig has shown that uric acid is present with salicyluric acid in the urine passed after salicylates have been taken, and believes that this is due to their action on the uric acid in the blood, and not on that excreted directly by the kidneys.

The action of salicylates is probably effective, both by reason of their chemical properties and by their ability to dispose of the uric-hæmic state. Like colchicum, salicylic acid is a powerful hepatic stimulant, increasing the quantity of bile, and also rendering it more watery. It is also a vascular depressant, and so far assists in allaying the pain of an acute inflammatory fluxion.

[1] *Union Médicale*, May 15, 1879.

[2] Trans. Roy. Med. and Chirurg. Soc., 1888.

It does not always afford relief so rapidly, even when successful, as does colchicum, and not till after a day or two is its efficacy appreciated, at all events by the patient. In some cases, however, it acts promptly and decidedly. I do not think it is likely to supersede the well-established value of colchicum as a prompt deliverer from the agony of a gouty paroxysm; but it is a remedy of considerable power and usefulness, by favouring excretion of uric acid, and preventing other acids in the system from causing retention of the latter.

Ebstein's experience of this agent is not satisfactory. He found that gouty inflammation tended to shift quickly from one joint to another when the patient was kept under the influence of the drug.

Lecorché finds it inferior to colchicum, and without power to shorten the attack. It lessened the pain and violence of the paroxysms, and within a day or two promoted a large excretion of urea, phosphoric and uric acids. This excretion he found to last for three or four days, when gradual diminution set in. He prescribes four to six grammes in the day; and in chronic gout with visceral troubles, save when there is interstitial nephritis, he maintains this medication for months with intervals of a few days after each fortnight. Bouchard is in favour of this treatment for acute gout, but declines to employ it if there be signs of cardiac or renal degeneration.

Professor Latham is a strong advocate of the use of the salicylates in gout with the precautions already mentioned. His theory of their beneficial action is that they seize on glycocine, or its antecedent, and so prevent formation of uric acid. Dr. Noël Paton has also found that salicylates diminish the excretion of uric acid.[1] This view is not in agreement with the observations of Lecorché and Dr. Haig, who found increased excretion of uric acid.

Of the value of medication by salicylates in doses sufficient to secure relief in this fashion, I feel grave doubt. Bearing in mind the marked intolerance of some patients for the drug, and the severe symptoms sometimes produced, I am inclined to think that the remedy may be worse than the disease, and that in respect of the patient—*ægrescit medendo*. The treatment is certainly disagreeable, and many experienced gouty patients would refuse to submit to it.

For some phases of chronic gout smaller doses certainly prove beneficial, and may be combined with alkalies.

Of the use of salicin in any form of gouty disorder I have no

[1] Journal of Anatomy and Physiology, January 1886, p. 26–32.

experience. Antifebrin (acetanilide) is a modern remedy alleged to have considerable power in relieving acute paroxysms of gout. The dose recommended is eight grains three or four times in the day. I have no experience of its value.

Cases are met with in which no relief to the pain of acute gout is afforded by colchicum. Patients are apt, when their advisers fail to secure decided mitigation of their suffering, to resort to some nostrum, and it must be conceded that some of these succeed when regular means prove unavailing. The fault probably lies at our own door. Insufficient dosage with suitable remedies may be a cause of failure. Neglect of purging and of mercurial remedies will certainly account for some inadequacy of bedside art. Patients tell of relief secured by some vaunted nostrum; but the benefit is probably not far to seek in such instances, and consists, generally, in the employment of drugs which both purge and allay pain, the remedies being taken in sufficient amount to secure the needed effect. A fuller dose of colchicum, with a purge of calomel and colocynth, was all perhaps that was needed. We must not follow the patient in his belief that he found in his specific any drug unknown to the regular profession, for no such agent exists, and it is perfectly well-known that the various nostrums for gout are compounded of certain aperients together with colchicum or some preparation of white hellebore.

The measure of relief found by any gouty patient from a vaunted specific is, therefore, the measure of our own inefficiency as clinical physicians. It is probable that none of these specifics of the shops are harmful in themselves; but inasmuch as they are not prescribed by regular practitioners, and are taken by patients themselves on their own responsibility, they are used in excess, and otherwise improperly. I have seen many gouty patients who had thus dosed themselves with several varieties of these nostrums, and some of them appeared the worse in consequence. In these cases, very simple and ordinary treatment for gout quickly sufficed to procure relief.

A fallacy attached to the use of these agents is that no restriction in diet is necessary while suffering from gout, and, hence, the attempt is fruitlessly made to cure the disorder while ordinary or excessive indulgences are continued.

Alkalies in Acute Gout.—The condition of the blood in gouty states has for a long period led to the employment of alkaline remedies to rectify it, and they may fairly be credited with thera-

peutic power of high order. They are when freely diluted rapidly absorbed, and pass through the system mainly by way of the kidneys. Preference is given to the salts of potassium, ammonium, lithium, and sodium, the first two being most used. The employment of alkalies in gout relates to the carbonates, bicarbonates, and citrates, and also to the phosphates of sodium and ammonium. The carbonates and bicarbonates neutralize any free acidity in the alimentary canal before excretion by the kidneys. The neutral citrates and tartrates are discharged as carbonates. Phosphates pass out in the same form. The potassium salts possess diuretic properties, and form more soluble combination with uric acid. Some gouty persons cannot take sodium salts without aggravation of their state. Potassium citrate, acetate, and bicarbonate are the most valuable salts to employ in acute gout.[1] The dose varies from fifteen to thirty grains of each, and should always be given well-diluted. Distilled water, plain or carbonated, is of use when alkaline remedies are prescribed as a diluent, and may be given in conjunction with colchicum, salicylates, or any other treatment in acute gout. Water-drinking unquestionably aids in washing out urates from the blood, and aids all methods used for this purpose.

The key-note of all treatment in any gouty state is to seize upon the uratic excess or stasis in the system, to keep it moving, and to promote its elimination by every channel. Parkes found that potassium citrate caused in gouty cases large elimination of urea and of phosphoric acid, while sulphuric acid was also excreted in excess.[2]

Lithium salts, first introduced by Garrod in 1858, are of great value in gout, but do not enable other approved methods of treatment to be dispensed with in the first instance. They form the most soluble of all salts of uric acid, and possess more neutralizing power than those of any other alkali. Five to ten grains of the citrate or carbonate may be given, alone or combined with potassium salts. Lithia water may be used, containing five grains in ten ounces, to the extent of three or four bottles each day in any case of gout as an ordinary drink. No harmful effects have been observed from its use. On the whole, it is a remedy better adapted to chronic than to acute phases of gout. Hard potable waters are best avoided, as a rule, by the gouty. The lime contained in most of them is commonly noxious, and tends to form

[1] A formula much in use is the following :—℞ Potassii Bicarb. gr.xv. ; Vin. Colch. ℥xv. ; Aq. Menth. Pip. f℥i. M. fiat Haustus ter die sumendus.

[2] On the Urine, p. 297.

very insoluble salts with uric acid.[1] I have certainly observed gouty patients who drink them grow worse and more liable to varieties of uric acid disturbance.

Magnesium salts are certainly useful in acute gout; but after the paroxysm has subsided, it is proper to suspend their use and resort to potassium salts. The latter should be given on an empty stomach. It is often useful to vary the alkali employed during the course of the disorder. Colchicum is a valuable adjunct to them in many cases.

Phosphate of sodium is of particular value in gouty states, providing an excellent and natural solvent for uratic excess in the system. It is tasteless, and acts as a mild aperient.

Dr. Haig finds it almost impossible to procure specimens free from sodium sulphate, and has noted that this impurity retards the desirable effects of the salt to a considerable degree. With the chemically pure drug he has obtained very good results in respect of elimination of uric acid. The dose is from two drachms to half an ounce three or four times a day.

Ammonium phosphate acts much as does the sodium salt, but is beneficial where a slight stimulant action is desirable. It may be given in doses of ten to forty grains three times daily, freely diluted. In gouty glycosuria it is sometimes very useful.

An objection to alkaline treatment is the general depression which is apt to supervene in the system in consequence. Alkaline salts, especially those of potassium, act as cardiac depressants.[2] Hence, it is well to reduce the larger doses necessary at the outset of acute stages, and to employ smaller ones in combination with ammonium salts. To prevent the depressant effects it is desirable, in some cases, to combine quinine or cinchona bark with them. This quino-alkaline treatment is of particular value in chronic gout, and will be again referred to. The profession is indebted to Garrod for this excellent combination.

Sodium salts are better borne for a long continuance, and as existing in the waters of Vichy are of high value as anti-gouty remedies.

It is often desirable to combine several alkalies in one pre-

[1] The good effects of Bath and Contrexéville waters would apparently contradict this opinion, since they contain carbonate and sulphate of calcium. They increase the alkalinity of the blood, but not of the urine.

[2] Potassium salts tend to arrest the heart's action in diastole, to inhibit the rhythmical action, and to render it less and less susceptible to the effect of continuous faradization. The salts of ammonium are less powerfully depressing, and those of sodium least so. (*Ringer.*)

scription, as potassium and sodium, or either with lithium salts and ammonium.

Diluent drinks should always be taken during a course of treatment by alkalies.

I have treated many cases of acute gout with alkalies alone, and have often been well-satisfied with the result. Where colchicum has been abused, or is undesirable, the alkalies may be given in doses of from fifteen to twenty-five grains every four or six hours for two or three days.

Iodide of potassium is hardly available in primary or early paroxysms, but is of much service in various phases of subacute and chronic gout, and in gouty cachexia.

After acute attacks of arthritis, it is a good practice to strap or bandage the joints for some time. Subsequently, gentle friction, after soapy ablution daily, with any simple liniment, is advisable; and if deep-seated pains remain, warm pediluvia at night, containing half an ounce of compound tincture of iodine, are to be recommended, or the compound mustard liniment may be used.

Dietetic Treatment of Acute Gout.

In respect of the diet suitable in any particular case, regard must be had to the special circumstances, age, and habits of the patient. The dietary for a sthenic case in a young and plethoric man may not be the same as for an asthenic case in an elderly and broken-down man. Hence, there must be variation according to circumstances.

In a primary paroxysm in an over-fed man, it is important to prescribe a sparing diet, consisting chiefly of light and diluted nutriment. Farinaceous food, bread and milk, simple rice, tapioca, semolina, or sago pudding, weak tea, cocoa-nib infusion, thin mutton or chicken broth, milk, and arrowroot or gruel may be given. Jelly and all gelatinous foods are objectionable, as they tend to furnish glycocine. Alcoholics in all forms should be withheld, unless specially indicated. Boiled whiting or sole may be allowed after a day or two if there is positive hunger, and a morsel of mealy potato. Meat and nitrogenous foods generally are contra-indicated. Without doubt, cases of the kind under consideration do best on the diet just mentioned. In elderly persons, accustomed to free stimulation, it is important not to allow the patient to lose power, and the dietary may be improved by a little white meat, chicken, or fish, and a small allowance of mature brandy or whisky, well-diluted. This should not exceed two ounces in

the day, unless distinctly called for. The state of the pulse and of the heart's action, together with that of the kidneys, will aid in the determination as to the quantity of stimulant needed. If there is more than a cloud of albumen in the urine, it will be well to limit alcohol as much as possible, and the same applies to any undue pulse-tension.

It has lately become a fashion to give port wine in cases of acute gout. I am sure that this is wrong, and a practice to be deprecated. It may please the patient, but it is a mischievous prescription. Without question, such a practice may be sometimes necessary and very useful, but it must be reserved, with other varieties of treatment, for particular cases. In any case, it is the patient, and not the disease, that is to be treated, and no routine habits are safe at the bedside. Within the domain of rational therapeutics, there can be no fashion in this or in any other plan of treatment.

2.—Medicinal and other Treatment of the Gouty in the Intervals between the Paroxysms.

When an acute attack of gout has passed off, there commonly remains some general debility, although the patient may feel in better health than he has done for some time previously. Much will depend on the treatment during the fit, and still more on the individual habits and proclivities of the patient, in respect of the future conduct of the case. If the previous habits have been bad and gout-inducing, and the patient be possessed of sufficiently strong will, much may be secured if a better manner of life be followed. Unfortunately, in most cases, by the time gout is overtly manifested, a patient has fallen into routine habits, which he is commonly averse from altering to any material extent. His tastes and appetites are formed, and he is little desirous of being dieted, or converted into a valetudinarian. So much the worse for him, since his future welfare depends on a life guided by rule. The balance of health is dependent on no one circumstance, but is entirely maintained by the observance of many, often small, matters. Hence, to treat the gouty proclivity, or state, with success, demands minute attention to most of the points which concern the course of daily and nightly life. Inattention to some of these seemingly trifling matters may prevent the full measure of benefit that may be hoped for, and often secured.

The main lines of treatment for those goutily disposed are directed to the diminution of uric acid generally in the system,

and to the rapid circulation and removal of any excess, stasis, or deposition of that which is formed. In addition to this, attention must be paid to the general nutritive processes, and in particular to the evolution of nervous energy. Under the head of Preventive Treatment, I propose to discuss these points more at length. The management of the patient after an acute paroxysm now demands attention.[1] In many cases it will be found necessary to alter existing habits of life, more especially in regard to brain-work, diet, and exercise. After earlier attacks, it is imperative to insist on bodily activity, regular hours for mental work, and strict dietetic regimen. In cases occurring early in life, with strong hereditary proclivity, it may be important to forbid the use of all alcoholic liquors. No routine practice can, however, be pursued even in cases of this class, for not a few of such patients are far from being robust, and may have weak hearts, a feeble circulation, and sluggish nutritional power. If there be vigorous circulation, and anything approaching hæmic plethora, and if the appetite is good, abstention from alcoholic drinks may be tried for a year or two, the patient being kept under observation.

Later in life, it may be well to resume the use of a little wine. Early hours at each end of the day should be the rule, and walking exercise or equitation regularly practised. The patient should walk not less than three or four miles, or ride not less than an hour daily. The ordinary cold hip-bath should be taken, in winter with the chill off, followed by vigorous towelling, prompt dressing, and breakfast. Some exercise should be taken in the forenoon, and any occupation followed till luncheon. The character of this meal must vary according to the exercise, brain-work, and the amount of food eaten at breakfast. If the latter meal is slender, a better luncheon is necessary. Women, as a rule, eat most heartily at midday, and men make a better breakfast than the former. Butcher's meat should be taken but once in the day, presumably at the evening meal. No wine or stimulant is desirable at luncheon. Occupation and exercise should be carried on in the after part of the day, and dinner taken not too late. No food is requisite after the latter meal; only weak tea, plain water, or some mineral water should be taken two hours afterwards. No coffee should be taken at night. Not less than seven, and no more than eight hours should be spent in bed. Curtailment of sleep is very injurious to the gouty, and excessive

[1] "The absence of typical attacks of gout is no proof that the gouty process, as such, is cured."—*Ebstein, op. cit.*

indulgence in bed is equally harmful. Regular habits and equability of life, as far as possible, are to be maintained. With the wealthy and well-to-do there will, probably, always be a temptation to break in on the even tenour of such habits. "Exposure to luxury" is the most serious and malign influence to be combated. All excess is surely harmful for the gouty, whether in harmless or pernicious things. Too much study and mental strain are about as bad as sheer idleness or mental vacuity; the morality of neither can be justified.[1] *Ne quid nimis* must be the motto of a gouty man's habits of life. Moderation in all things, in work and play, in eating, drinking, and in exercise, is the key-note of the physician's instructions to a goutily-disposed patient. Any break in the equability of life suitable for such a patient is certain to carry penalty with it, and to induce some degree of disturbance of health which it may take long to remove. Great care is necessary in exercising restraint at public or private dinner-parties. The habitual diner-out is very apt to develop or acquire gout. The ordinary fashionable dinner, though now, happily, more refined and less ponderous than formerly, is a terrible ordeal for a gouty man. If well-advised and prudent, he can but pick his way warily through the *ménu*, and must avoid especially excess of meats and wines, and any indulgence in the sweet courses. If any indiscretion be committed, it must be rigidly atoned for afterwards, even if the *hesterna vitia* of the dinner-table do not compel more strict abstinence for some days subsequently. Not more than one such dinner in a week can be safely indulged in; and if the claims of public or official life compel greater frequency than this at certain periods, a rigid habit of extreme abstinence on each occasion should be forthwith begun, and never deviated from.

The best dietary is, in truth, that which is most suitable for the dyspeptic. The food should be well but plainly cooked. It is wise to avoid all things boiled, baked, or stewed. Pastry is

[1] The effects of brain-work are, however, not always harmful, even during an acute attack. The case of Lord Palmerston was one in point. One of the traits of this remarkable man was "his wonderful power of *mastering*—I might call it *ignoring*—bodily pain. I have seen him under a fit of gout which would have sent other men groaning to their couches, continue his work of writing or reading on public business almost without abatement, amidst the chaos of papers which covered the floor as well as the tables of his room. To Lord Palmerston work was itself a remedy. The labour he loved 'physicked pain.' No anodyne I could have prescribed would have been equally effectual in allaying it, or, as I may better say, in lessening that sense of suffering which is always augmented by the attention of the mind directed to it." —*Recollections of Past Life*, by Sir Henry Holland, Bart., M.D., p. 197. London, 1872.

bad. All fats and fatty tissues should be sparingly used when roasted or browned; otherwise, fats are not unwholesome for the gouty. Suet and bacon fat are the least harmful. Oily fishes and game are unsuitable. Roasting and grilling are the best methods of cooking. White fish is harmless in any quantity likely to be taken, but it cannot be long-continued instead of butcher's meat, as most patients tire of it. Lean meats, in moderation, are not hurtful, but should be taken with but little wine, and plenty of diluents subsequently. Sweet-bread and thymus gland of the calf, liver, ox-tongue, chicken, turkey, and tender game birds are all admissible, the skin and fat of the latter being carefully avoided, that of the duck and goose being especially bad. Lightly boiled or poached, but not fried, fresh eggs may be taken. Sauces of any kind are not admissible, or only sparingly. Puddings and sweet courses should be eschewed, and a small portion of savoury omelette may replace them occasionally, or a morsel of not over-ripe cheese with toast or "pulled" bread may conclude the dinner.

My friend Mr. H. W. Jackson, formerly in practice at Lewisham, long a martyr to gout, has found immunity from all symptoms by adherence to the following dietary. He had previously tried every method of treatment and every variety of diet unavailingly:—

Dietary.

A.M.		
8.15	10 oz.	hot water.
9.0	16 „	coffee with hot milk.
	6 „	bread and butter (six drachms); four lumps of loaf sugar; salt.
P.M.		
1.0		Meat (about half a ration) with little fat—no browned fat; potatoes, with or without green vegetables; a little mustard, if any; no pepper.
	11 oz.	cold water, freed from lime by boiling.
5.0	11 „	hot water.
6.0	16 „	tea, with pinch of soda bicarbonate.
	6 „	cold milk in the tea.
	6 „	cold milk alone; bread and butter (six drachms); toast and butter; loaf sugar (two lumps); a large piece of cheese; salt.
9.0	11 „	hot water.
	93 oz.	total of fluids.

A larger allowance than is customary of pure water or simple mineral water should be taken. Some persons rarely drink any water in the course of the day. Not less than a pint of

pure water should be added to the ordinary allowance taken, and it may be drunk hot after meals. I think it is a good plan to take half a pint of water in slow sippings in the hour before retiring to bed. The common habit of adding some spirit to this is bad, and, even if desirable, must be a matter for the physician to decide. For younger persons there can be no question that this is a harmful practice.

Tobacco-smoking in strict moderation, at a short interval after meals, I do not believe to be unwholesome for the goutily disposed, provided it causes no dyspepsia and disagrees in no obvious manner. The least excess is harmful. Cigarette-smoking is the worst and most insidious form of the practice.

To regularity of daily habits and general equability of life, must be added the practice of chastity. Sir Henry Halford quotes, and lays stress upon, Pliny's word for this special virtue —"*sanctitas.*"[1] It is certain that sexual excess is provocative of gouty paroxysms and manifestations, and I feel sure that such indulgence early in life, by its general enervating influence, is potent not only in determining early and severe attacks, but also in the premature induction of gouty cachexia. As may be well-understood, nothing is more harmful for those predisposed to this malady than combined and inordinate indulgence in venery and wine.

Arthritic Obesity.—Ebstein is of opinion that a tendency to obesity is an untoward symptom in those of gouty habit, and believes that fatty deposit affords a favourable soil for the disease; further, that by checking this tendency we may remove the most active exciting cause of gouty symptoms. The dietary recommended by him to prevent such obesity includes meat and fat, and reduces to a minimum carbo-hydrates. Thus, he forbids sugar, pastry, potatoes, and beer. His view is that carbo-hydrates protect albumen from destruction, and that the portion of the latter which is not taken in, or metabolically disposed of, is added as fat to the system. Fats also protect albumen from destruction, but in far less degree than other carbo-hydrates, and that part which is decomposed with a corresponding use of fat is transformed completely, and is not stored up in the body in an intermediate state as fat.

I am not prepared to agree with the opinion that there is any special danger for the gouty, more, that is, than for other persons, in an obese tendency, unless it be decidedly manifested. It is, perhaps, a less common deviation in England than in Germany.

[1] "Pedum dolorem fregit abstinentiâ et sanctitate."

The tendency to gouty glycosuria is recognized in this connection. As to the utility of the dietary recommended by Ebstein, I am in full agreement. Fat in any form readily digested is good for most gouty patients, but there are many who cannot, and will not, take it save in the form of fresh butter or bacon. With respect to beer, the noxious principle is probably rather to be found in its free acid than in its carbo-hydrates. Both together, however, are as bad as can well be conceived.

The treatment of arthritic obesity is hardly amenable to other measures than those which are commonly recognized as useful in other forms of polysarcia. Where this peculiarity of nutrition is early manifested, it is important to secure active habits of life, and amidst rural surroundings, if possible. Exercise, riding on horse-back, and open-air life are all advisable. The dietary should consist of fish and butcher's meat in fair proportion, with fat in moderation. Hydro-carbonaceous matters should be limited, especially too great indulgence in bread and amylaceous food. Sugar must be largely abstained from. Liquids should be restricted to absolute requirements. Some form of alcohol is commonly advisable (red Bordeaux wine with a little water is the best form to employ), and should be taken once a day with the principal meal. Holiday periods should be passed in alpine or sub-alpine regions, with regular and active exercise, and any games that can be played be assiduously cultivated. In the case of adults, resort to the Spas of Marienbad and Carlsbad for several successive seasons is advisable.

3.—Treatment of Chronic and Irregular Gout.

Evidence is not wanting to prove that, if the victims of gout and gouty states are not adequately treated, the disease is apt to make mischievous progress, to lead to crippling and the onset of gouty cachexia with wide-spread textural degenerations. Hence, not only are acute fits to be carefully treated, and the general health restored, as far as possible, in the intervals, but all indications of goutiness are to be met as they arise. Successful management of the multiform phases assumed by the malady demands accurate recognition of the gouty element always and everywhere, and a large experience in clinical medicine.

The general principles of treatment in chronic gout relate more particularly to the condition of the blood, of the nervous system, and of the general textural nutrition. All measures, therefore, which tend to maintain the best bodily health will be favourable

to the patient, and render him as little vulnerable as may be to the various assaults of the disease. The better the constitution, the less mischievous the gout. The malign combinations of diathetic states in any individual must be especially taken note of. Activity of habits must be fostered. Indulgences and ease-taking must be resisted. Exercise in all forms is to be practised. A good action of the skin and a vigorous circulation must be maintained. Too much sedentary and town-life should be avoided. Equable brain and muscular energy are desirable. Mental and bodily depression from any causes are harmful. Cheerful surroundings should be sought, and mental irritability and outbursts of temper should be restrained. No violent change in any wholesome habit of life should be permitted.

Hence there is much to be accomplished before any special medication is resorted to, and the patient must be impressed with this, and not be encouraged to rely on physic alone for relief from his varied ailments. The belief that the physician can conjure away their troubles with "something in a bottle" is, unfortunately, not confined to patients of the humbler classes, and a better philosophy than this has to be expounded daily to those who should know better. Any who fail in this duty to their patients will as certainly often fail to afford all the relief that is rightly procurable. Under suitable treatment, applied to meet all the requirements of individual cases, long immunity from attacks of gout may be secured, and the paroxysms may be reduced in intensity.

The tendency to urichæmia and localized deposits of urates is best medicinally treated by courses of alkaline remedies given at intervals, together with occasional aperients containing mercury and colchicum, or with a hepatic stimulant such as euonymin. Lithium salts are useful, and may be taken in the form of lithia water. Where a saline aperient is advisable, crystallized Carlsbad, or Homburg, salts may be taken in doses of two drachms in eight ounces of hot water before breakfast. Any of the "bitter" waters act well, such as Hunyadi Janos, Friedrichshall, Püllna, or Rubinat in appropriate doses. The disorder of the liver associated with too free production of uric acid, or induced by storage therein of that acid, is well-treated by these salines taken for some mornings in succession, and their efficacy is largely due to the sodium and magnesium sulphates contained in them. A larger amount of water-drinking than is customary is useful in most cases of chronic gout. For subacute attacks of articular inflammation, colchicum may be advantageously used in small doses. For many phases of chronic gout in and around

joints, and to keep in check degenerative processes in many tissues, I believe the iodides to be of much value. The sodium, lithium, potassium, and ammonium salts may all be used. They tend to alleviate pain in many situations. Their action is sometimes depressing, and this may be prevented by combining them with cinchona bark or with quinine and nux vomica; or tincture of iodine may be given with ammonium chloride and spirit of chloroform.[1] I have found citrate of potassium, iodide of lithium, and nux vomica a useful prescription. Three or five grains is a dose of the lithium iodide. Diluents should be freely used when these salts are prescribed, and I know no better one than a pint (taken daily) of the compound decoction of sarsaparilla or of hemidesmus, especially if the patient be frail and poorly nourished. Smaller doses of the latter drugs are, I believe, of no avail whatever. Their value is only seen when used as "diet drinks." From three to five grains of potassium, lithium, or sodium iodide is a sufficient dose.

Cinchona bark and quinine are very useful in chronic gout, promoting better digestive and nervous power, and nux vomica and strychnia are also of high value. Quinine may often be advantageously given with bicarbonate or with iodide of potassium in from two to five grain doses. Guaiacum is in repute in cases where the circulation is languid, and in atonic forms of gout. It may be combined with cinchona, colchicum, and with iodides. The great objection to it is its unpleasant taste. I believe it to be of sufficient value to warrant the endurance of this distasteful quality. In the form of lithium guaiacate, the effects of guaiacic acid may be secured in pill, two to five grains being the dose, made up with glycerine and water, and administered twice or thrice in the day. Guaiacum may be taken with benefit over long periods, and it is often sufficiently aperient to be helpful in many cases. Garrod highly recommends a powder composed of the following ingredients, and I know that it is valuable:—

℞ Pulv. Resinæ Guaiaci ʒvi., Pulv. Cinchonæ Flavæ ʒi., Ammonii Carbonatis ʒij., Potassii (vel Lithii) citratis ʒij., Potassii Iodidi ʒi., Pulv. Cormi Colchici ʒi. M. ut fiat Pulvis. Sig. Forty grains for a dose in a wine-glassful of peppermint water, to be taken once a day, continuously or in alternate weeks.

This combination smacks of polypharmacy, and may, therefore, shock the therapeuto-purists of the present day. In reply to any objections on this score, I would affirm that our business, as

[1] Dr. Mortimer Granville has especially recommended the employment of free odine in gout.

physicians, is to cure our patients, and that our duty is to use remedies in any fashion which will best secure that object.

I think it is not improbable that both alkalies and iodides help to prevent degenerative processes in various tissues, and so stave off the worst forms of pulmonary, cardiac, vascular, and renal disorders so commonly associated with gout.

Benzoates of sodium, ammonium, and lithium have been employed in the treatment of chronic gout during the last twenty years with a varying degree of success. Garrod has extolled them, but other observers are less enthusiastic as to their merits. Benzoic acid is excreted by the kidneys, after uniting there with glycocine, or its antecedent, in the form of hippuric acid, thus preventing formation of uric acid. It is especially to be noted that benzoic acid contains no nitrogen, and while undergoing transformation in the system into hippuric acid it incorporates that element. According to Dr. Noël Paton, benzoate of sodium diminishes secretion of uric acid. Benzoate of lithium I have frequently prescribed in chronic gout, and sometimes with benefit. I often combine it with tincture of nux vomica. The dose is from eight grains to half a drachm. The benzoates may be combined advantageously with phosphate and carbonate of sodium, as recommended by the late Dr. Golding Bird.

From time to time hæmatic medicines are called for in the treatment of chronic gout. Where anæmia, cardiac debility, or albuminuria is present, the value of iron as a remedy naturally occurs to the physician. This is found to disagree in many cases, and to induce recurrence of gouty attacks. It has been found to check the elimination of uric acid. Small doses are, however, often well borne, especially of the non-astringent preparations. Three or four grains of reduced iron or of the ammonio-citrate, tartarated, or carbonate of iron may be given. The ammonio-citrate may be prescribed with a few ounces of Nassau Selters water; or any chalybeate water, such as that of Spa, St. Moritz, Pyrmont, or Tunbridge Wells, may be taken in small quantities. Some saline or other aperient is especially necessary for the gouty while taking a course of iron, and may prevent headache and other disturbance as consequences of it.

If iron disagrees, it is not easy to find a substitute.[1] Dr. Munk informs me that he sometimes prescribes manganesium salts in such cases, believing that they act as tonic and hæmatic

[1] Dr. Haig found that iron caused retention of uric acid in the system after twenty-four hours had elapsed. Urate of iron is quite insoluble, and so is urate of lead.

remedies. Dr. Haig, at my suggestion, kindly undertook to observe the influence of manganesium on uric acid excretion, and he reports that it acts much in the same way as iron and lead. When first taken, it causes retention of uric acid with pricking pains in the joints; but later on, about the second or third day, it causes very decided intestinal irritation, and the results of this (fall of urea and acidity) overcome its primary retentive action, and cause increased excretion of uric acid. He took the sulphate, and afterwards the precipitated oxyde, each in doses of ten grains three times a day. Retention of uric acid and joint-pains were most marked with the sulphate, but the oxyde also caused them. He remarks:—"I should expect that, given in gout, they would slightly increase the pains at first, and then, when intestinal irritation and falling acidity caused *plus* excretion of uric acid, they would relieve them; this being, as I have suggested, the way in which colchicum acts."

Clinical experience must, however, be the ultimate appeal as to the real value of any drug, and I conceive that salts of manganese may prove useful as hæmatics in doses smaller than are needed to irritate the bowels. There is evidence of their action as emmenagogues in cases where iron is commonly useful in improving the quality of the blood, and I see no reason why they should not be employed in chronic gout. For each part of iron in the blood there is about one-twentieth of manganese, and the latter is contained in potatoes and many other kinds of food. Three to ten grains of the sulphate or of the precipitated oxyde may be prescribed for a dose. Tabloids, containing two grains of the latter, are available.

Arsenic is, perhaps, the best substitute for chalybeate treatment. This drug has, without doubt, a powerful effect for good in a large number of ailments connected with the arthritic habit of body. As a nutrient and nervine tonic, and as a hæmatic remedy, it has a high value. It is of particular use in the neurosal condition associated with gout, and also in cases of cardiac debility and albuminuria met with in gout and its cachexia. It may be combined with alkalies or with iron, and beneficially given over long periods with short intermissions. The following formulæ I have found very serviceable:—

℞ Tinct. Ferri Perchloridi f℥iss., Liq. Arsenici Hydrochlorici f℥ss., Liq. Strychninæ Hydrochloratis f℥ss., Syrupi Tolutani f℥vi., Aquam Destillatam ad f℥viii. M. ut fiat Mistura. An eighth part for a dose twice daily after the principal meals.

℞ Potassii Bicarbonatis, Sodii Bicarbonatis, aa. gr.lxxx., Liq. Sodii Arseniatis f℥ss., Tinct. Nucis Vomicæ f℥iss., Aquam Chloroformi ad f℥viii. M. ut fiat Mistura. An eighth part to be taken twice daily two hours after the principal meals.

℞ Ferri et Ammonii Citratis gr.xl., Potassii Bicarbonatis ʒij., Liq. Arsenicalis fʒss., Infusi Calumbæ, Aqua Menth. Pip. aa. fʒiv. M. ut fiat Mistura. An eighth part to be taken twice daily between meals.

Iron may be combined with iodine in cases where the latter is thought desirable. I prefer the mixture of tartarated iron with potassium iodide to the officinal syrup of iodide of iron.

Sulphate of nickel has been employed as a tonic, but I have no experience of its use.

If the action of the bowels is defective in cases of chronic gout, it is best to avoid too frequent resort to aperients of a depressing character. In plethoric persons who live well, and take insufficient bodily exercise, great advantage will be gained by a periodical aperient containing mercury, taken over-night at intervals of three weeks, followed by a dose of any natural bitter water the next morning. In cases marked by general asthenia, a simple "dinner-pill" will commonly suffice, containing aloes, nux vomica, and extract of anthemis. Cascara sagrada, or small enemata of glycerine (fʒi.–fʒij.) will be found serviceable in such cases. Small doses of castor oil are of use, taken at night, or equal parts of this and oil of sweet almonds. A pill taken at bed-time containing four grains of compound rhubarb pill with one of quinine, will sometimes avail to secure a return of natural daily evacuations. The objection to strong purgings so commonly mentioned by the old authors on gout has been shown by Dr. Munk to have arisen, probably, from the fact that in Sydenham's time, and for long afterwards, purgatives were too commonly violent and irritating, and very different from those employed in the present day.

4.—Local Treatment of the Joints in Chronic Gouty Arthritis.—Treatment of Tophi.

In connexion with repeated attacks of gout in any joint, certain textural changes are apt to remain, and lead to discomfort and deformities. The œdema, which soon passes off in acute and early attacks, may tend in later and subacute paroxysms to linger. This is found in both upper and lower limbs, perhaps more so, and naturally, in the latter. It may be aided in dispersion by frictions and moderate pressure; liniment of soap or of camphor may be rubbed in, and domet bandages skilfully applied. Elastic stockings, and suitable position, are sometimes necessary for the lower limbs. Friction and electrical stimulation

are of great value in many of the local troubles of chronic gout, and will be subsequently referred to.

Stiffness is a common result after uratic arthritis, and may be present in all degrees, from the slightest up to firm synostosis (true ankylosis). Partial or false ankylosis is commoner than that which is true, and may be due to fibroid thickening, bony outgrowth, and to uratic incrustation of bones, tendons, or ligaments, or even to all of these combined. Friction is very proper in such cases, and some stimulating liniments may be used with benefit. One of the best is the linimentum sinapis compositum, recommended by Garrod. Passive movements and friction, so far as they can be borne without pain, are to be employed. Tincture of iodine and small strips of cantharides plaster are of great use in restoring movement and dispersing deformities. The latter is to be applied only as a "flying" blister, and may be repeated several nights in succession. Menthol,[1] oil of peppermint, and cocaine[2] are local remedies of value, if there be much pain in connection with stiffened joints, also camphor and chloral-hydrate in mixture. Proper support for the arms and the gout-stool are necessary in these cases.

Where large uratic incrustations and deposits occur, I would enforce caution in undertaking any kind of treatment. It is best not to be too active in endeavours to remove these. Tophi in the ears and other superficial parts sometimes drop out spontaneously, or are picked out by the patient. Larger masses are apt to burst of themselves, a small diffluent collection usually pointing in some direction previously. Bread-poultices may be applied, with unguentum resinæ spread over them. Compresses wetted with alkaline solutions are also available, in which bicarbonate of potassium, carbonate of lithium, and iodide of potassium are the active solvent and dispersing agents. Five grains of any of these salts in an ounce of distilled or rose water is sufficient.

All interference with the knife is to be strongly deprecated in such tophi as have deep connections. I have known serious mischief follow a cutting operation. In Fig. 12, p. 80, is depicted the linear scar of an incision made into a large tophus, which was sufficiently inviting, though not at my instigation, to a surgeon's scalpel. The subject of it had a narrow escape for his life in consequence of erysipelas. When the uratic discharge is scanty, it is also bad practice to try and press out more than flows spon-

[1] ℞ Menthol ʒiss., Linimenti Saponis ℥iij. M. ft. Linimentum.

[2] ℞ Cocaine ʒss., Acidi Borici gr. x., Aq. Destillatam ad fij. Solve et M. ut fiat Lotio. To be applied on lint.

taneously. No probing or use of caustics is justifiable. Erysipelas and gangrene may follow meddlesome therapeutic efforts.

Sometimes, a bursal sac containing tophi proceeds to suppuration, as over the olecranon, and in this case we have to deal with an abscess incidentally containing gouty deposits. The ordinary rules of surgery must, of course, be followed, and the matter let out. These cases usually heal well.

The action of the skin is to be maintained by warm clothing and regular exercise. The Turkish bath is of value in averting gout, and when it agrees and is taken with proper precautions, it may be recommended. I should not advise its employment in patients over fifty years of age, and, in any case, not more frequently than three times in the course of a month. It is, however, a remedial and preventive measure for robust persons whose gouty manifestations are of sthenic character. Where signs of cardiac, vascular, and renal degeneration are present, the use of Turkish baths is to be forbidden.

Warm baths are, however, of great use, and may be employed twice a week. I shall reserve what I have to remark on the use of baths and mineral waters in the treatment of gout for another chapter.

Warm pediluvia are sometimes found to be soothing in cases of lingering gout in the feet. One experienced sufferer told me of the great relief he obtained from a foot-bath containing an ounce of carbonate of potash and half an ounce colchicum wine in three pints of water. In his case the application of six leeches to a great toe-joint had failed previously to afford any relief.

5.—Treatment of Retrocedent and Incomplete Gout.

From a clinical point of view retrocedency of gouty processes may be best regarded as occurring both suddenly and acutely, and also in a quieter and more abiding form as substituted or irregular manifestations.

(1.) Sudden retrocedence is commonly a violent process, and sometimes, as in the case of metastasis to the brain, heart, or stomach, sufficiently alarming both to the patient and the practitioner. It is important to recognize the gouty element in each instance, if treatment is to be efficient.

The therapeutical indication is to afford relief to the oppressed organs by the reinduction of a regular and articular paroxysm. The most valuable means of promoting this is the hot pediluvium with mustard-flour in the proportion of half an ounce to each

gallon of water. Where this is undesirable, mustard-poultices may be placed round each great toe and instep. The urgent symptoms are best relieved by some diffusible stimulant, such as æther or brandy, with or without opium, according to the degree of renal adequacy.

The two varieties of gastric gout demand different treatment. In the milder form occurring in chronic gout, without pyrexia, where the pain is relieved by pressure, and there is no vomiting, but rather cramp and sinking sensation, emetic action may be encouraged by draughts of warm water, containing a little alcohol. Sinapisms should be placed over the epigastrium, and applied to the joints that have previously suffered. In the severe inflammatory form, opium is called for, and stimulants must be withheld. Leeches or a blister should be applied to the epigastrium, and effervescing draughts with potass and hydrocyanic acid be given. The gout should be recalled to the joints. Moderate purgation with calomel and compound colocynth pill is commonly advisable.

Sinapisms to the epigastrium are useful in cases of metastasis to the heart or stomach. If much flatulency is present, as is often the case in a gastric or cardiac crisis, æther, with spirit of cajuput and camphor or peppermint water, is of value, and, sometimes, stimulants are needed in large quantity, owing to collapse and profound cardiac failure. In a plethoric patient with gouty cerebral metastatic crisis, where stupor and mental confusion prevail, or coma supervenes, and where the pulse is unduly firm, venæsection may be advantageously practised, and from ten to twenty ounces of blood be let.

There is need for care in diet for some time subsequently, and no solid food should be given for several days.

With the return of regular articular gout, there is commonly complete relief to all the urgent symptoms. If gastritis or enteritis is not completely relieved, appropriate treatment for these conditions must be applied, liquid food being given in small quantities, and, as medicine, sodium bicarbonate with bismuth and dilute hydrocyanic acid.

(2.) In the second class of gouty metastases, the symptoms are not urgent or violent. The patient suffers from incomplete or misplaced gout. This may follow the suppression of some skin-eruption, or result from any cause capable of inducing gout in a predisposed subject, whose health is less vigorous than formerly. It has been shown that atonic gout is the form most likely to be aberrant or retrocedent. The line of treatment to be pursued is that of promoting a regular gouty articular process, and the same

method must be followed in cases of suppressed gout. Any skin-disease is best neglected, or it may even need to be recalled into activity by some mild irritant. Patches of dry eczema on the limbs, when not active, or when subdued by treatment, are sometimes found to alternate with some renal difficulty; while those on the trunk may subside and induce pulmonary trouble, such as bronchitis or asthma.

Where a gouty paroxysm is felt to be imminent and yet does not come on, an articular attack may fairly be encouraged, if the vexatious symptoms fail to yield to appropriate general treatment by medicine and by diet. This may sometimes be promptly accomplished by the prescription of a pint of champagne and a subsequent hot pediluvium. Other wines, such as port or Madeira, are effectual for this purpose, but are less swift in operation. The mode of action here I conceive to be the reduction of the alkalinity of the blood to a point whence some decided uratic precipitation is rendered certain, and the irritant to the extremities determines the localization of the gouty outburst.

The treatment subsequently is that for ordinary gout. Colchicum is commonly effectual in such cases, and need not be given in large doses, but rather in small ones over an extended period. It is important in all cases of gout with retrocedent tendency to bear in mind the attendant asthenic state, and to endeavour to restore the general health to the highest condition possible. Appropriate tonic medicines and suitable climatic change, with recourse to some mineral waters, are plainly indicated.

6.—Treatment of Special Disorders Dependent on the Gouty Habit.

It is essential that the existence of a gouty dyscrasia be recognized in any case where it exists, and to determine this factor requires skill and experience. I have sufficiently insisted on the importance of discovering gouty taint in cases where no overt articular attacks have occurred, and of recognizing *goutiness* apart from *gout*, commonly so called; and I have also tried to indicate the directions wherein such knowledge may be certainly gained.

As may readily be supposed, many diseases occur in the gouty which are quite unconnected with their special habit of body, and in some of them it is hardly necessary to modify the treatment in relation to that habit. A gouty tendency tends to modify, not seldom, the features of other implanted disorders, and often affords grounds both for prognosis and for treatment.

I propose to discuss methodically the treatment of special gouty disorders according to the several systems of the body. It will be convenient, however, to refer to some gouty ailments apart from a strict adherence to this method. Thus, I shall treat of asthma under the head of the respiratory rather than that of the nervous system, and of angina pectoris as a cardio-vascular rather than a neurotic ailment.

Nervous System.—The gouty affections of the nervous system have already been shown to be numerous, and they certainly cover its whole area. In many of them, an attack of overt gout is the natural cure, and, not seldom, this occurrence is the first feature which discloses the true nature of the ailment in question. This is certainly the case in acute mania, melancholia, and hypochondriasis. Attention to the condition of the functions of the liver and to the state of the circulation is of high importance in determining proper treatment. Where the cardio-vascular system is degenerate, purgatives, which otherwise are of value, must be cautiously employed. With a firm pulse and a hard belly, purgation is very advisable, and calomel is indicated.

Alkaline remedies, such as sodium phosphate and potass salts, are efficient, also ammonium chloride in full doses. The action of the skin should be stimulated. Butcher's meat is to be given sparingly, and fish, fats, farinaceous and vegetable food, with milk, are advisable. Exercise in the open air is necessary as soon as practicable. Any articular symptoms call for ordinary treatment by colchicum or sodium salicylate. The restraint and beneficial influences of a well-ordered *maison de santé* are, of course, necessary in acute mania and profound melancholia. Minor degrees of hypochondriasis and many phases of psychopathy may be best treated at home, or by change of scene amongst suitable surroundings. Regular gout may be induced to alight in the extremities in some of these cases by stimulating pediluvia, mercurial purgatives, or by a full dose of wine, when relief will come to many distempered conditions if dependent on lurking gout. The condition of the renal functions must be ascertained in any case, especially with reference to the employment of opium.

Insomnia.—Where this is dependent on any obviously direct gouty condition, appropriate remedies must be given to meet it. The diet and whole regimen require revision, special attention being paid to the last meal taken. Too long a fast before retiring to rest is harmful, but a heavy meal is best avoided. Beef-tea or any animal broth, cocoa, or arrowroot with a little brandy,

are good taken an hour before bedtime, if there has been no late dinner. Sometimes, a little fluid extract of meat taken in cold water will induce sleep. Hypnotics constitute the last resort, and the best success is attained without them. The bromides are the least harmful of such agents, but should be given early in the day in order to secure their soporific effect at night. Paraldehyde is useful where the heart is weak, and may be prescribed in doses of thirty or fifty minims.

Where there are acidity and gastro-duodenal dyspepsia, or slow digestion—common causes of insomnia in the gouty—a drachm of compound rhubarb powder in peppermint water at bedtime is very efficient. Exercise in the open air, driving especially, is a powerful aid to sleep, and bed should not be too early sought. A dull book read monotonously by some one is often effectual in promoting sleep. Opiates are to be shunned, and I am confident in affirming that they can safely be dispensed with. In some cases cannabis indica alone, or with henbane and bromides, is very useful. Chloral hydrate is best avoided, if possible. Monobromated camphor (gr.ii.–gr. x.) or bromide of lithium (gr.xv.–gr.xxx.) may be employed.

In winter, if the feet are apt to be cold, a hot bottle in the bed is very useful. In many of these cases the patient's statements as to his rest are quite untrustworthy, and more sleep is obtained than is acknowledged. In severe instances of insomnia the patient requires assurance and encouragement from his attendant, and to have his dread of dire consequences allayed.

If an opium-habit has been formed, it is at once to be broken off, and every care taken to prevent the acquisition of the drug. So far as my experience guides me, no risk, but, on the contrary, every benefit attends this line of practice.

The air of the bedroom should be fresh, and not too many articles of furniture allowed to remain in it. The bed should be placed in the centre of the room, not facing a window.

Attention to details is all important if success is to be gained, and such minutiæ are as much a part of clinical medicine as the exhibition of any drugs. They are too often disregarded.

Epilepsy.—Where this neurotic tendency prevails in the gouty, there is evidence sufficient to prove that it can be powerfully modified by recasting the dietary. Butcher's meat and nitrogenous materials are to be withdrawn as far as possible, and fish, fatty, vegetable, and farinaceous food must replace them. Meat may be sparingly taken on alternate days, or even less often.

In some cases a paroxysm of regular gout may be induced with

much benefit. As medicines, the bromides are of as much value as they are in any case of so-called central, or idiopathic, epilepsy, but smaller doses will be found effective when combined with alkalies, as potass salts or hypophosphite of sodium. From ten to twenty grains of potassium bicarbonate may be given, with as much bromide of potassium or ammonium, or ten grains of the sodium hypophosphite. Chalybeates are to be avoided. The action of the bowels must be attended to, and a very equable, though active, life is the best to enjoin, with due proportions of cerebral and muscular exercise.

Occasional mercurial aperients, at intervals of three weeks, are likely to be helpful in averting attacks. Duration of sleep should not exceed seven or eight hours, and none should be permitted after meals. Venereal excess is especially harmful. Stimulants must be taken in strict moderation, and may often be dispensed with. Over-study and undue excitement are to be avoided. In such cases marriage should be discountenanced, or at any rate postponed, till several years have elapsed without any epileptic manifestation.

It is important to ascertain the presence of lead-impregnation in cases of gout with epileptic manifestations, for the latter may possibly be due to saturnine influence rather than to gout, or they may depend upon the mixed cachexia induced by both taints.

Headache.—Headache of gouty origin demands treatment for the general state of which it is the symptom. Purgatives containing mercury are commonly useful, and alkalies or saline remedies. Colchicum is found to be of value, and was recommended by Sir Henry Holland for this purpose.[1] Exercise and aëration are always indicated, also regular exercise in respect of brain-work and nervous activity. Periodical purgatives and suitable diet generally avail, and chloride of ammonium may be taken at intervals.

Hemicrania.—Gouty megrim is one of the gravest manifestations of imperfect development of gout, and is a sore vexation to sufferers by reason of its prostrating and incapacitating character. The treatment relates to the paroxysms, and to the intervals between them. The attacks may begin early in life, but happily tend to become fewer with advancing years, and they may pass away altogether, or become less intolerable after the fourth decade.

Any obvious gouty symptoms must be treated on general prin-

[1] Medical Notes and Reflections, p. 266.

ciples. The conditions leading to attacks are usually recognized by sufferers, and must be avoided as far as possible. The determining causes vary infinitely in different individuals.

The paroxysm may be best treated by rest in a darkened room, in bed. Hot pediluvia are proper. Cold may be applied to the head by an ice-bag or spirituous lotion. A smart purgative is often advisable, and may be given in the form of a few grains of calomel, followed up by compound senna mixture, with colchicum. If the stomach is irritable, it is best to abstain from all food, and take iced soda-water only. Remarkably good effects follow the employment, in doses of ten or fifteen grains, of antipyrin, repeated in two hours' time. This is found by some sufferers to rob the disorder of its terrors. Bromide of caffeine is sometimes useful in doses of one to five grains. Tea is of service in some cases, and so is a short period of sleep. Undue sleepiness sometimes ushers in bad attacks on the morning of a day of suffering, and should be resisted. I have known a short journey into the country abridge an attack, and have also found an additional quantity of wine at dinner disperse the final twinges of agony. The more important part of treatment consists in care after the attack, with a view to avert fresh ones. Regular hours, equable life, freedom from worries, abstention from evening entertainments, especially from crowded assemblies, dinner-parties, hot rooms, and mental exhaustion, should be enjoined. Any undue fatigue is bad. Excessive tobacco-smoking, sight-seeing, visits to picture-galleries, are all to be deprecated. Overtaxed emotions with mental anxiety, or tension, often induce a paroxysm within a few hours' time. Periodical purgatives containing mercury are certainly of use, and a course of arsenic is one of the most potent remedies in the intervals of attacks. Alkalies with chloride of ammonium are indicated in many cases. Change of air and all means which raise the level of health are favourable; and all the powers of the mind and body should be regularly and equally exercised. *Ne quid nimis* is the motto for any sufferer from severe migraine, but each case is commonly idiosyncratic in respect of provocatives, determinants, and therapeutic agency.

Gouty Neuritis.—Gouty neuritis is one of the most painful and tedious manifestations of irregular gout. It is, happily, not very common. Ordinary local anodynes seldom avail much to mitigate the suffering. So much so is this the case, that it would appear to be the best practice to begin early with more thorough methods. I think most highly of blistering in the vicinity of an inflamed

nerve-trunk. Strips of cantharidine plaster should be placed parallel to the painful tract, and the resultant blisters kept open by Albespeyre's paper (No. 2) for some weeks. This is not so severe a measure as it would appear, and is more rapidly efficient than most other means. Mercurial ointment may be applied over the affected nerve.

If these plans are undesirable, chloral and camphor, or menthol and camphor, one of the former to two or three of the latter, may be smeared on the part. Iodine liniment may be painted in streaks beside the nerve. Cocaine ointment may be applied occasionally as an anodyne. Corrigan's thermic hammer applied along the course of the nerve is sometimes efficacious.

Internally, quinine with iodide of potassium is indicated; full doses, three or five grains of the former, being employed twice or thrice a day. In rebellious cases, arsenic or perchloride of mercury may be used. I do not recommend resort to subcutaneous use of morphia. This is but of temporary benefit, and is not likely to prove of lasting use in a trouble of this nature. With the best treatment, such cases are not likely to recover in a short time, and with ineffective measures the painful nerve may continue to cause torment for many weeks or months. Some relief may possibly be gained from time to time by the action of the voltaic electrical current, slowly interrupted, for five minutes at each operation; but if the pain is aggravated by this proceeding, it must not be repeated. As the swelling on the nerve-trunk subsides, the pain gradually passes away, but dysæsthesia may remain for some time in the parts supplied by the affected branch. Care is sometimes needed in making the diagnosis between gouty and peripheral alcoholic neuritis. Alcohol is often a common factor in both varieties of cases.

Gouty Neuralgia.—Neuralgia, as an expression of the gouty habit, demands treatment directed to any symptoms of that habit which may be present. The ordinary causes of neuralgia are to be sedulously avoided, exposure to cold winds and damp being particularly provocative. Any dyspeptic troubles, and constipation, are best treated by mercurial aperients and alkalies. Quinine with alkalies is a very useful combination, and chloride of ammonium is valuable in full doses. Locally, great relief, sometimes immediate, is gained by the voltaic electrical current, the negative pole being applied to the nape or spine, and the positive one to the course of the affected nerve. Only feeble currents may be used for any cranial nerve, not more than six or eight cells of a Leclanché battery being employed.

Menthol and camphor paste or veratrina ointment may be applied locally. In severe cases morphine may be required hypodermically. It is important to check each paroxysm as far as possible, and not to permit a neuralgic habit to supervene. Sometimes, a full dose of calomel and quinine at bedtime proves effectual to cut short a series of bad attacks.

The neuralgia following herpes zoster after middle life is occasionally very incoercible in the gouty, and calls for anodynes in addition to tonic measures.

Arsenic is of great value, and may be given with alkalies for long periods. Change of climate greatly aids other methods of treatment, and this is especially observed in the case of patients who live in damp and low-lying country-houses. A change to the drier conditions of town-life sometimes proves rapidly beneficial. A succession of flying blisters applied near, but not over, the affected nerve affords relief.

Sciatica.—Sciatica, or hip-gout, is sometimes due to gouty perineuritis of the great ischiatic nerve. In other cases it is a true neuralgia; while again, at other times it is a symptom of arthritis in the hip-joint. The latter is but rarely of uratic variety, and most frequently due to chronic rheumatic arthritis. Corrigan's thermic hammer and successions of blisters are very useful in the treatment of sciatica, and morphine may be applied in dressing the blistered surfaces. Hypodermic injection of morphine is also useful in rebellious cases. Internally, quinine and purgatives are proper. I have found acupunctures sometimes efficient.

Daily exercise in the open air is useful for post-herpetic neuralgia, pursued till the skin becomes moist. Care must be taken to avoid subsequent chill. All sources of depression and exhaustion must be shunned. The dietary should be plain but nutritious, and a fair proportion of fatty food is proper for most cases. Port wine may be taken with one meal, and, in most cases, no fear need be entertained of inducing gouty symptoms by this means.

The patient, and not the disease, is to be treated. In every case, the urine should be examined for glucose.

In summer, a high inland health-resort should be sought, Malvern, Ilkley, or Braemar being especially suitable localities, and a winter-season may be advantageously spent out of England, as, for example, at Pau.

Local Gouty Paralysis.—Local paralyses due to gouty neuritis or perineuritis may occasionally be met with. Facial palsy has

been observed in this connection, as in a case mentioned by Garrod, where the symptoms resolved at once on the supervention of regular gout. The musculo-spiral nerve may be thus affected, and of this form of paralysis I have met with several examples in gouty men, all of whom made good recovery after suitable treatment.

In the following case there were no tender points detectible in the affected arm, but the ulnar nerve appeared to be involved.

W. D., æt. forty-seven, came complaining of numbness and tingling, with marked weakness in the left hand. He was a window-cleaner. He had had several attacks of gout in the feet. He drank beer. No lead-impregnation. The fingers were cramped, and he could not close his hand. He had not slept on the arm, nor suffered any pressure on it. Strict diet was ordered, and bicarbonate of potassium with colchicum. The hand and arm were bathed with hot water, and cotton-wool was applied as a dressing. In four days there was improvement, and this was more marked in a week. The muscles of the affected arm reacted normally and as readily as those of the sound limb. Electro-sensibility was unimpaired. The ring and little fingers remained feeble, with kakæsthesia, for some time. No swelling occurred anywhere. Perfect recovery followed.

Cramp in Gout.—Cramp of the sural muscles is sometimes a very annoying symptom of a gouty state of body. It may precede a paroxysm in a joint, or may indicate a more than usually gouty condition. Antacids are of use in removing it. A very useful prescription, recommended by Dr. Munk, is subjoined.[1] A bandage firmly applied from the dorsum of the foot to the knee is sometimes efficacious in preventing recurrence of attacks.

Gouty Vertigo.—This in most cases is a result of gastric disorder (*vertigo à stomacho læso*), but may be purely of central origin, as an expression of the gouty habit. In the former case, treatment addressed to the prevailing form of dyspepsia is usually successful, and rapidly so. A nightly dose of compound rhubarb powder, and an alkaline and bitter mixture by day commonly avail to remove the symptom. If the digestion is slow, pepsine with strychnine and mineral acid, taken during the principal meals, is of use. In the case of "central" vertigo, an attack of articular gout sometimes removes the symptoms. Aperients containing mercury and colchicum are then fit remedies. To prevent discomfort from attacks of giddiness when walking, it is well to adopt the simple precaution of closing one eye. The eyes should be examined in each case. Sometimes, a degree of ophthalmoplegia

[1] ℞ Magnesii Carbonatis, Sulphuris Præcipitati Pulveris Cubebæ, aa. ʒi. M. intime, ut fiat Pulvis. Sit dosis coch. parvum ex lacte horâ somni.

externa may explain the vertigo, demanding full inquiry into the exact nature of the case.

Hysteria.—Hysteria, as a manifestation of inherited gout, can hardly be said to demand any special medication in respect of its ætiology. This is perhaps true for most examples, but there are some in which direct anti-gouty treatment is called for.

The action of the bowels should be regulated, and the circulation kept as active as possible. The dietary should be as nutritious as can be taken. These patients are apt to be very poor eaters, and very whimsical in their appetites. A measured quantity of wine with at least one meal is often called for. Exercise is imperative. Great care is necessary in repressing any craving for stimulants or narcotics. The type of such cases is commonly atonic, and no lowering measures or drugs can be borne. Patience, kindness, and firmness are necessary both on the part of the friends and the attendant. Every encouragement to effort should be made, and all morbid introspection and attention directed to symptoms should be as far as possible dissipated. Chalybeates are often badly borne, in which case chloride of ammonium with nux vomica, and cod liver oil may be usefully employed. Climatic change is of value, especially in winter and spring.

Respiratory System.

Bronchitis.—A tendency to bronchial catarrh, chronic bronchitis, and emphysema is especially noteworthy in gouty subjects. The gouty element in any such case should be duly noted, and treatment modified accordingly. Alkalies with iodides are of especial value, combined with ammonium salts. Stimulating expectorants, such as senega, serpentary, and nux vomica, are useful, and where there is bronchorrhœa, terebine in five-minim doses may be advantageously employed. Where there is spasm, belladonna or stramonium is of value, together with iodide of potassium. Climatic change is important when acute symptoms have passed off, and certain mineral waters may also be taken with benefit.

Chronic eczema or patches of psoriasis may alternate in activity with gouty bronchitis; and, sometimes, it is well not to treat the former too vigorously, unless they prove unduly vexatious.

Where much emphysema exists, there is especial risk of pneumonia in those of gouty habit, and it is apt to prove fatal. High arterial pressure may be met with in some cases.

Pneumonia.—Pneumonia may suddenly supervene, and is, sometimes, plainly a form of visceral gout. It demands a supporting line of treatment. Quinine with alkalies, or iodide of potassium, and alcohol, according to the state of pulse and degree of pyrexia, are the essential remedies. The pulmonary changes may rapidly alter. The condition of the urine should be examined. Colchicum may prove useful in conjunction with the remedies already mentioned. Jacket-poultices are advisable, as in ordinary cases. Embolic pneumonia demands special attention to the state of the circulatory system, and calls for stimulants and supporting treatment. These cases may prove tedious. Severe diarrhœa may occur in connexion with gouty pneumonia.

Asthma.—This is most often associated with bronchitis, but may occur in pure neurotic forms, or as a phase of retrocedent gout. In the latter case, it is proper to try and recall the gout to the part vacated. Indiscretions in diet may provoke bronchial spasm (peptic asthma), and the special peccant article comes, in time, to be discovered. Various inhalations prove of value, the fumes of a powder consisting of tobacco, stramonium, and nitre being often helpful.[1] Brown paper steeped in nitre and chlorate of potassium, dried, and impregnated with compound tincture of benzoin, is useful to burn, and the fumes should be inhaled freely. Two grains of caffeine in a cup of coffee will give relief in some cases. Belladonna, stramonium, and alkalies are best given in mixture, or a pill containing either the extract of belladonna or that of stramonium in doses of a fourth or third of a grain may be taken once or twice a day. Iodide of potassium and belladonna may be employed together, or with small doses of liquor arsenicalis. In severe and rebellious cases it may be necessary to employ morphine by the mouth or subcutaneously. Chloral hydrate and bromide of ammonium are available, and a few whiffs of chloroform or æther may be inhaled, at the hands only of the practitioner, the patient being forbidden to dose himself in this fashion. Nitrite of amyl, or isobutyl, capsules may be thus employed.

For the neurosal condition attaching to the arthritic diathesis arsenic is of supreme value, but must not be abused.

Mercurial aperients with colchicum are proper at intervals in these cases. Lobelia in the form of ætherial tincture is sometimes useful in doses of fifteen or twenty minims; and so, too, are small doses of ipecacuanha wine and spirit of nitrous æther.

[1] ℞ Pulveris Fol. Stramonii ℥ss., Pulveris Anisi, Potassii Nitratis, aa. ℥ij., Pulveris Tabaci gr.v. M. intime ut fiat Pulvis. Sig. A teaspoonful to be burnt in a saucer, and the fumes inhaled. (Brompton Hospital.)

Smoking of tobacco, or of Joy's, Grimault's, and datura tatula cigarettes may be employed during the paroxysms.

Attention must be paid to the condition of the digestive organs, and a dietary suitable for the gouty must be enforced. The presence of any textural degenerations and the degree of bronchial catarrh must be noted in any case.

The influence of climate is very marked, and no precise directions can be given without regard to the special features of the case. Moderately high and dry localities are, in general, preferable to wooded sites, unless in the region of pine trees, which flourish best on sandy and dry soils. The atmosphere of towns, being dry and smoky, suits some cases best, as is well known, while others only find comfort in the purest air, as that of the sea. Most gouty cases do well at Mont Dore or Arcachon, at Aix-les-Bains and Dax. Subalpine resorts anywhere on the Continent, or the climates of Bournemouth, Bagshot, Clifton, Malvern, Ilkley, or Braemar in this country, are available. Inland or mountain influence is commonly best suited to all arthritic cases, and in Britain it is difficult to get away from all marine impregnation. The variations of climate anywhere may be extreme within very circumscribed limits, since much depends on soil, drainage, vegetation, exposure, and shelter; and, hence, important differences may be met with in respect of a few feet of altitude, or a few furlongs of distance.

Circulatory System.

Disturbances of this system, due to gout, are revealed by such symptoms as inordinate vascular throbbings, irregular cardiac action, varieties of cardiac valvular disease, paroxysmal cardiac neuroses, and the disorders due to faulty nutrition of the heart's walls and the blood-vessels generally.

Inordinate Arterial Pulsations.—Inordinate pulsations of the thoracic and abdominal aorta are often associated with phases of gastric disturbance. They come and go, but may persist for long periods. Remedies and dietary suitable for any prominent dyspeptic symptoms are to be employed. For the special symptom of rapid and violent throbbing, aconite in doses of three or four minims of the tincture, repeated every two or four hours, is useful. This drug is not permissible, however, unless the patient is generally well-nourished and has fair digestive capacity, and it must be given only under supervision of the attendant. In other cases, bromides of potassium or sodium are often valuable

in doses of ten or fifteen grains thrice a day. Subsequently, the mineral acids with strychnia are available.

Neurotic Tachycardia.—For pure neurotic tachycardia, aconite, given with the above precautions, may be prescribed. Vascular throbbings in the neck, affecting the thyroideal distribution, may be treated with ergot or with digitalis, and these drugs may also be employed in other forms of undue pulsation. Arsenic is useful in long-continued courses, together with ordinary measures for promoting nervous tone and stability.

Insomnia.—Insomnia is sometimes a result of these conditions, and may be relieved by bromides, moderate alcoholic stimulation, especially in the elderly and cachectic, and by half-drachm doses of paraldehyde at bedtime. Monobromated camphor in doses of three or five grains, musk in two to five grain doses in pill, or tincture of sumbul in half-drachm doses, are all available, and may be had recourse to in turn. Climatic change, if suitably secured, is often of value in re-establishing the condition of neurasthenia, on which many of these untoward symptoms depend.

Irregular Cardiac Action.—Irregular cardiac action may, or may not, be a symptom indicating gravity. It sometimes persists through a long gouty life without serious significance, or it may occur occasionally, as a phase of visceral gout, in which case treatment for that state is demanded. If it be a retrocedent manifestation, endeavours must be made to restore gouty action in the part lately affected. The condition of textural nutrition, and that of the valves of the heart, is to be determined in any such case. Stimulation is usually necessary where failure exists, whether due to defective nerve-power or to parietal weakness. Defective compensation in the walls, with dilatation of cavities, calls for digitalis and strychnia, alone, or together, with æther, and a subsequent dosage with arsenic and strychnia, with or without some chalybeate, according to circumstances. Digitalis is to be avoided in cases of aortic valvular disease involving reflux, and replaced by belladonna; and the ordinary rules must guide the treatment of mitral reflux. In mitral constriction digitalis is often useful for a time, but the patient must be carefully watched while taking it. Tincture of strophanthus is of value in any case of cardiac parietal debility, and in doses of four or six minims has no tendency, so far as I know, to induce unwelcome symptoms. Consecutive changes on the right side of the heart, such as dilatation with tricuspid reflux, may be treated in the gouty, as in any other patient, on general principles, not omitting venæsection, if need be.

The prognosis is especially unfavourable in cases with progressive vascular degeneration and interstitial nephritis. Iodides in small doses with nux vomica are amongst the best general remedies for this state, and paraldehyde and cannabis indica are the safest hypnotics when such are required.

Pseudo-Angina Pectoris.—Pseudo-angina pectoris is usually associated with gastric disturbance and extreme flatulency. The symptoms may thus simulate gout of the stomach. They occur in persons younger than the subjects of true angina. The heart is found to be free from signs of organic disease, and there may be no signs of vascular degeneration. For immediate relief, stimulants are necessary. Brandy, sal volatile, or æther may be given, or a carminative draught containing bicarbonate of sodium, spirit of cajuput, compound tincture of lavender and peppermint water. A large linseed and mustard poultice should be applied to the epigastrium. A dose of compound senna mixture, with half a drachm of wine of colchicum, may be given on the subsidence of the urgent symptoms, and a mercurial purge may sometimes precede this. A hot pediluvium will afford relief.

True Angina Pectoris.—True angina pectoris with associated organic cardio-vascular changes demands different management. Capsules of amyl nitrite, or iso-butyl nitrite, should always be at hand for inhalation, and carried by day in the pocket of the sufferer. A tablet of nitro-glycerine (1.100 gr.) taken twice or thrice in the course of the day is often effectual in warding off paroxysms. Stimulants are necessary, and in protracted pangs, morphine used hypodermically must be used, but only by the medical attendant.[1] Veratrina ointment, or belladonna and chloroform liniment in equal proportions, may be applied to the præcordia. Compound spirit of æther in a dose of twenty minims may be given with as much of the liquor morphinæ acetatis in camphor water, regard being had to the state of the kidneys. The special cardiac and neurosal condition demands treatment—arsenic, strychnia, belladonna, and iron being of use for cases where parietal softening is surmised to exist. Inasmuch as paroxysms are apt to occur on the supervention of sleep, amyl nitrite capsules and an anodyne draught should be in readiness at the bedside. The patient should be kept quiet, and free from all forms of emotional disturbance.

Hæmorrhages.—Hæmorrhages occurring in the gouty demand little or no special treatment. They check themselves, and work,

[1] No nurse, or even "trained" nurse, should ever be permitted to practise any hypodermic form of treatment.

possibly, some good in many cases, certainly little, if any, mischief. Management, not treatment, is called for.

Phlebitis.—Phlebitis, as a gouty manifestation, requires careful treatment. The gouty nature of the ailment must be clearly recognized, as also the full gravity of the case. It may be very unimportant in many instances in the long-run, but may never be made light of while present. In any case, the patient should be kept on suitable regimen, and treated for at least two months till all signs of the disorder have subsided, and the clot is disposed of or rendered harmless.

The calf of the leg is the commonest site, but the upper extremities may be implicated; and the most serious examples are those involving the axillary vein, or some vein at the root of the neck. Recumbency is essential, and due warning must be given of the danger of sudden movements or flexion of the limbs. Erect posture, if assumed, must be arrived at with care, and if a lower limb is involved, it should be spared in such actions as getting into bed or dressing. The dangers are of embolism of the pulmonary artery, and syncope with apnœa, and they are apt to occur both early and late in the course of the disorder. There may be, in some of these cases, latent chronic nephritis, and a feeble condition of the heart.

The condition of the blood is that of hyperinosis, with diminished alkalinity. This is to be met by a diet sparing in nitrogenous elements, and by a free use of vegetable food and of diluents. Fish and farinaceous food may be given; and alcohol, best in the form of whisky or brandy, is generally of value to maintain a vigorous circulation and check the tendency to blood-stasis.

Vegetable food is preventive of hyperinosis. Some mineral water, as that of Vichy or Selters, is useful to the extent of a pint in the day.

If, as sometimes happens, there is much pain in the track of the inflamed vein, warm fomentations with belladonna or laudanum, or both, are desirable. No leeching, and no frictions, are to be practised. The limb should be placed at rest, slightly raised, and a cradle placed over it. Belladonna ointment may be spread thickly on lint and applied, or oleate of morphina smeared over the part, a tailed domet bandage covering all.

Internally, saline aperients are proper, as sulphate and carbonate of magnesium, with senna and colchicum, which may be given as an occasional morning dose; or four ounces of any bitter water, as Hunyadi Janos, Rubinat, or Püllna, may replace this at intervals. Quinine with potassium bicarbonate and ammonium

carbonate is serviceable at first, and quinine, mineral acids, and strychnia are to be employed later.

Some degree of œdema of the affected limb may remain, with enlargement and unnatural sensations, for varying periods after an attack; and if a considerable vein has been sealed, these may be abiding conditions, requiring an elastic stocking to be worn. Gentle friction and warm douchings may do much in due time to overcome these, but are not to be thought of till all signs of activity in the ailment have long passed away.

Recurrence is very apt to happen if a thorough recovery is not secured, and, hence, much disappointment may come from unheeded advice, or too pliable counsels on the part of the attendant. On trivial provocation, attacks of phlebitis are also prone to occur in those predisposed to this form of gouty trouble, and, hence, such pursuits and exercises as tend to determine them must be forsworn for the future. With lessened tendency to gout comes also lessened tendency to this variety of it.

In cases of embolism, if the clot be large, a fatal result within a few moments is almost certain. Smaller portions of a friable clot may reach the lungs and induce sudden symptoms, sometimes alarming enough, but commonly eventuating in pneumonia with bloody sputa, pyrexia, and, sometimes, septical symptoms, demanding quinine, ammonia, and stimulants.

Gouty Affections of the Tongue and Fauces.

Lingual Neuralgia.—Neuralgic pains in the tongue commonly pass off without treatment. Sore tongues, with psoriasis and thickening, are relieved by taking food not too hot, and by avoiding salt and tobacco-smoking. A saturated solution of chromic acid may be painted, at intervals of a week, over white patches occurring on the tongue. Balsam of Peru may be thus used more frequently. Boracic lotions are soothing, with or without chlorate of potassium. Regulated dietary, with (if any) a moderate allowance of stimulants, is to be prescribed.

Tonsillitis.—Tonsillitis is best treated at first by sucking ice, and a dose or two of two ounces of the mistura guaiaci. After twenty-four hours guaiacum is of no avail. Salicylate of sodium is often efficacious, and is to be given in doses of from fifteen to twenty grains every two hours at first, subsequently every four or six hours. Warm gargles of borax and camphor water are soothing, or solution of cocaine, ten to twenty per cent., may be painted over the fauces. A mercurial or saline aperient is neces-

sary, and quinine with tincture of cinchona may be given for some days as the trouble subsides. The throat may be rubbed with compound camphor liniment, and cotton-wool applied for some days. It may be necessary in some cases to give three or four ounces of port wine daily with meals. One tonsil is commonly more involved than the other. Suppuration is the exception in these cases, and ulcerative or herpetic forms are less frequent than in other varieties of tonsillar angina. The trouble may rapidly subside on the occurrence of gouty arthritis. After middle life, gouty angina faucium is less frequently met with.

Parotitis.—Gouty inflammation of the parotid gland may occur spontaneously, or by metastasis. It has not been known to precede orchitis, or to be in any relation to it. Treatment must be conducted on general principles, colchicum proving promptly useful. Belladonna liniment should be applied, cotton-wool laid over the part, and gout be recalled to any previously involved joint. Sometimes, no treatment is of avail, relief only coming with the supervention of arthritis. One gland may be attacked, then a joint, and after an interval the other gland may become inflamed.

Digestive System.

I have already discussed the treatment of the varieties of dyspepsia met with in the gouty, and, in particular, the special forms of visceral gout which affect the alimentary canal. It remains to be stated that many of the disorders of this system, including hepatic derangements, are most efficiently treated by hydro-therapeutic measures conducted on principles derived from experience at the various Spas and health-resorts. I shall refer to these more at length in the chapter on balneology and hydro-therapeutics. A course of hot-water-drinking may often be taken with advantage at home.[1]

Gouty Affections of the Liver.—In most gouty cases the disorders of the liver, with the exception of formation of biliary calculi, belong to the so-called functional class, and have as yet no definitely recognized morbid anatomy, because they are for the most part fleeting, and do not entail structural changes. In a minority of cases, where alcoholism has prevailed, ordinary cirrhotic

[1] Dr. Haig believes that dyspepsia may result from hepatic congestion due to retention of uric acid in the liver. There thus results a general diminution of absorption and of nutritive changes, with lessened formation of urea and uric acid, and a fall in acidity of the blood. As a result of the latter comes a rush of uric acid into the blood, with the supervention of headache or a gouty attack. When any drug disturbs digestion, there is always a fall in acidity, and a rise in excretion of uric acid.

changes may prove complications, demanding treatment on general principles. Most often, we have to deal with some degree of tumidity, and more or less hepatalgia. The portal venous system may be engorged, and piles may result. Gastric catarrh is a necessary concomitant. Constipation may result, and the stools be paler than natural, and knotty in character.

Attacks of this nature constitute a variety of visceral gout. The tendency for uric acid to be retained in the liver in persons of gouty habit must be borne in mind, and such retention cannot fail to be noxious in various ways. Headache, mental depression, irritability of temper, and lassitude, with loaded uratic urine, are but some of the symptomatic indications. Mercurial purgation and sodium salts, with colchicum, prove effectual in relieving these troubles, and alkalies with chloride of ammonium are also of value.

Biliary Calculi.—The same line of treatment is proper when biliary calculi form, and threaten to pass. Restricted diet is of great importance, and farinaceous food is sometimes to be sparingly used in these cases. Fish, thin broths, and green vegetables are rather indicated than more concentrated liquid nourishment. Many mineral waters are of great value. Exercise is imperatively necessary, and brisk walking or riding are the best forms of it.

Glycosuria and Gouty Diabetes.—Glycosuria is one of the most marked symptoms of hepatic gout. I have already discussed the causation and symptoms of this disorder at length.

It is before all things important to recognize the arthritic element in these cases. The treatment is in the first instance regulated by such recognition. As in other forms of the malady, it is fortunate if the glycosuria be early detected. The amount of urine daily passed, and the degree of saccharine impregnation should be ascertained, as also the influence of digestion on the amount of sugar. The patient's weight should be taken weekly or monthly. At first, it will certainly be proper to employ restricted diet, and to watch its effects carefully. The glycosuria may, or may not, pass off completely, and if it disappear, the diet may be made more natural by degrees. Digestion must be kept at the highest standard. It will generally be found that the patient is conscious of better digestion when the special diet is not too strict, or too long persisted in. If there is a strong gouty tendency, fish and fowl, and avoidance of red meats may be beneficial; and whatever is bad for gout will in any case be bad for the glycosuria. It is usually found in the earlier stages that the sugar is easily removed by dietetic measures alone.

The medicinal treatment necessarily varies with each individual. Alkalies occupy the first place, both for regulating the digestion and dispersing the glucose in the blood. Effervescing citrate of sodium, citrates of ammonium and potassium may be used, and cinchona and nux vomica may be given with them. It is a good plan to give such a course for a week or ten days in each month, as was advised by Trousseau. Arsenic is another drug of decided value as a nutrient and nervine tonic, and is best given in from five to ten minim doses of Fowler's solution twice daily after food.

All possible aid must be secured from the best hygienic measures. The action of the muscles must be enforced by regular open-air exercise, and that of the skin be well-promoted. An open-air life is of the highest importance. Physical exertion appears to diminish glycosuria. In summer, recourse should be had to some high inland health resort or Spa, and amongst these Carlsbad, Kissingen, Vichy, Contrexéville, Plombières, and Neuenahr are in repute. Cases of "diabetes" are annually reported to be "cured" at each of these, and at other watering-places. M. Debout d'Estrées has observed at Contrexéville that the elimination of uric acid is simultaneous with the disappearance of glucose during treatment there.[1]

Without doubt, these cures sometimes occur, and it is in cases of this class, and in other mild forms of the disorder, that they are wrought. The out-door life and the regular habits, with freedom from cares and vexations, do much to aid the hydrotherapy. The only stimulants permissible are good Bordeaux wine, taken with water, or old whisky well-diluted.

In the chronic form, the diet must be relaxed in respect of amylaceous matters, lest the patient waste, grow dyspeptic, discontented, and become cachectic. Well-toasted white bread, "pulled" bread, brown bread, rice, macaroni, onions, and beans may be allowed in moderate quantity, and, at intervals, half a potato. Cream and fatty food are of much value, and milk need not be stinted. Asparagus should be avoided. Mischief due to errors of diet will soon betray itself in wasting, diuresis, languor, thirst, and by examination of the urine, and such symptoms call for stricter diet, at all events for a time.

All sources of irritation to the liver are to be carefully guarded against. If purgation is called for, Homburg salts, castor oil, or blue pill and colocynth mass, are amongst the best agents.

Dr. Schmitz of Neuenahr remarks, that "it is exactly in this form of diabetes that alkaline waters and salicylate of sodium are

[1] Brit. Med. Jour., February 23, 1884, p. 587.

found to be so beneficial, because both are known to be efficient remedies against the root of evil, viz., the gout." He has often observed an immediate and material improvement, and frequently a complete disappearance of diabetes after an acute attack of gout; also, that diabetes reappeared whenever usually recurring attacks of gout failed to come out.

He emphatically condemns strictly anti-diabetic diet in these cases, inasmuch as it is too nitrogenous, and tends to maintain the gouty state; and he urges that the measure of assimilative capacity for farinaceous food should be ascertained carefully in each case, white and brown bread being allowed, as also rice, macaroni, dried peas, lentils, and beans, and a fair quantity of milk; cane-sugar and dextrine being rigidly excluded in all cases. Saccharin may be employed as a sweetening agent, if desired.

My own experience is fully in accordance with that of Dr. Schmitz.

As a rule, cases of this class respond very promptly to restricted diet, and in a few days all traces of glucose may disappear from the urine. A corresponding improvement takes place in the general health and comfort of the patient. Weight is regained, and the sense of malaise passes off. Ordinary diet may then be gradually resumed. No hard and fast line can be laid down as to restricted diet for these cases as a class. Each one must be separately studied.

In all cases it is well to regard the patient as an invalid, and, without causing undue anxiety, it is important to let him regard himself as a valetudinarian. There will thus be a better chance for implicit obedience as to diet and regimen, and less chance of exposure to overwork, worry, or bad weather. It is necessary to lay stress upon this point, because many of these patients feel, at times, in excellent health, and look remarkably robust. They thus deceive themselves and their friends. No case may be lightly regarded. The chances of textural damage to the kidneys and cardio-vascular system must not be lost sight of in any chronic case. The glycosuria becomes of less importance as such graver indications supervene.

In cases where there are plainly-marked arthritic concomitants, it has long been observed as a good sign that uratic deposits occur. Elliotson mentioned this, also Prout. More recently, Bence Jones, Garrod, Pavy, and Beale have affirmed the same. Another promising indication is a moderate amount of urine, the specific gravity of which does not exceed 1.035. Very much depends upon early recognition of the glycosuria. Many cases

have unfortunately made much progress before sugar is detected, its presence not being suspected, because so many of the common symptoms of ordinary diabetes are absent. Sir William Roberts is inclined, from his experience, to give a rather gloomy prognosis, regarding the health as broken, as he found death occur in from two to four years from cerebral disease or pulmonary complications.

On the other hand, cases have been known to go on for ten, twelve, and even twenty-three years. With such a disparity before us, it is plain that no definite opinion can be given in a general way. Each case must be regarded by itself, and the effects of the disorder be studied upon the individual. The significance of the glycosuria thus varies infinitely in different cases. So much so is this the case, that, as M. Lasègue declares, one must study diabetes indefatigably for fifteen or twenty years before one can know much about it: "C'est une maladie à l'usage exclusif des vieux praticiens."

Many of the subjects of this variety of diabetes are robust and of good constitution, and the disorder must cease to exist in a latent, intermittent, or mild form before any marked derangement of general health is declared. Even then, much may be done to restore the wasted textures and the accompanying loss of energy, and happily, in many instances, with advancing years the glycosuria loses its importance as a symptom.

Schmitz observes that gouty diabetes has the best prognosis of all the forms of the disorder.

It is well if restricted diet speedily removes the glycosuria, and long intervals are passed without a return of it.

From time to time sugar appears in the urine, and a recrudescence takes place, and this occurrence would seem, sometimes, to indicate that attacks of glycosuria replace those of more obvious gout, to which the patients may have previously been subject. The same causes which are effective to bring out gout will here elicit glycosuria in its stead.

In some of these cases urine becomes of low specific gravity, and albumen appears, the quantity of urine continuing large, not, as Sir William Gull points out,[1] because there is much sugar to be discharged, but as a result of damage to the kidneys, which, together with the blood-vessels, become fibrotic, as part of the general gouty cachexia. Hence, glucose may be found in these cases in urine of as low specific gravity as 1.006, but the patient's condition is to be estimated in this stage rather by the renal and

[1] Private communication.

vascular changes than by the degree of glycosuria, which may be unimportant.

Intelligent patients, on learning that they present symptoms of diabetes, are naturally apt to be greatly perturbed and depressed. This mental condition is a most disastrous one. It is of especial importance to hold out as encouraging a prognosis as possible, while the necessary dietetic directions are vigorously enforced, and every effort is made to improve the general health.

Cases.

I shall not do more than record the leading features of some cases which have come within my own observation, and which sufficiently illustrate the character of this variety of diabetes.

CASE 1.—A robust and rather corpulent man was found to have glycosuria when about forty years of age. He came of distinctly gouty family. Had suffered from gout for some years at intervals, and had been actively treated for it. Restricted diet did not avail to remove sugar entirely from the urine. No loss of flesh, but sense of muscular weakness, and fatigue easily induced. Attacks of gout continued, and seemed to be kept in check by several Turkish baths taken in each month. Subsequently the sugar disappeared from the urine, and was at one time apparently replaced by free uric acid. This patient has remained for about seventeen years in good health, and leads an active life. A brother suffers from diabetes in a severe form, and for years has passed urine of specific gravity varying from 1.060 and upwards; he takes an ordinary mixed diet.

CASE 2.—A lady, aged about forty-four years, inheriting gout from one and perhaps from both parents, after exposure to cold and damp while travelling, presented all the symptoms of diabetes. She was rosy and robust. At the age of thirty she became unduly stout, having been previously spare and slim. There had been decidedly gouty pains in the feet and hands, and an irritable lichenous eruption sometimes appeared on the arms. Was a rather large eater, and partook freely of potatoes. Restricted diet caused the glycosuria to disappear in a short time. Occasional gouty attacks in minor degree occurred. Recrudescence of glycosuria three years subsequently, followed by improvement on use of dietetic precautions. Gradual loss of weight to the amount of eighteen or twenty pounds during this period. Muscular power enfeebled, and fatigue sometimes readily induced. Occasional deposits of lithates in the urine. Improvement always secured by open-air life, with exercise, in the country. Diuresis not copious, averaging thirty to forty ounces daily; no thirst, no undue appetite. Karlsbad salt, citrate of sodium, nux vomica, and Fowler's solution, at intervals, proved beneficial. Absolutely restricted diet badly borne, causing disgust and anorexia. Digestion always improved by addition of some amylaceous food. Sugar not entirely withheld. Gradual progress of mental irritability observed. Case now of twelve years' duration, glycosuria continues, the sugar varying from three to seven and eight per cent. Occasionally the *U. sanguinis* contains most sugar, and sometimes the *U. cibi.*

CASE 3.—C. B., æt. forty, mother of four children, came to the Hospital suffering from glycosuria. The urine was acid, of specific gravity 1.040, highly charged

with glucose, and free from albumen. Diabetic symptoms for four months. Her father lived to be seventy-three, and was subject to "rheumatism." A brother, aged forty-one, suffers much from gout, and lost a daughter, aged six and a half years, from diabetes.

The following example of gouty glycosuria is worthy of note, because the history of it is given by the patient himself, a country surgeon. This account was sent to me in 1883:—

CASE 4.—X. Y., æt. thirty-six, height 5ft. 9in., weight 13st. 12lbs., suffered in early years from chronic bronchial catarrh, which quite left at sixteen. About ten years since had several joints affected with "rheumatic gout."

Mother, a great invalid, died at thirty-seven, of bronchitis and rheumatic arthritis (? gouty bronchitis, D. D.).

Father died suddenly at forty-seven; was an accomplished man, and the best operator in this part of the country; would give way to fits of alcoholic intemperance, lasting some weeks, and then again would strictly abstain for months. Had occasional attacks of decided podagra.

Brother, also in practice, at times gouty.

"I first noticed diabetic symptoms in winter or early spring of 1880—thirst, frequent desire to urinate, and amount of urine passed first attracted attention—sp. gr. at that time 1.042; quantity passed in twenty-four hours about 120 ounces; sugar-reaction well-marked with Moore's and Trommer's tests. At once commenced strict abstention from starch and sugar, with the result that in less than a week reaction of glucose was just noticeable, sp. gr. 1.028, quantity passed sixty-eight ounces. Since then I have been rather careless. I live well but temperately, play cricket a good deal in summer, enjoy life generally, and have two young children. At the present time sugar-reaction is well-marked, sp. gr. 1.036, quantity ninety ounces. Am not dieting at all. I forgot to mention that I was laid up last winter with passage of an uric acid renal calculus."

Six years later he wrote:—

"I feel very well, occasionally tired from overwork, but have plenty of energy; a little thirsty, but appetite normal. Sp. gr. of urine 1.036, sugar-reaction well-marked with the usual tests, and acid; no renal calculi since I left Lincolnshire. I have a little gout occasionally in my toe, tongue clean, bowels very regular; desire not perhaps quite so frequent, but vigour unimpaired; sleep well at night, and, as a rule, do not get out of bed to pass water unless I have supped rather heavier than usual. I was weighed about a week since, and was then, under the same clothing, as heavy as I was six months ago; in fact, I have not varied for five years—12st. 2lb.

"I take plenty of exercise, doing all my work on foot, walking from two to eight miles a day, with an occasional longer stretch. I am now aged forty-two, live temperately but well, and have not dieted myself for the last six years beyond limiting my quantity of potatoes, sweet puddings, and wine. Spirits I seldom touch, but have a pint and half of ale in the day.

"I may as well mention, in fact, I think it important, or at least interesting, that my brother, who is five years my senior, began to develop glycosuria about four years since to about the same extent as myself. He has a large country practice, hunts two days a week, and although he drank to excess at one time, has for the last six years been a strict teetotaller. He also has had occasional attacks of gout."

CASE 5.—Mr. W. B., æt. fifty-two, brought to me by Mr. Earle of Brentwood in 1880. A stout, ruddy man, suffering from severe eczema almost all over his body, with much itching. Occipital headache and drowsiness complained of. The

action of the heart had been irregular of late. Habits alcoholic. Is very thirsty. The urine was of specific gravity 1.020, and contained a little glucose. Has had no regular gout, but his father was gouty, and was alleged to have died of the disease. The eczema proved obstinate for several months. Mr. Earle subsequently informed me that this patient often passed considerable quantities of sugar, but presented no ordinary symptoms of diabetes. He was then in fair health.

Case 6.—A lady, æt. sixty-six, seen in June 1882 at Milbrook with Mr. Dayman. Arthritic diathesis well-marked. Abdomen obese. Not married till after fifty. No family. Her mother was the twenty-eighth child. No distinct gouty history in the family (maternal grandfather possibly gouty). At the age of thirty-five had severe iritis, and took much mercury. The iritis was considered to be rheumatic. For the last five or six years has suffered for the most part of the year, but chiefly in winter, from pain in the lower dorsal region. At first, the left side was affected, then both sides. There is extreme tenderness over the last three ribs on each side, increased by pressure, full inspiration, or any forced movement. Can only walk very little with sticks. No crackling on flexing the joints, but sometimes snapping is felt on movement. Three months previously she felt unusually weak and ill, and was thirsty, and the urine was discovered to contain a good deal of glucose, and to be of specific gravity 1.040. Rigid diet soon caused removal of the sugar and improvement of general health. Some swelling and pain occurred in the right knee-joint before the glycosuria was detected. The urine fell in specific gravity to 1.025. The dorsal pain became worse. No signs of organic disease of the spinal chord or membranes, nor of spondylitis or aortic aneurysm. The glycosuria returned with slightly relaxed diet; the urine contained a moderate quantity of sugar; specific gravity 1.022. The *urina sanguinis* contained as much as the *urina cibi*. On some days no sugar was passed, and none would appear for weeks. Deposits of lithates not observed. In September the pain almost passed away, and walking became possible. Mr. Dayman reported (December) that this improvement had continued. "The urine, examined every few weeks, shows traces of sugar. All fat and plumpness are gone. She has much muscular power, can walk any reasonable distance, and expresses herself as 'very well.' The diet is partially restricted, and cod liver oil is now taken. She has lost no more bulk than the mere absence of fat would account for, and this loss has gone on *pari passu* with the decrease in the glycosuria, which, I take it, is a point in the patient's favour, being the reverse of what happens in phthisical diabetes." She died one year subsequently.

The following cases illustrate grave forms of diabetes in the sons of gouty men:—

Case 1.—J. M., a painter, æt. thirty-six, admitted to Mark Ward, November 1881. Has suffered from diabetes for twelve months, and been passing about twenty pints of urine daily during that time. He has had colic, but has no blue line on the gums. He has xerodermia, and a brother has also this affection. His father is gouty. He himself has had no gouty or rheumatic troubles. The urine is of sp. gr. 1.034, acid, and contains abundance of glucose. The abdomen has been enlarging for three months, and is now tense. The liver does not appear to be tumid. Has not been submitted to any anti-diabetic treatment hitherto.

Case 2.—W. K., æt. twenty-one, a waiter, admitted to Mark Ward, March 1882. Pallid, poorly nourished. Always temperate. Subject to fainting attacks with giddiness—but no loss of consciousness—about once a month for greater part of his life. He had been in New York for the last year, but felt weak and unequal

to his work there. Increase of appetite and thirst observed for about a month, with increased flow of urine. Has wasted much. Has been subject to boils for some years. Was found to pass eighteen pints of urine of specific gravity 1.035 while taking extra unrestricted diet, and this became reduced to eight pints. On taking regulated diet, he passed an average of twelve ounces of glucose daily. No physical signs of disease in chest or abdomen. His parents were both strongly arthritic. The father, aged sixty-eight, had twice had gout in his feet and eczema of the right arm. He was a hotel-waiter. The mother, aged seventy, had suffered from rheumatoid arthritis for fifteen years. (I examined them both.)

This patient chafed under anti-diabetic diet, and made his escape from the Hospital.

Dr. Mahomed[1] recorded the case of a man, æt. forty-two, who had had gout in both great toes two years previously, and who presented all the signs of granular kidneys with cardio-vascular degenerative changes. The father died at eighty years of age, "asthmatic;" the mother was living, æt. seventy, with dropsical legs, and two brothers had died of diabetes.

A case of acute diabetes came under my observation in a man æt. twenty-seven, whose father was gouty, and presented tophi in the ears. The paternal uncle was also gouty. There was diabetes in one of the cases of hæmorrhagic retinitis, reported by Mr. Hutchinson in his communication to the Clinical Society,[2] in a man æt. sixty-seven, although it is mentioned that there was no history of gout. As many of these cases occur in connection with gouty habit of body, I think it not unlikely, having regard to the age of the patient, that this was an instance of gouty diabetes.

The following case is of particular interest, illustrating the co-existence of acute gout with persistent glycosuria. This is, in my experience, a rare event.

H. F., a commercial traveller, æt. fifty-four, came under my care in St. Bartholomew's Hospital on November 10, 1888. He looked ten years older than his age, was a florid, white-haired man, of large frame. His father lived to seventy, his mother to sixty-five. His maternal grandmother was "rheumatic," but lived to eighty. A brother and sister died of phthisis, one brother died of heart-disease, and another poisoned himself with morphine.

Fifteen years ago he began to be diabetic. Ten years ago had what he called "rheumatism." Eight years ago had boils. Two weeks ago had pains in right hip, thigh, and swollen right knee and foot. Six days ago right elbow and wrist swelled and were very painful. On admission, the right wrist and hand were in a state of gouty inflammation. His tongue was red and "beefy;" breath "diabetic." Gums retracted; teeth large and strong. Pulse 156, of good volume and tension. Temperature rose on the 11th November to 100.4°, and slight febrile

[1] Guy's Hospital Reports, 1881, p. 373.

[2] Trans., vol. xi. p. 134, 1878.

movement continued till the 18th, the rise occurring at night. Afterwards, subnormal temperatures.

Signs of pulmonary emphysema. Slight cough. Heart a good deal overlapped. Epigastric pulsation. No murmurs. Sweated freely. Liver and spleen impalpable. Urine sp. gr. 1.034, glucose 15 per cent., albumen a trace, average quantity 119 ounces for first four days, and 78 ounces for next seven days. Acetone and diethylacetic acid reactions. He had lost two stones in weight in the last two years. He confessed to drinking about three pints of beer daily, but no spirit. He was put on a partially restricted diet; colchicum and citrate of potassium were given, and belladonna lotion applied to the gouty wrist. In a few days, there was marked relief to the pains, and the appetite, which was not lost, increased. His diet was improved, and bark, nux vomica, and citrate of sodium were given by day, and colchicum and Dover's powder in pill at night.

On the 23rd November he was out in the Square, and his pains began again in the wrist. He was now treated with sodium salicylate in gr.xv. doses four times a day. Dr. Haig made some analyses of the urine under the influence of this drug, and found a large excretion of uric acid by night, with a small out-put of glucose, and a large excretion of glucose and small one of uric acid by day. The excretion of urea fell as that of uric acid increased. The patient was, however, very unruly and odd in his manner, and would not lend himself to investigations of an exact nature. The effect of the salicylate was marked in causing free excretion of uric acid.

Patients suffering in this manner cannot, and must not, be treated as for ordinary diabetes. They crave for theoretically contra-indicated food, and languish if it be denied, and are worse if they submit to restricted diet. Like other diabetics, they will often break through any enforced rules, and deceive the physician. This man would doubtless have been much benefited by continued treatment with salicylate of sodium and a carefully arranged diet, but he preferred to go out of the Hospital.

Salicylate of sodium may be given in cases of gouty glycosuria in doses of fifteen grains thrice daily, the urine being examined quantitatively for glucose. If no marked benefit ensues within a few days of this treatment, it is not likely to prove useful in any case. Aperients containing mercury, given at intervals, are certainly useful. Opium and codeia are unsuitable in all but the worst and aggravated cases. The amount of sugar in the urine has been found to be increased after taking sodium sulphate.

The symptoms of most evil import in these cases are those indicating cardiac failure, general loss of nerve-power and tone, loss of weight, due to disappearance both of muscle and fat, and impairment of appetite. There are to be observed distinct periods in which the disorder is aggravated, all its symptoms being prominent, and others in which improvement occurs, weight is recovered, and the general health in a measure restored. Albuminuria is always a grave symptom. Acetonuria and loss of

knee-jerks may long precede the onset of more serious symptoms.[1] Coma may be the last event, and is not so infrequent in this class of cases after the age of thirty-five as the statistics of Dreschfeld would lead one to believe.[2] Cataract is not observed with any frequency in this class of diabetics, and the same may certainly be affirmed of pulmonary phthisis.

It is worthy of note that the Hebrew race appears to be prone to glycosuria, especially amongst its wealthy classes.

Many of the adipose patients lose a good deal of their fat, but it is not desirable to pursue any dietary or method that is rapidly weight-reducing. No sudden or violent measures can be sanctioned.

The patient's weight should be taken at regular intervals and duly recorded. So long as weight is retained, there is little likelihood of grave symptoms arising.

Genito-Urinary System.

Gouty Interstitial Nephritis.—The gouty affections involving the urinary tract are numerous. The condition of the kidneys in respect of cirrhosing lesion has been already dwelt on at some length, and stress laid on the importance of duly recognizing its presence both in early and advanced stages. All forms of treatment for the gouty must be regulated with reference to the existing renal condition, and, hence, care must be taken in prescribing both dietetic and medicinal measures, especially as regards nitrogenous food and the employment of drugs, such as opium, colchicum, or sodium salicylate. The influence of climate is very marked in these cases, and renal fibrosis may be materially checked in its course by combined attention to diet, and recourse to warmer surroundings in winter-time. Renal adequacy is to be gauged by observation of the amount and density of the urine passed, especially where there is little or no albuminuria. The percentage of urea should be estimated at intervals, and persistent out-put of less than two per cent. should excite serious attention in any case. The diet should consist mainly of fish, vegetable and farinaceous food. Milk-diet is sometimes very serviceable for periodic, or, when it can be borne, for continuous employment. Butcher's meat and alcohol are to be avoided as a rule, or very sparingly taken. The condition of the patient rather than the nature of his ailment demands attention in every case. Arsenic, strychnine,

[1] S. West, Proc. Roy. Med. Chir. Soc., November 1888.

[2] Bradshawe Lect. Roy. Coll. of Physicians, 1886.

and the mineral acids are often valuable, especially when iron is contra-indicated or ill borne. Frequency of micturition at night in elderly persons with granular kidneys may be treated by bromides or monobromated camphor, and the amount of fluid taken in the later part of the day should be restricted.

Paraldehyde in half-drachm doses is serviceable in cases of dilated and labouring heart in these cases. The headaches so commonly experienced are susceptible of relief by nitro-glycerine tablets (gr. 1/100) taken three or four times daily. Uræmic convulsive states are best treated with five-grain doses of hydrate of chloral every two hours, beginning perhaps with a larger dose. Sheltered winter climates should be sought, and Algiers is one of the most favourable of these.

Renal Calculi.—The main lines of treatment relate to free dilution of the blood and the consequent diuretic action thereby induced. Alkaline medication is important, and is best employed by prolonged use of these agents in diluted form. The most significant indications of renal calculi are attacks of ureteric spasm; but in many cases, stones may be lodged in the kidneys which show no tendency to pass away, and declare their presence by localized pain and hæmaturia, more or less grave and continuous. Little benefit is derivable from drugs in such cases. Rest is very important. A dietary suited to the patient's general and dyscrasic condition must be enforced, and diluents must be freely exhibited. Barley-water or linseed infusion and skimmed milk may be given, but it is best to employ distilled water to the extent of three or four pints in the day if recourse cannot be had to some appropriate Spa. Citrate of potassium is of value, and citrate of lithium taken freely diluted. The bowels should be regulated by Friedrichshal or Püllna water. The best results are, however, to be gained from a course of treatment by water-drinking at Buxton, Bath, or Malvern in this country, or at Contrexéville or Vittel, near the Vosges Mountains. Large quantities of these waters are commonly necessary to ensure solution and dispersion of the calculi. It is noteworthy that the waters of the two latter stations are rich in lime salts, which would theoretically appear to be contra-indicated in calculous disorders, but the results of their employment are unquestionably satisfactory, calculi and masses of uratic concretions being expelled, with cessation of the exhausting hæmaturia, and complete relief to the patients. Baths and douches to the loins also aid the expulsion of calculi and gravel. These waters are portable, but it is difficult, if not practically impossible, to conduct the treatment satisfactorily away from the

Spas. In the case of large calculi, the patient must submit to several courses of treatment in the above fashion.

Chronic cystitis, either gouty or calculous, is capable of great amelioration and cure at Contrexéville and Vittel.

Renal Colic.—Renal and ureteric spasm due to passage of calculi demands ordinary treatment—opium, belladonna, henbane, or chloral, or morphine, subcutaneously, being employed, together with hot stupes and hot hip-baths.

Vesical Calculi.—Vesical calculi, if not removed by diluents, demand crushing or cutting operative procedures. Tendency to calculous formation may be averted by early recourse to Carlsbad water-treatment, or by the steady use of any of the bitter saline waters taken each morning.

Balanitis.—Balanitis is best treated by warm fomentations, followed by the application of equal parts of starch and boric acid, a piece of dry lint being inserted under the prepuce. Boroglyceride lotion is useful.

Vulvar Pruritus.—Vulvar pruritus is often very intractable. It is most often associated with, if not directly dependent on, glycosuria. Sponging with hot water, or carbolic acid lotion, one to forty, is sometimes efficacious. Boroglyceride (pure) or boric acid ointment may be used after hot fomentation. Calomel ointment, two drachms to the ounce, is of good service, followed by dusting of the parts with a powder composed of one drachm of camphor and four each of oxyde of zinc and starch. Goulard extract, one drachm, and milk, two ounces (lactate of lead), or cocaine ointment may prove useful. The applications may have to be changed before relief is afforded. Compound tincture of benzoin is efficacious as a local application.

The general condition of which the pruritus is the expression must be met by appropriate treatment.

Affections of the Penis.—I have already alluded to persistent priapism as a symptom in certain cases of gouty habit. It may affect elderly men, and prove annoying at night. Alkaline treatment and bromide of potassium, with carefully regulated diet, are generally efficacious to remove this.

Fibrous thickening of the sheath of the corpus cavernosum may occur, producing irregularities on erection, firm masses being felt in the septum or on other parts of the penis. Chordee may occur in consequence. Paget has described a general gouty inflammation of the whole organ, causing enlargement, but not the hardness, of erection.

Thrombosis in Penis.—Thrombosis may occur spontaneously

in the corpus cavernosum after the fashion of gouty phlebitis. One or more hard nodules of the size of a pea or bean may be felt, and they may prove painless even on pressure, and remain for a long period, slowly becoming smaller. Little is indicated in the way of local treatment. The unguentum iodi may be rubbed in, and any overt gouty indications be treated on general principles.

Herpes Præputialis.—In the gouty there is more than ordinary tendency for herpes to occur on the prepuce. Some forms of balanitis are herpetic. There is tendency to recurrence, and pure coitus may be an excitant. Zinc ointment, with carbolic acid in the proportion of half a drachm to the ounce, or boric acid ointment, are suitable applications.

Orchitis.—Orchitis is sometimes distinctly a manifestation of acute gout. It may be a phase of retrocedent gout, or suddenly set in after minor gouty premonitions in other parts.

Treatment consists in supporting the testicle by adjusted padding. Lead and opium lotion should be applied hot at frequent intervals, or equal parts of glycerine and belladonna extract may be freely painted over the scrotum. A mercurial purgative should be administered, and the bowels kept freely open. Colchicum with iodide and bicarbonate of potassium is very efficacious. Rest is imperative, and a light dietary. The enlargement may remain for many weeks, with some induration of the epididymis. There may be some fluid effused at first into the tunica vaginalis. Atrophy of the organ is not apt to supervene.

Vesical Hæmorrhage.—Vesical hæmorrhage has been already discussed. It demands little or no treatment. Clots may, however, have to be withdrawn from the bladder by means of a large-eyed catheter, and by suction.

Cystitis.—Cystitis is sometimes very severe, and calls for treatment by restricted "slop" diet, diluents, especially milk, and hot belladonna fomentations or poultices to the hypogastrium. Metastasis of eczema from the skin may occur, and an enanthematous cystitis supervene. Citrate of potassium, or liquor potassæ, with henbane, given in infusion of buchu or decoction of pareira brava, are useful, and opium or belladonna may be employed, if required. Decoctions of barley, linseed, or triticum repens, with liquorice, are useful as diet-drinks. Rest in a warm bed is essential, and hot hip-baths at the bedside may be used twice daily. Colchicum may be given at night with Dover's powder in pill, and a mercurial purge, followed by a saline cathartic, will prove of value at intervals.

Urethritis—Prostatic Gout.—Gouty urethritis, which often simu-

lates gonorrhœa, and prostatic gout, demand similar treatment—rest, light diet, and sandal-wood oil proving useful. Hot hip-baths are very soothing.

If the attacks in the bladder, prostate, or urethra are due to retrocedency from a gouty joint, efforts may be made to reinduce the arthritis. In severe and sthenic cases, half a dozen leeches may be applied to the perineum, followed by fomentation. Tartar emetic in small doses, with opium, may prove of service in these cases, and aperients are called for. Retention of urine must be relieved, if necessary, by a soft rubber catheter.

Ovaritis.—Ovaritis calls for rest in bed and hot poultices or stupes with compound camphor or belladonna liniment. Colchicum and alkalies with moderately strong aperients are useful.

Recumbency is imperative till all pain is past, and it is well to continue this till the next catamenial flow has subsided. A fly-blister over the region of the affected ovary is sometimes useful, and may have to be repeated. Belladonna and other anodyne pessaries are also serviceable.

Congestive Dysmenorrhœa—Ovarian and Pelvic Neuralgia.—The gouty habit predisposes to menstrual suffering. There may be, with hysterical tendency, ovarian neuralgia or uterine pain alternating with other neuralgic manifestations.

Exposure to cold and damp is to be carefully avoided in these cases. Saline aperients are of use. Pain may be relieved by cannabis indica, sumbul, and bromide of potassium. Antipyrin is also very serviceable in ten or fifteen grain doses. In atonic cases, guaiacum thrice a day is sometimes useful, partly perhaps by its laxative action.[1] Ten grains of powdered guaiacum with as much carbonate of magnesium may be given each morning. This prescription is especially useful where shreds of membrane or clots are discharged. Warm hip-baths should be taken nightly. The employment of alcoholic stimulants and sedatives by the patient herself must be sedulously guarded against. Henbane, with musk or valerian, may be given with quinine for neuralgia in the pelvic region, and arsenic, with alkalies, is also advisable in some cases.

Much benefit is to be secured for patients suffering from these ailments by recourse to certain spas. In this country, Buxton, Bath, and Harrogate are available; on the Continent of Europe, Vichy, Schlangenbad, Ems, Wiesbaden, Baden-Baden, Marienbad, and Carlsbad are amongst the most suitable either for baths, or for combined use of these with water-drinking.

[1] Recommended by the late Dr. Rigby of St. Bartholomew's Hospital.

Gouty Affections of the Eye.

Arthritic Ophthalmia.—For special methods of treatment of arthritic ophthalmitis, iritis, irido-cyclitis, retinal hæmorrhages, episcleritis, and glaucoma, I must refer the reader to works on ophthalmic surgery. I would only mention in this place that the gouty habit of body demands at all times appropriate treatment whenever any local manifestations of it appear. Mercury, quinine, aconite, colchicum, iodide of potassium, and alkalies are the best medicinal agents for internal use. Locally, collyria of borax or lead subacetate, applied warm, with a four per cent. solution of cocaine, if there is much pain, leeches to the nares or temples, and atropine to maintain pupillary dilatation,[1] are proper remedies. A shade must be worn. Purgation, hot pediluvia, and rest in a darkened room are all advantageous for acute arthritic ophthalmic disorders.

It is noteworthy that these troubles are much influenced by season and climate, being, for reasons which are not far to seek, most apt to occur in spring and autumn.

Integumentary System.

Gouty Skin-Diseases.—Successful treatment of skin-disorders dependent on gouty habit of body demands much attention and skill. It is important to recognize their connexion with this dyscrasia, and as important to refer skin-diseases not so dependent to their proper category. In many instances, there is no history of articular gout in the patient, but the family history and personal proclivities afford the clue, if carefully sought.

The lines of treatment are both local and constitutional. Many of these disorders tend to recur from time to time as does articular gout, and they are often exceedingly vexatious to the patient, and sometimes very difficult to treat effectually. In some cases, it is well not to treat these disorders too actively, and to recognize that they are a lesser evil than others which might ensue were they suppressed. Some are acute and of sudden evolution,—metastatic outbursts; others are very chronic, persisting for months and years.

Pruritus.—This is sometimes very rebellious to all forms of treatment, and occasionally incoercible. I have known instances in gouty old men to persist more or less for years. Accurate

[1] A good collyrium is the following:—℞ Cocaine gr.i., Atropinæ Sulphatis gr.iij., Aq. Rosæ f℥i. Solve. (Atropine is to be avoided if there is any *plus* tension in the eyeball.)

diagnosis is of importance, and the absence of prurigo due to pediculi must be assured. No skin-lesions may be apparent, but some papules may be excited around hair-follicles by constant scratching.

Errors in diet must be corrected; rich dishes, sugar, acids, fruits, and acid vegetables abstained from, likewise all wines, not excepting that of Bordeaux. A little whisky and water is allowable. Sherry is particularly harmful. Occasional mercurial aperients and alkalies may be given with advantage, and a pint of Vichy, lithium, or other alkaline water taken daily with food.

Locally, warm alkaline,[1] sulphur,[2] or bran baths[3] (the bran being placed in a muslin bag), are proper every second or third day. Fine cotton or silk underclothing covered by flannel or merino, according to the time of year, is desirable rather than flannel worn next the skin.

Lotions of borax or of carbolic acid (f℥ij. ad oi.), used hot, are often soothing. Tincture of benzoin may be painted over any very irritable places. Bromides and cannabis indica may be given to secure rest at night, if required. Zinc ointment with a drachm of powdered camphor, or of carbolic acid, to each ounce is useful, and should be frequently applied; or an ointment composed of a drachm of oil of cade to an ounce of vaseline may be rubbed in. A course of waters at Homburg or Kissingen, or the baths of Schlangenbad may be tried. Where any signs of debility are present, mineral acids with bark and strychnine are useful, and arsenic may be employed as a suitable tonic. The treatment of *pruritus vulvæ* I have already discussed (p. 405).

Pruritus Ani.—Warm alkaline or carbolic lotions are useful, and so is ferric sulphate lotion (gr. ij. ad f℥i.). Dusting powders of bismuth carbonate, boric acid, talc, fuller's earth, or zinc carbonate should be freely used after bathing. Any associated hæmorrhoidal tendency or indications of portal venous congestion demand mercurial aperients and restricted diet, soups and wines being especially abstained from. Sulphur in the form of an electuary, or Garrod's compound sulphur lozenge,[4] is often very serviceable in this disorder. Half a drachm of the former, or one or two lozenges may be taken at bedtime for a week or two.

Furunculi-Anthrax.—In these disorders the condition of the urine should be ascertained. Glucose and albumen may, or may

[1] Sodii. Bicarb. ℥iv., Aq. cong. xxx. 96° F.

[2] Potassæ Sulphuratæ ℥viij., Aq. cong. xxx. 96° F.

[3] Furfuris lb.v., Aq. cong. xxx. 96° F.

[4] ℞ Lactis Sulphuris gr.v., Potass. Tart. Acidæ gr.i. ft. Trochiscus secundum artem.

not, be present in it. Sodium salicylate with cinchona, calcium sulphide (gr. ¼ quater die), fresh yeast in ounce-doses thrice daily, or quinine with mineral acids may be tried, if necessary, in succession for recurrent boils. A piece of belladonna plaster or liniment of iodine painted around each furuncule is sometimes soothing and abortive, and so is a saturated solution of boric acid. When the boils point, they may be opened with a carbolized lancet, and dressed with iodoform or resin ointment. Poulticing is objectionable.

In the treatment of carbuncle, which is a disorder of middle or advanced life, especially in persons of gouty habit, supporting, but not stimulating, diet is called for. Alcohol is best avoided unless there is prostration. Milk-diet is best. Poulticing is now commonly disapproved, and applications of belladonna extract with glycerine, in equal parts, preferred. When the surface becomes cribriform and discharges, resin or iodoform ointment may be applied. Opinions now differ as to incising carbuncles. In some cases it may be necessary to practise incisions, and the risk of this is probably less than in former times before antiseptic surgery had won its triumphs. Bark and ammonia, quinine, iron, and mineral acids, with wine, will be useful during discharge of sloughs, and as convalescence proceeds. In cases of chronic glycosuria carbuncle may prove very dangerous and fatal.

Psoriasis.—Treatment relates to the acute and the chronic forms of the disorder. In the former, there is not much to be expected from local or any other treatment in the first instance. Saline aperients with alkalies and colchicum are of most service,[1] and if there is any general plethora, mercurial purges may be given at intervals. The diet must be strictly regulated, so as to ensure as little acid-formation as possible in the system. Local treatment at first is best confined to simple anointing with almond oil or spermaceti ointment. Afterwards, mild tarry ointments, as oil of cade (℥ij. ad ℥i. of simple ointment or soft paraffin, or the same proportion of liquor carbonis detergentis and soft paraffin) or chrysophanic acid (gr.x. ad ℥i.) may be rubbed in, alkaline baths being taken thrice a week. When fresh patches cease to appear, arsenic may be begun, four minims of Fowler's solution or of the liq. sodii arseniatis being given with sodium, or potassium bicarbonate, and nux vomica. Vichy, La Bourboule, or Royat water may be taken with meals.

Obstinately persisting patches should be rubbed with liquor

[1] ℞ Potassii Nitratis gr.x., Potassii Bicarb. gr.xx., Magnesii Sulph. ℥i., Træ. Sem. Colchici ♏xv., Aq. Menth. Pip. ad f℥i. M. ft. haustus ter die sumendus.

potassæ, well-washed subsequently, and smeared with equal parts of zinc and pitch ointment. Sometimes, no treatment avails to remove these patches, and they are then better left alone. They subside occasionally, and other gouty phases appear—in the bladder by metastasis from the legs, and in the thoracic viscera by retrocedence from the trunk.

A hydrotherapeutic course at the Spas is sometimes answerable for a removal of psoriasis and an induction of arthritis.

Care is required in treating psoriasis in persons suffering from bronchitis with pulmonary emphysema, since congestion and hæmoptysis may replace the less grave disorder.

These alternations of process are sometimes very noteworthy in sufferers both from psoriasis and chronic eczema.

The sulphur waters of Harrogate and other Spas are very useful for the treatment of gouty psoriasis.

Eczema.—Eczema demands treatment for the acute and the chronic form. The former is best conducted on the lines already laid down for the treatment of acute psoriasis in respect of constitutional and dietetic measures. Ointments or lotions may be used as preferred. The severe burning and itching of the early stages is best mitigated by zinc ointment, with camphor or carbolic acid, the parts being lightly covered up. The daily bath must be given up if the trunk or extremities are involved. Washing must be less frequent, warm rain, or distilled, water and thin gruel or starch decoction being used, and no soap. Soft diaper towels should be employed, and the parts thoroughly dried and quickly anointed afterwards. Many cases are prevented from healing because the affected parts are too often uncovered and washed.

Useful ointments are the unguentum calaminæ, unguentum acidi borici, unguentum zinci oleatis, and the unguentum glycerini plumbi subacetatis. It is of the last importance for successful treatment to have these freshly prepared whenever possible. Stale and rancid preparations are worse than useless. Carbonate of bismuth, ʒi. ad ℥i., is a very useful preparation, and so is precipitated chalk ointment of the same strength. Pasty lotions of calamine, zinc oxyde, with glycerine and lime-water, are available, but for efficient employment demand care on the part of the nurse and patient. Boric acid in impalpable powder is sometimes very serviceable, and should be dusted on the patches with a powder-puff.

After the acute stage has passed off, washing with soap may be practised, good Castile soap being the best to employ, and the ointment or lotion reapplied for a term.

The too common resort to arsenical medication in these cases I believe to be unavailing, or perhaps worse; alkalies with some bitter tonic being more effectual.

A hydrotherapeutic course at Ems, Schlangenbad, Wildbad, or Royat, with some mild alkaline or aperient drinking-water, is often of use to restore health. Arsenic is available in later stages, or where the eczema shows tendency to persist. It may then be given with alkalies, or taken in the form of La Bourboule water on the spot.

Certain local patches of eczema demand special treatment. That affecting the ears is benefited sometimes by glycerine of tannin, or by mild nitrate of mercury ointment. Compound tincture of benzoin will help to heal others. The unguentum hydrargyri subchloridi (gr. lxxx. ad ℥i.) is sometimes useful for eczema affecting the perineum and for pruritus ani. Chronic eczema in the dry form demands similar treatment to that available for patches of psoriasis, but there is often difficulty in securing full measure of relief. Many cases will only yield to vigorous internal treatment combined with local applications, and sometimes little good comes till mercury and colchicum are employed. Persistent errors of diet must be corrected.

Change of scene and climate is often helpful in chronic cases. Harrogate is a valuable resort both for its summer climate and its waters. The inland air and influences of the European Continent sometimes avail much when drugs and British health-resorts fail to bring relief. Sea-air and influence are particularly noxious for this class of patients, and it is difficult to get beyond these influences anywhere in these islands. Hard waters, both for internal and external use, are always harmful in eczema.

Caution must be exercised in treating cases of inveterate eczema in such gouty persons as are subject to visceral complications. If such occur during treatment, the eczema must be neglected forthwith, and efforts be made to induce regular arthritic gout at a distant part.

Urticaria.—Urticaria demands alkaline treatment. Magnesium and sodium salts, with colchicum, are amongst the best remedies. Persistent tendency to this disorder is sometimes cut short by an emetic of ipecacuanha.

Locally, lead and opium lotion or lactate of lead afford most certain relief. Any idiosyncrasies with respect to food as direct provocatives must be guarded against. Eczema may co-exist with urticarial tendency, causing a very teasing disorder.

Herpes.—The several varieties of herpes vary in importance,

and, indeed, in significance, according to the locality implicated. Zoster is the most severe form liable to be met with in the gouty. The pain may be very great, especially after the vesicles dry up. Boric acid or zinc ointment on lint, firmly bandaged round the chest, affords best relief at first. Quino-alkaline mixture may be given at once, and the subsequent tormenting neuralgia is best treated by quinine in full doses, and by arsenic.

Change of air and walking exercise are of high value in promoting recovery. In elderly patients the suffering may be very severe and prolonged, even for months, and in spite of all forms of treatment.

7.—Treatment of Gouty Cachexia and of Gout in Elderly Persons.

When gouty cachexia is established in any case, the subject of it must be regarded as a confirmed invalid. Such patients are sometimes very pitiable, especially if severely crippled. Life is burdensome, and each day brings a renewed struggle with varieties of incapacity and weariness. The conduct of each case naturally varies according to its precise nature. Degenerations of tissues, more or less wide-spread, are the prominent features, and the symptoms may consequently be multiform and complex. There is commonly advanced renal cirrhosis with associated cardio-vascular change, and progressive failure of cardiac power together with renal inadequacy. There may be grave textural decay with little, or even no, tophaceous deposit. In some cases the latter predominates. Bronchitis is common, and there may be an abiding eczema in parts, usually of dry character. As the latter happens to become active and annoying, relief may come to various symptoms in other parts, and as the eczema becomes quiet, other manifestations may be aroused, either in the lungs or kidneys. Signs of vesical irritation, hæmorrhage, or prostatic gout may supervene.

The mental state varies, and hence there may be great depression or great irritability, peevishness, or querulousness.

Such patients, if crippled, do best at home, and should, when possible, live on one floor. Otherwise, they must be carried about. If the strength permits, winter should be passed in a sunny and sheltered spot. Warm spots on the Riviera may be resorted to, or a climate such as that of Algeria may be recommended. Not many patients, however, could wisely be transported so far from home. Hastings, Bournemouth, and Torquay are amongst the best English winter resorts for most cases.

The diet must vary according to the special indications of each case, and be regulated mainly with reference to the digestive capacity, and the functional activity of the kidneys. Milk, fish, and farinaceous foods suit best, but sometimes a little meat is advisable on alternate days. Wine is not proscribed, as a rule, on the principle that the patient, and not his disease, is to be treated. Two to four ounces of mature port wine, or a smaller quantity of whisky, well diluted, may be taken with meals. The general nutrition is to be maintained as far as possible. Any simple mineral water may be taken. Gentle daily exercise, by walking, or in a carriage or wheeled chair, in the sun, is most desirable. Failing this, or in addition to it, friction of the body should be practised once or twice daily, so as not to induce subsequent fatigue. When the fingers and hands are stiff, a small piece of soft wax may be rolled from time to time in the hands, to promote some degree of flexibility in the parts.[1] Net-making, as an occasional occupation, is also useful.

No treatment by means of baths or mineral waters is practicable or advisable for the subjects of gouty cachexia. The time for this has gone by, and it was noted by Sydenham that no good was to be expected from such methods.

The clothing must be loose and warm, and woollen materials be used for all parts of the body.

Exposure to chill and cold winds must be especially avoided. A fire should be kept up all night in the bedroom during the winter months, and the rooms be kept as uniformly as may be at a temperature of 60° to 65° F. A warm pediluvium at bedtime is very soothing.

Regular action of the bowels is important, and must be secured by the simplest aperients when necessary. No strong purging is permissible.

Sometimes a little tonic and hæmatic medicine is beneficial. Iron may be given in small doses, as the syrup of the iodide, or the ammonio-citrate with some nux vomica. A wineglassful of Spa or Pyrmont water may be taken after a principal meal once in the day. Flatulency, which is often troublesome, may be relieved by spirit of cajuput and compound tincture of lavender, or by the spiritus armoraciæ compositus. In old people, when there is no renal complication but much pain (and they commonly bear pain badly), recourse may be had to bromide of potassium or to some mild form of opiate at night. Twenty to thirty grains of any bromide salt may be given, or four grains of compound soap pill

[1] Recommended by Aurelianus.

with rhubarb or colocynth pill. Paraldehyde in doses of thirty or forty minims is sometimes very serviceable.[1]

Benzoate of lithium with nux vomica is sometimes useful during the day, especially for any vesical uneasiness. An occasional mild saline aperient in the morning may be necessary every tenth or fourteenth day, when a small tumblerful of warmed Püllna water may be used.

The mind must be withdrawn from gloomy thoughts and forebodings, and undue introspection must be discountenanced. Pleasant reading, and the society of cheerful friends avail much to reduce *ennui*, and help to render the little miseries of the cachectic gouty patient less unbearable.

The treatment of gout in elderly persons must be conducted on very different principles from those which are called for in younger persons. All violent and vigorous measures are bad. When more or less acute articular symptoms supervene, they had better not be too much regarded. Colchicum is not often available in such instances. Rest in bed, warm applications, and very simple alkaline remedies are best. Bromide of potassium is a very suitable drug, and may be given several times in the day.

Retrocedent manifestations may be treated by hot pediluvia and moderate purgation, and stimulants are commonly needed and useful. Opium may be required if not specially contra-indicated. The great points to be observed are to avoid *nimia diligentia medici*, and to enforce patience. Special complications may be gently treated as they arise.

8.—Preventive Medicinal Treatment of Gout.

Such measures as may be adopted to avert recurrence of acute paroxysms or of other gouty phases may be appropriately classed under two heads, viz., (*a.*) treatment for long continuance between the attacks, and (*b.*) that for preventing an impending attack.

In the former case, the management of the patient is best conducted by attention to the habits, diet, and mode of life as already discussed. All conditions likely to depress nervous energy are to be strenuously avoided, and the tone of the entire nervous system must be maintained in every possible manner. For the prevailing tendency to urichæmia, the most rational line of treatment is careful attention to the dietary in accordance with the prin-

[1] ℞ Paraldehyde ♏xl., Tinct. Aurantii Recentis f℥ss., Syrupi Simplicis f℥ss., Aquam Destillatam ad f℥i. M. ft. Haustus. Sig. To be taken at bedtime.

ciples already laid down. Medicinally, alkaline remedies are of the highest value; but inasmuch as these cannot always be taken, and constant dosing with physic is objectionable, I believe the best plan to be that of maintaining the due alkalinity of the blood by some potable mineral water, taken for ten or fourteen days in each month. Of these, the most powerful and suitable, I think, is Vichy water, taken to the extent of a pint in the day at and between meals. The Celestins spring furnishes the most appropriate combination. Potass and lithia waters may be used for the same purpose, also Giesshübel, Kroninquelle, Contrexéville, and Bath waters. These, with the Nassau Selters and any of the Rhine waters of the Apollinaris district are suitable at other times. In this country, Buxton and Harrogate may be resorted to in summer, and Bath in winter.

For more direct medication, and for dyspeptic fits, which are often premonitory of paroxysms, there is no better treatment than that by drachm doses of the pulvis rhei compositus taken in peppermint water, with forty minims of aromatic spirit of ammonium, at bedtime. The tinctura rhei in half-ounce doses, with fifteen grains of bicarbonate of potassium in an ounce of chloroform water, may be taken with advantage an hour before dinner.

Patients sometimes dose themselves with strong aperients in order to avert impending paroxysms, and compound colocynth pill with colchicum and quinine is in some repute for this purpose.

Some practitioners combine calomel with acetous extract of colchicum and morphine in a pill to be taken over-night, and followed up next morning with some simple aperient. This plan is sometimes advisable, and may suit such patients as are still vigorous, and unharmed by gouty dyscrasia. There are two cautions to be noted respecting treatment of this kind. One is, that strong aperients may tend to precipitate the evil combated, and aggravate a slight into a severe gouty paroxysm. The other is, that patients relying on the efficacy of this smart medication are apt to be very imprudent in their diet and manner of life, and so fail to restrain their appetites, trusting to the remedy to ward off the evil effects of their vicious indulgence. Treatment of the kind indicated may prove a rude interruption to the general equability of functions which is essential for the comfort of the gouty, and is often better replaced by the milder measures referred to, even if they be repeated over several nights. In either case, the patient is not to consider the medicinal dosage a warrant for continued indulgences, but is to combine with this, as an essential part of the treatment, a careful regulation of his life and dietary.

A remedy of considerable power in warding off attacks of gout is castor oil, taken in doses of one or two drachms early in the morning for two or three days. Where hepatic fulness prevails, with plethora of the portal venous system, and especially in the olive-complexioned arthritic habit of body, this drug is sometimes of great use.

Hepatic stimulants, such as euonymin in gr.ss. to gr.ij. doses, also act well in these cases, generally better with as much calomel taken over-night, and followed by four ounces of Püllna water in the morning.

Two doses of the following medicine taken during the day are of use in many cases:—

℞ Quininæ Sulphatis, gr.ij., Acidi Sulphurici Diluti, ♏iss., Potassii Iodidi, gr.iij., Tinct. Colchici, ♏xij., Decoctum Sarsæ Comp. ad f℥i. M. ut fiat Haustus. Sig. To be taken in a wineglassful of water between meals.

Quinine in a dose of ten grains has been known to cut short a paroxysm, but this practice cannot be commended.

An alkaline aperient such as the following proves useful, and is best taken early in the morning:—

℞ Sodii et Potassii Tartratis, ℥i., Sodii Bicarbonatis, gr.xx., Decocti Aloës Comp. f℥i. M. ft. Haustus.

Colchicum in small doses may be added to any medicine given after gouty attacks, and when any gouty phases are present. No ill effects, so far as my experience goes, are likely to ensue from its use, even if continued for weeks at a time.

Treatment of Blended Gout and Struma.

Treatment for blended diathetic states will vary according as one or the other is more urgent for the time being.

Where gout and struma co-exist, the constitution is naturally frail, and a supporting line of treatment is called for. Strumous ailments are naturally more prominent in early life, but may crop up in later years in the form of senile scrofula. Advanced age may be reached with this untoward combination. Such patients may do well, if happily circumstanced as to means and calling in life. They are more than others vulnerable and liable to break down under excess or riotous living. Country and open-air life is desirable, and sedentary occupations should be shunned. A career in a healthy part of India or on the Prairies may avert many varieties of ailment, and set up the constitution for the remainder of life. Boys thus affected should

be sent from homes in town to good schools in the country, and be forced into all the wholesome activities of English school-life. Girls do best at home, carefully avoiding Continental schools, even under the best auspices, and should lead open-air lives with plenty of active exercise, and not too much study, to which their quick wits may sometimes urge them. Chlorosis may occur in these subjects, also obesity, and iron may be badly borne. It is better replaced by quinine and aperients containing magnesium salts, and by those containing aloes. Sea-air favours repression of the strumous element, but may, unhappily, prove harmful for the gouty proclivity. High and dry situations suit best, with a short seaside residence each year, when sea-bathing may be had.

Good diet and wine are generally necessary with advancing years, and no very vigorous medicinal treatment for any acute gouty outburst is desirable. The waters of Harrogate and Woodhall Spa are especially indicated in these cases, the good influence of sulphur, barium, calcium, and iodine salts being in repute.

Treatment of Blended Gout and Syphilis.

Syphilis, if treated from the first infection *secundum artem*, should after two years give little (if any) subsequent trouble in any case. Owing to many circumstances, inefficient and insufficient treatment is, unhappily, undergone in many cases, and, hence, later phases of lues come to exert a malign influence in the system for many years. Gouty dyscrasia exercises a modifying action on these manifestations, and venereal taint, in its turn, comes to modify the varied expressions of gout. The treatment must vary according to the nature of the earlier management of the syphilitic infection, and the constitution of the patient. Note must be taken, too, of the possibility of unconscious infection, especially where secondary symptoms have been little marked, unobserved, or unheeded.

If the patient be robust, and has had imperfect mercurialization, a mercurial course should be carried out with prudence, and followed up by iodides, alkalies, and sarsaparilla in full doses with tonics. A "washing out" hydropathic course is of much value in these cases, and may be carried out at Harrogate, Strathpeffer, Aix-la-Chapelle, or at Aix-les-Bains. Careful management is required for at least two years, and good results are hardly to be looked for in less time.

Nourishing but unstimulating dietary is called for. The skin-affections may be amongst the most troublesome to treat.

Alkalies with arsenic or Donovan's solution are very useful, and mercurial lotions may be required in the local treatment of obstinately recurring patches on the integument, or on the tongue and fauces. Arthritic swellings and tertiary changes in bones call for iodides and hot douching, and a course of sarsaparilla taken as a diet-drink is of high value. Not less than a pint of the compound decoction should be given daily for weeks together.

A very regular and wholesome life should be led, and all excesses avoided. With temporary decline of health, there is apt to be recurrence of both syphilitic and gouty manifestations, and, hence, worry and overwork are to be guarded against.

CHAPTER XXII.

ON THE SUITABILITY OF ALCOHOLIC AND OTHER DRINKS, WITH GENERAL REMARKS ON THE DIETARY PROPER FOR THE GOUTY.

I HAVE already discussed the dietary proper for cases of acute gout, and that suitable for the intervals between the paroxysms. It now remains to add some special advice respecting alcoholic and other diet-drinks for those goutily disposed, and I shall also discuss here in more detail the employment of certain other articles of food by such patients.

One of the most important points in treating cases of gout relates to the diet in respect of fluids, and more especially to the particular alcoholic fluids that are permissible. On this subject the greatest variety of opinions prevails both in and out of the profession. It would be of interest to draw up a list of these opinions, and it would then not improbably be found that every known drinkable fluid, alcoholic or otherwise, had been found suitable in particular cases. The profession is often twitted with inconsistency and changes of opinion on this matter, and it must be conceded that such charges are not always unfairly made. For a long time it was held that port wine was especially to blame for inducing goutiness; that the stronger wines, such as Madeira, Burgundy, and sherry, were all gout-producing. Beer is notoriously held in disfavour, together with all varieties of malt liquors. Cider has been held to be innocuous. The sparkling and incompletely fermented wines, of which champagne in its many varieties and qualities is the type, are likewise commonly held in disrepute for the gouty. On the other hand, if we were to believe what we often hear alleged by the goutily disposed amongst the laity, we should hold nearly all these liquors to be innocent, and even wholesome for such sufferers. Thus I meet with patients who stoutly affirm that they hold their gout at bay by taking port

wine regularly, and have never been troubled since they found benefit in this fashion. And the same with respect to champagne and cider. There is a practical unanimity regarding malt liquors as being unsuitable and gout-provoking, yet one meets with gouty patients who can take daily with impunity a little mild ale.

My experience has taught me that this impunity is not always so complete in the long-run as is asserted, and further, that it is in most cases quite impossible to lay down rules for patients of whose life-histories and special capacities one is practically ignorant.

Taking the statements of gouty patients as usually made by themselves in these respects, what is the practical outcome of their evidence? We should ask this question with an open mind, and weigh the evidence dispassionately; and a similar question relates equally to the digestive capacity for the various articles of solid food taken by the gouty.

My experience leads me to affirm confidently that each man, and in particular each gouty man, is practically a law to himself; and that the patient, if he be honest with himself, and not a grossly sensual person, can often better prescribe for himself, in respect of the fluids he can best digest, than any physician. For those who have only begun to manifest gout, there are general rules of obvious importance; but for those who have the experience of several or many attacks, it will commonly be found that they well-know what may be taken with impunity, and what is fairly certain to be harmful. Such an experience does not by any means always bring wisdom or the control necessary to forego the harmful agent. Were this so, our task would be greatly lightened. It happens, unfortunately, that amongst the characteristics of the gouty is often a keen and fine sense of perception of the best and most subtle agents that minister to the palate, and with this is commonly found a disposition to yield to these gratifications, and to various other indulgences, and an equally strong indisposition to control such appetites.

It is certain that some gouty persons can take strong liquors with impunity, provided the quantity be small, and they do not mix several varieties at one meal, or take several in any one day. Few can for any long time take champagne, even of the best sorts, regularly. Many can take port wine daily, while others are at once rendered acutely gouty by a single glass of either of these wines. There is fairly unanimous agreement as to the gout-provoking qualities of Burgundy, and few can take even a

little of this for many days with impunity. Bordeaux wine, when mature, is much better borne by the majority of gouty subjects, but the base compounds which pass for the products of the Bordeaux districts under the name of "claret," and which are either factitious, or the mixed products of Spain, Portugal, Algeria, or Australia, having nothing in common with the real article beyond the colour, are commonly very noxious. White Burgundy and white Bordeaux wines are ill-suited to the gouty,[1] unless well-diluted and taken in strict moderation, not exceeding half a pint in the day. Rhenish wines are also acid and harmful; those of the Moselle districts are, however, less acid, and rather better borne. Australian, Californian, Hungarian, Italian, Greek and other Mediterranean wines are too strong, and after a time generally disagree. Exceptions are, however, met with, some gouty persons being able to take some Hungarian wines in moderation.

In the case of all wines, it is probably true that much depends on the quality, and everything on the quantity. Wines that have been long matured in the bottle are least harmful, and, as has been pointed out by Dr. Burney Yeo, those which favour diuresis are also least likely to do mischief. In warm weather, with a freely-acting skin, diluted wine is less likely to be harmful. Strong and sweet wines are most certain to disagree. The fact that many gouty men can drink with seeming impunity, and with alleged benefit, wine that is proverbially known to be gout-inducing, can only be explained on the ground that such wine is really suitable for them as individuals; and it will generally be found that no other wines are taken by them. Hence the apparent paradox that, for some gouty persons, port wine is not gout-inducing, and that even champagne can be long taken with impunity; while, for others, a single glass of either is sooner or later provocative of more or less goutiness. Each individual is therefore a law to himself, and it is certain that no hard and fast rules can be universally appropriate. In practice this is certainly found to be the case. Disregard of these facts has led to certain wines being, as it were, from time to time in fashion amongst the gouty, the doctors being said now to recommend this, and now that—all of which is very unworthy of the profession, since *fashions*, either in physic or dietetics, may be safely pro-

[1] Lecorché is of a different opinion, and recommends white wines for gouty persons. They contain little tannin, a good deal of potash, and act as diuretics. Red wines he is less in favour of, as containing more tannin, which he affirms to cause increased formation (retention) of uric acid.

nounced wrong. The capacity of the gouty for alcoholics of all sorts varies not only in respect of the quality, but very much also in respect of the amount taken; thus, some can only take a little wine occasionally with impunity, and must not indulge in that for many consecutive days. This capacity varies infinitely, too, accordingly as the individual has acquired what may be called an alcoholic habit, and also whether he leads an active life in fresh air, or is confined indoors, in sedentary pursuits, and in the exhausted air of large towns.

In any case, it is certain that the alcoholic habit can only be gratified so far as is compatible with the patient's honest experience of his best digestive state and general health. If digestion be impaired or the general health lowered, the gouty patient is rendered a prey to renewed attacks, or to various phases of discomfort and goutiness.

Cider is sometimes taken with impunity by goutily disposed persons, but it is apt to disagree with most of them after a short time, even when it is sound and in good condition. Unlike beer, cider is not a diuretic. It can seldom be long borne by those leading town-lives. My friend, Mr. Richard Davy, of the Westminster Hospital, has kindly noted his experiences of the employment of cider in North Devon in relation to gout-inducing qualities, as follows:—

"I cannot recall nor ascertain any single case of gout happening to a farm-labourer who has made cider his staple drink, but can mention cases, more than one, of gout complicated with rheumatism where men have been free drinkers of cider associated with gin, beer, or alcohol in mixed form.

"Thirty-seven years ago I recall my grandfather and all the resident yeomen in the Bow district, who were well-to-do and passed easy lives, being the subjects of true gout (evidenced by the passing of chalk-stones), but not one instance of any of their servants or farm-labourers suffering similarly. Their distress was crippling rheumatism, due to exposure and hard work.

"Cider is popularly deemed by them to be a very wholesome beverage; to grant an immunity from stone in the bladder; not to provoke gout, but, as they say, '*if they have the gout in them, cider might feed it.*' Cider is acid, and turns blue litmus paper red. The drinking of three quarts per diem does not affect their loins nor big-toe. In this harvest (1888) a consumption of ten quarts *per diem* per man has not been an unusual quantity. The supply of cider in the harvest-field is practically unlimited.

"Good living, free and mixed drinking, and furthermore, no work with absence of mental anxiety, are generally (by the Devonshire people) supposed to be the prime factors in the production of gout.

"The use of cider in Devon is mostly associated with hard labour and free transudation. It is not drunk (as a rule) in large quantities in the public-houses. Should drunkenness ensue, the resultant headache is prolonged and insufferable."

These observations relate to "rough" or fully fermented cider. Sweet or imperfectly fermented cider is certainly provocative of

gout; the same rule, therefore, holds good with respect to this beverage as obtains in the case of wines. The amount of alcohol in cider is under five per cent.

The drink so commonly taken by the lower classes in London, known as "four ale," is exceedingly gout-provoking. It is acid, and not always free from some degree of lead-impregnation, especially the portion that has lain over-night in contact with pewter pipes, and which is sold at a low rate to topers.

The fact that many persons find themselves compelled to abstain from all strong drinks because they cannot drink them with impunity and comfort, is probably in itself significant of gouty proclivity. In respect of any alcoholic drink, it may be affirmed that, for those goutily disposed, it is often possible to take a certain quantity regularly with impunity. Any deviation from the daily habit, either as to quantity or as to the particular wine taken, may at once induce indications of goutiness.

In this case, as in others relating to the routine and nutritive rhythm of daily life, the importance of equilibration and an even tenour of habit is well seen.

Though the fact does not admit of full explanation, it is practically certain that the gout-provoking qualities of alcoholic liquids are in relation to the more or less completeness of the fermentation they have undergone. It is proved that gout does not exist or tend to develop amongst spirit-drinking populations; that it is somewhat prevalent where wines are largely used, and most established where incompletely fermented wines and malt liquors are freely partaken of. Thus, porter, ale, champagne, Madeira, sherry, canary, Australian, Italian, Greek, and Californian wines are all gout-producing liquors. Good Bordeaux wine is the best natural wine, because most completely fermented, and is of all such liquors the least harmful for the gouty. The wines of the Moselle and the Rhine come next in order, but some of the highest classes amongst these are powerful and very acid. These are admitted to the Carlsbad dietary, and often, I think, with bad results. Those wines in which fermentation is checked and sugar added are the most gout-provoking of all. The lighter beers of Germany, Austria, and Scandinavia appear to be harmless for the gouty, unless taken immoderately. Residents in towns, goutily disposed, leading sedentary lives, are seldom long tolerant even of light Lager beer.

The expense necessary to procure trustworthy wine is a great drawback to the use of even the little that is suitable for the gouty, and hence in recent years there has been recourse to purer

alcohols in the forms of brandy and whisky. The latter is well-borne by the gouty, especially if it be old and mature, diluted and taken with one meal in the day, presumably at dinner.

It is clear from the foregoing remarks that, as a general rule, all alcoholics must be regarded as entailing a measure of risk for the gouty, and Sir Thomas Watson's excellent advice to any young person showing signs of the disease may well be enforced. It was to the effect that it was well worth any such person's while to give up the use of all alcoholics, and become a water-drinker.[1]

It is well-established, however, that total abstainers are by no means exempt from gout in many of its manifestations; and in the case of certain persons who show signs of gout for the first time in middle or late life, it is, in my experience, not a wise practice to enforce complete abstinence from all forms of alcohol. For younger persons the case is often very different, but no hard and fast rule can be laid down. I feel sure that Sydenham was right in condemning water-drinking for the gouty. "Water alone is bad and dangerous, as I know from personal experience. When taken as the regular drink from youth upwards, it is beneficial." Herein lies a great clinical fact which needs to be duly considered, especially at the present time.

The gout-provoking qualities of alcoholic liquids vary in a remarkable manner in different individuals. I have collected from various sources many noteworthy examples in proof of this. Thus, Marchal (de Calvi) records a case of a man, son of a gouty man, who had gravel, in whom a little rum invariably caused pain in the right great toe-joint. No other liquor acted similarly. I have notes of a gouty man in whom whisky caused pains in the joints, and of another in whom Burgundy and champagne taken at dinner were sure to bring out gout on the following day. Another man, if he took any white wine, was sure to be awaked in the night suffering from severe cramps in the legs. Champagne, especially if of inferior quality, will frequently induce this symptom, and cause pain in the small joints of the hands and feet, as well as burning sensation (causalgia) in the soles.

In another case, any wine will, within twelve hours, cause pain in the right metacarpo-phalangeal joint, and set up headache next day. A few glasses of port wine or Madeira will often induce gouty pains in various joints within a few hours, and the same

[1] "With an absence of alcohol in any shape, coupled with an absence of hereditary predisposition derived from alcohol-drinking ancestors, gout would, practically, be unknown."—*Garrod, Lumleian Lectures*, 1883. Lancet, April 7, p. 582.

result has followed within an hour in other cases. The adage that "a man, after the age of forty, is either a fool or his own physician," is especially true in respect to alcoholic habits for the gouty.

In my experience, it is very unwise for a man to make any profound change in his habits of life after the age just mentioned; hence, I cannot countenance, as a rule, any change from moderate wine-drinking or meat-eating to habits of water-drinking and, so-called, vegetarianism. These alterations are surely wrong, and it is within my knowledge that they are sometimes distinctly harmful. If such deviations from civilized life—which, be it remembered, is seldom, if ever, a theoretically *natural* life—are thought proper, they had better be begun in early youth, in which case the results are likely to be very different.

I have, exceptionally, met with a few gouty persons who have found better health after abstention from butcher's meat and all alcoholics, but it will seldom be found practicable, or even desirable, to prescribe such a regimen.

The influence of heredity upon the acquirement of habits by individuals has not been much considered, but it must be certain and potential. In the practice of physic this factor has to be reckoned with, and it can hardly be right, as a matter of routine, to interfere rudely with acquired dietetic habits and tastes which properly represent the requirements of the individual in respect of his trophic equilibrium.

The average amount of alcohol in spirits varies from thirty-five to forty-four per cent. Port wine contains nineteen, Madeira eighteen, sherry seventeen, champagne eleven, Burgundy ten, Bordeaux and Rhine wines eight per cent. Porter contains over six, ale three to six, and cider four per cent. of alcohol.

Wines are more acid than malt liquors, and spirits contain least acid. Malt liquors are less acid than wines, but as much greater quantity of them is taken, they are *virtually* as acid as wines for general consumption. A pint commonly contains twenty-five grains of free acid.

The most saccharine wines are Tokay, malmsey, port, champagne, Madeira, and sherry. Burgundy, Bordeaux, Rhine, and Moselle wines are void of sugar. Porter, ale, and cider all contain sugar, sometimes as much as an ounce in a pint.

The gout-provoking qualities of alcoholic drinks mainly depend on the amount of acid and sugar contained in them. The combination of these two principles gives any one of them its special noxious qualities.

Sugar.—For some years past it has been a common practice to forbid the use of sugar, or to limit it very much, in the case of gouty persons.

It is certain that gout is not prevalent amongst the largest consumers of sugar, and the production and use of this food extend annually all over the world.

It is not proved that sugar by itself is harmful to the gouty; but there is evidence to show that if it be freely taken in addition to a varied and mixed diet, especially with certain articles and with wine, an imperfect fermentative process is set up in the stomach and small intestines, which tends to provoke flatulency and acidity. *It is, therefore, in this manner that sugar proves harmful to those disposed to gout.* The products of the digestion of almost all fruits, with or without sugar, appear to be particularly noxious to such persons, and thus all the varieties of jams, tarts, and so-called sweet courses, are found to be improper for gouty patients. The capacity for digestion of these, as of various fruits, is found to vary much in different gouty individuals. Some can take fruit, raw or cooked, without sugar with impunity, but few can continue to take both. Fruit by itself may be borne in small quantity early in the day, and apart from meals, by some gouty persons, who would suffer if the same were taken after an ordinary meal, and especially if rich dishes or wine formed part of that repast.

The capacity to digest different fruits varies also in healthy individuals. Thus, some persons can take strawberries, raspberries, apples, pears, and bananas with impunity; others digest some of these with difficulty, and are certainly rendered gouty by them. Taken with sugar, fermentative change is very apt to arise, and, hence, fruits are often rightly proscribed together with sugar in the dietary of the gouty. It is certainly safer for such patients to avoid fruits altogether.[1] The vegetable acids constitute the noxious principle contained in them.[2]

Sugar, together with plain food, and in moderation, is scarcely to be reckoned harmful, but the idiosyncrasies of each gouty individual must be taken into account. Patients tell of relief from pains and uncomfortable symptoms after giving up sugar, and, hence, it may be assumed, as in the case of malt liquors, that such food is unsuitable for them, and they are wise to

[1] Linnæus is affirmed to have been cured of gout by eating strawberries. I am not prepared to lend credence to this. Crude cherries have been known to induce a gouty paroxysm.

[2] Sir Andrew Clark informs me that he absolutely forbids all fruits to gouty persons.

abstain. In such cases satisfaction is sometimes gained by the substitution of saccharine, a flavouring matter, be it observed, and not a food, albeit practically harmless. Glycerine may be similarly employed, but is less palatable for continuance.

The sweeter fruits, peaches and grapes, are often ill-borne by the gouty, especially if taken with other food. Melon in moderation, at the beginning of a meal, is probably little harmful in any case. All candied and preserved fruits, such as citron, oranges, and preserved ginger, are very bad for the gouty.

That no untoward fermentative change results during digestion of saccharine and alcoholic matters is proved by the general harmlessness for the gouty of liqueurs, and of, so-called, toddy in strict moderation; but a very different result follows the combination of sugar and wines, or of these with fruits. The vegetable acids constitute the peccant matter both in fruit and wines. Those goutily disposed will, therefore, do well to abstain, as a rule, from sweet or incompletely fermented wines, fruits, and sugar, and are best advised, if they will take alcoholics, to employ either a little matured brandy or whisky, well-diluted, or some genuine and sound Bordeaux wine, also diluted, with one meal in the day, presumably at dinner, and in quantity not exceeding four or six ounces. Sometimes, one or two glasses of mature port wine agree well with the gouty. The point of greatest importance is for the patient to discover for himself what best agrees with him, and leaves him most free from gouty symptoms. It is found that one wine may be best taken by itself, and mixing of several is generally harmful. That which suits should be persevered with, and regular habits as to quantity should be rigidly maintained. In any case, the quality of the wine should be of the best; and if this is not procurable, it is better to avoid wine altogether and take matured spirit with water. The least excess is harmful, but a little good wine is better for most gouty persons than water-drinking, especially after middle life. Changes of dietetic habit, and interruptions to the ordinary routine are apt to upset digestion and to determine gouty symptoms.

Sugar leads by fermentation in the alimentary canal to the formation of lactic acid, which in turn is decomposed into carbonic acid, to combine with sodium and potassium salts. As pointed out by Dr. Ralfe, excess of lactic acid entails excess of carbonic acid, which then forms acid salts of these bases, with acid reactions, by decomposition with neutral salts.

In some persons farinaceous food in excess, or what is for them

excess, will cause too free production of uric acid. Hence, the supply of bread may have to be limited, even if it be apparently well-digested.

A very gouty medical friend of mine declares that "the three poisons for gout are browned fat, grape-sugar, and alcohol."

Certain vegetables are injurious to the gouty. Amongst these are rhubarb, tomatoes, asparagus, and sorrel. Some gouty persons can partake of these in moderation, while others are soon disturbed in their digestion, and suffer vague or localized pains in consequence. Vegetables containing acid are, as a rule, harmful, and if sugar be taken to counteract the acid quality, the combination is likely to be still more noxious. Cooked tomatoes appear to be less well-borne than when in a raw state, probably because a smaller quantity of the latter is apt to be taken. Asparagus in moderation, and taken at intervals, may be tolerated, but if largely indulged in daily, may cause, as I have several times found, lumbar pain and urinary irritation. Dr. George Harley pointed out that glycosuria, lasting for a day or two, and in one case for two weeks, may be sometimes induced by asparagus; and, hence, it may be presumed that the liver is irritated by some active part of the plant, whether asparagin or aspartic acid is not known.[1] The cruciferæ, if well-digested, are harmless and wholesome, and artichokes, salsify, celery, onions, and beetroot may be taken moderately with impunity. Spinach may be taken with advantage in any quantity; turnips are harmless, but carrots should be taken in moderation. Potatoes should not be too freely partaken of, and are best when well boiled or cooked in their skins. Fried and mashed potatoes are less digestible. Peas, fresh or dried, and beans of all kinds, in moderation, are not contra-indicated. Lettuce, fresh and tender, is excellent, but must not be taken unless plain, or in salad with oil and a very little vinegar, in French fashion. English salads are unwholesome,

Rice, in all forms, is a valuable food for the gouty, and sago and tapioca are admissible, provided no large amount be taken at any one meal. Laver is unobjectionable.

Pickled vegetables of all kinds are extremely bad for the gouty. Strongly spiced or salted foods, seasonings containing vinegar, and all acid things are to be carefully avoided. Mushrooms and truffles had better be dispensed with. Most varieties of nuts are

[1] Boerhaave noted the harmfulness of asparagus. Van Swieten, commenting on this, remarked that "healthy people may eat asparagus in any quantity; but many gouty people who have eaten largely have observed it to hasten the paroxysm." Oxalate of calcium crystals, and others of acicular or roughly angular form, are found in asparagus.

harmful, the almond being hardly an exception, unless in the form of fine meal. Chestnuts are perhaps unobjectionable if well-cooked and taken in moderation. Fruits and wine, as commonly taken together at dessert, in consequence of the mingling of harmful vegetable acids and alcohol, are highly noxious and gout-inducing.

Where little exercise is taken and little brain-work is carried on, the dietary may be more largely vegetable and less animal and nitrogenized. The latter induces increased tissue-metabolism. Sedentary habits with excess of food cause a retention of carbonic acid in the blood, and consequent diminution of its alkalinity. Excessive muscular exertion leads to the same condition temporarily.

Gouty patients are very apt to take up new fashions, and to diet themselves unsuitably according to some prevalent theory. They are generally the worse for these practices, and do themselves harm by omitting from their dietary important elements which only require to be moderately taken to prove of use in their general nutrition. Thus, one leaves out sugar, another butter, and another potatoes. No substitutes for these articles can be supplied with the same advantage, and it can very rarely be necessary to forbid *entirely* the use of any one of them for long continuance.

The *doctrinaire* in medicine, as in politics or other matters, is commonly a dangerous person.

In respect of the use of common beverages by the gouty, there is little to be stated. Whichever is best digested agrees best as a rule. A caution is only requisite against the use of strong tea or coffee. The latter is objectionable in any but the smallest quantity immediately after dinner, and is better avoided by the gouty. Sugar should be sparingly used, either with tea, coffee, or cocoa. As a rule, weak tea and cocoa agree best, and the latter is an excellent article for luncheon for those who eat heartily both at breakfast and dinner, and therefore require no animal food in the middle of the day.

All feeding between regular meals is to be avoided, and no alcoholic drinks should on any account be taken at other than meal-times. Much harm is often caused by beef-tea and other food taken at intervals by people who feel weak, but would be refreshed by wholesome occupation or exercise in the open air.

These remarks apply to the moderate use of foods and drinks, and have no bearing on excess in whatever degree, which must always be reprobated, and especially in the case of those of gouty heritage or proclivity.

CHAPTER XXIII.

HYDROTHERAPY, BALNEOTHERAPY, AND SEA-BATHING IN GOUT. USES OF FRICTION AND ELECTRICITY. CLIMATIC RESORTS FOR THE GOUTY.

Amongst the most potent methods for the prevention and removal of gouty ailments must certainly be reckoned treatment by water-drinking and by various baths.

The value of hydrotherapy in gout has been known from very early times. The more accurate knowledge now possessed as to the intimate nature of the disorder affords at once a better explanation of, and a fuller warranty for, its employment.

I have hitherto made little mention of this method of treatment for the varieties of gout, reserving what I have to state for more complete expression in this chapter.

Without doubt, the best results are commonly gained by a combination of water-drinking and bathing, and to these methods must be added the valuable practice of friction, and the varieties of muscular exercise which are usually enjoined at the same time. Over and above these methods there remains to be considered the supreme value of a rightly adapted dietary and regimen, which form an essential part of the course pursued at every well-ordered Spa. Cases of gout in elderly persons, and of gouty cachexia, are altogether unsuitable for hydrotherapy.

Little need be stated respecting the value of a resort to these stations for the many benefits indirectly due to them. In many cases of gout it is important to secure a break in the ordinary routine of life, to remove the patient from his common environments and habits, to provide a change of scene, fresh mental occupation, and a holiday in the true sense of that term. The *medicina mentis* has long been recognized to attach itself to hydrotherapy. The change, in most cases, should afford the enjoyment of a better climate, though it may often involve

diminution of many accustomed comforts, if not luxuries. So many gouty patients come from the class of persons who are daily exposed to luxury, that any trifling discomforts or privations experienced may often be reckoned amongst the most wholesome and beneficial influences met with for the purpose of regaining health. There is often difficulty in urging well-to-do patients to seek the health-resorts that are best suited for them, because they dread the discipline, monotony, and *ennui* attaching to the prescribed course of treatment. People nowadays travel so much and so far afield, that they not seldom have larger experience of the various Spas than those whom they consult, and whose opinion they would fain be guided by. They are commonly intolerant of any resorts that are dull and little frequented by their countrymen, however good they may be, and they occasionally rather seek to combine the exhausting round of excitement and gaiety they are accustomed to at home, with a plan of treatment as little irksome and privative as possible.

Such a pursuit may be harmless enough for such persons as are not seriously ill, but where there is disease to be dealt with, it is simply impracticable, and the physician will fail in his duty if he consigns his patient to any Spa on such conditions. There are many persons whose lives are very dull, and such as to leave little time for pleasure or relaxation, who may derive great benefit from bright and lively health-resorts, and who would be wearied by a serious course with all that is necessarily entailed by it. But these are not the subjects of gouty disease in any intensity, and do not call for consideration in the present connexion.

At the outset, it is worthy of note that mineral waters of the most varied qualities have been credited with valuable properties for those goutily disposed. It would be hard to explain the alleged usefulness of so many and widely differing agents, if there was but one object in view in urging their employment. The fact is that here, as in all therapeutic efforts, the endeavour is, or ought to be, to cure the patient, and not the disease, and to effect this purpose there must be at command, as in the case of other remedies, a variety of agents to meet a variety of conditions. I have tried to point out in various parts of this treatise that gout is, for many reasons, a very different malady in different individuals; that its phases and the degree of its impression vary infinitely, and this fact must be duly taken note of in hydrotherapy, as in any other mode of treatment.[1]

[1] Physicians at various Spas resorted to by both English and French gouty patients note a difference in the effects of the same treatment upon persons of each nation-

There is one main principle which underlies all hydrotherapeutic efforts in the case of the gouty, and it relates to what may be termed a "washing-out" process. Two indications should guide all forms of treatment for gouty patients, first, the elimination of salts of uric acid, accumulation and stasis being thus prevented; and secondly, the re-establishment of the general health on as high a level as possible, with a view to avert the recurrence of uric-hæmia, especial attention being paid to secure full vigour of all parts of the nervous system.

The first indication is met by the free dilution of the blood by water, and alkalies aid in rendering this organic fluid less acid than it commonly is in the gouty. A more complete elimination of uric acid, and an improved hepatic metabolism are secured by saline and aperient elements in some of the reputed waters. Adjusted dietary, friction, and exercise aid further in attaining these ends. The second indication is to be met by recourse after the depurative method to such a climatic health-station as will tend to invigorate and re-establish the general health.

Bathing comes to the aid of the gouty by reason of the influence of thermal mineralized waters on recently or formerly affected joints, and of the improved action of the sweat-glands, which thenceforward afford much depurative relief, and assist the kidneys, the latter being stirred to increased work by the water taken internally. Bathing further promotes active tissue-metabolism in parts less completely reached by water-drinking. The two methods conjointly carried out are most potent in promoting trophic changes.

Copious water-drinking is proved to increase metamorphosis of nitrogenous material in the body.[1] Within certain limits, the assimilation of nitrogenous substances is promoted. The quantity of urine and of its nitrogen is increased during the night. Too copious water-drinking increases the weight of the body, and gives rise to gastro-intestinal and cardiac disturbances.

Hot water-drinking is now frequently practised by gouty and other patients, and I have known excellent results follow its employment. It promotes excretion of uric acid and gravel. Eight ounces may be taken on rising in the morning, and ten to fourteen ounces late at night. Many gouty manifestations and

ality. A greater irritability and sensitiveness is observed in French patients, which is not so manifest in the English.

[1] Dr. Jer. Grigoriantz, Inaug. Dissert., St. Petersburg, 1886. *Vide* Lond. Med. Record, November 15, 1887.

paroxysms may thus be averted, and threatenings of these may sometimes be well-treated by adding some bicarbonate of potassium and lemon-juice to the water.

Not only for the frail, elderly, and cachectic subjects of gout is hydrotherapy or bath-treatment contra-indicated. It is also undesirable where serious cardiac, pulmonary, or renal complications exist, and must be avoided during any active gouty process. Sydenham declared that he expected no good to be gained from mineral waters by those who were advanced in years, phlegmatic, or infirm.

It may be affirmed that the indifferent and alkaline waters are available rather for direct treatment of gouty symptoms, and of less value as radical remedies—that is, as preventives—than saline or sulphureous waters. This knowledge has only been widely spread in more recent years, and, hence, may be explained in some measure the falling off in attendance at many of the purely alkaline Spas, while that at the alkali-saline, saline, and sulphureous Spas has shown tendency to increase.

The various Continental Spas are especially attractive. The more complete change and holiday that is procurable by distance from home, amidst novel surroundings, in more bright, dry, and sunny climate, avails much. The arrangements at these health-resorts, too, with few exceptions, are certainly better in most particulars than those to be found in this country. The patients are less distracted and more amenable to the necessary discipline. The expense in most instances is not greater than that entailed by recourse to home stations. One drawback attaches to nearly all such places, either at home or abroad, namely, their unsuitableness for the greater portion of the year, and especially in the winter months. I know of but one winter station for hydrotherapy in England, Bath, and two or three only are practically available at this season abroad—Aix-la-Chapelle, Dax, and Hamam R'Irha (in Algeria). Carlsbad has now a winter season, but it will be difficult to persuade patients to make a journey from England to Bohemia at that time of the year.

The varied qualities of the waters which are found useful in the treatment of gout and of gouty ailments are truly remarkable. There are for this purpose at least eight varieties or classes of springs, known as (1.) Pure and indifferent; (2.) Alkaline; (3.) Alkaline and saline; (4.) Bitter-acidulated; (5.) Saline; (6.) Sulphureous; (7.) Bromo-ioduretted; and (8.) Ferruginous. These are available to meet all the requirements of gout and gouty states. The difficulty is to deal with such an *embarras*

de richesses, and to consign each case to a station that shall meet its special wants.

It may be affirmed generally that hydropathic treatment is chiefly available for such persons as are possessed of means to benefit by such treatment, with all that it of necessity entails. Both robust and weakly patients may find great relief, but elderly persons are not, as a rule, able to undergo either the fatigue of travel or the various methods which must be enforced, if any benefit is to be derived from such a course.

I propose to discuss the use and value of each variety of water already mentioned for the purposes of treatment of the different forms of gout and gouty ailments; and for greater convenience my remarks shall include their employment both in the form of water-drinking and as baths.

1. Pure and Indifferent Waters.—Of these, there are at least a hundred recognized by authorities on the subject. Some of them are cold, others thermal. The best known and most frequented Spas are Malvern, Bristol, Bath, Buxton, Clifton, Plombières, Gastein, Schlangenbad, Teplitz, Pfeffers, Wildbad, and Ragatz. The thermal springs of Buxton, Bath, Schlangenbad, Wildbad, Gastein, Plombières, Pfeffers, and Teplitz are all available for baths.

BUXTON (Derbyshire).—Buxton occupies a very important place on this list. Its position, 1000 feet above the sea, well inland, with a dry soil and bracing climate, affords all the favouring conditions demanded in a sanitary station for the treatment of gout in the summer season.

A marked feature of the Buxton spring is the large amount of nitrogen gas (ninety-nine per cent.) contained in it, and the absence of oxygen gas. The water issues at a temperature of $81\frac{1}{2}^{\circ}$ F., and requires to be further heated for baths. The best and latest analysis of it is that of Dr. Thresh,[1] who is of opinion that much of the potency of its healing virtue is due to the large quantity (twenty-four volumes in a thousand) of nitrogen gas in a semi-nascent state. Dr. Munk has pointed out that the waters of Gastein and Wildbad are also rich in this element, though to a far less degree than that of Buxton, and this fact is important, and worthy of further study.

Buxton water has a detergent and softening action on the skin, is free from odour and taste, and has a faintly blue colour. It was certainly used more than three centuries ago in the treatment of arthritic diseases. Patients remain about ten minutes in

[1] Buxton as a Health-Resort. By John C. Thresh, D.Sc. 1883.

the bath. The water has a diuretic action. Many cases of all forms and stages of gout derive great benefit from a course at Buxton, and secure freedom from subsequent paroxysms in a marked degree. Advanced cases occurring in persons with constitutions broken down by excesses are not much, if at all, benefited by Buxton, or by any form of Spa treatment.

Cases of saturnine gout in early stages derive much advantage from treatment by indifferent waters. In advanced stages with renal cirrhosis and anæmia, no benefit is to be expected from hydrotherapy.

During acute paroxysms no form of water-treatment is to be practised, but as soon as the parts are free from all active symptoms, bathing is desirable. Great benefit is derived in all cases of incomplete gout. An acute attack sometimes supervenes during a course of baths or water-drinking which may bring relief, but it may be possible to avoid this by concurrent medicinal treatment, and no such attack can be considered a desideratum. In most cases the patients should both drink the waters and bathe.

Bath (Somersetshire).—Bath is available in winter and spring. The Berthollet (natural vapour) bath, as employed at Bath, is a very efficient aid in the cure. Acute gouty arthritis may be thus treated. It may be used generally or locally, and combined with douches and frictions. Hot douches may be used where immersion is found undesirable. It is found best to alternate baths with friction every other day, this plan being less exhausting to the patient. The details of treatment are, however, always best left in the hands of the local medical advisers, whose experience constitutes the greatest safeguard in each case; such details relate to habits of diet, exercise, and general regimen during a sojourn at any Spa.

As a rule, it may be laid down that treatment by the indifferent waters is best adapted for such persons as are not robust and gross in habit, for cases of strongly marked gouty heredity, where there is often an asthenic character in all the phases of the disorder.

Plombières (Vosges).—The waters of Plombières are chiefly used in the form of baths, but are also taken internally. The Bain des Capuçins there was formerly called the Bain des Goutteux. Cases of gout in which nervous erethism is a marked feature are reported to be much benefited at this station, also cases of visceral neuralgia and sciatica. Varieties of douches are much employed, and the Etuves, or hot chambers, are valuable

adjuncts. The water is but slightly mineralized, calmative in its action, and of high temperature, 159° F. The bathing arrangements are very complete, and the neighbourhood is attractive.

Wildbad, Teplitz, Gastein, Leuk, Pfeffers, and Schlangenbad are all available sources for cases of the class just indicated. The high altitudes of many of these Spas constitute one of their most noteworthy features, and doubtless prove useful in promoting recovery from gouty states. Cases in which crippling and deformities of joints are prominent symptoms mostly do well under the varieties of treatment provided at any of them. It is usually advisable to send patients to some subalpine station after a course of bathing in thermal waters. Garrod recommends a course of the Elizabeth spring at Homburg after treatment at Wildbad. Distilled water, plain or charged with carbonic acid gas, is very useful for the goutily disposed, and may often be drunk with advantage.

2. Alkaline Waters.—There are many Spas affording alkaline waters. Those most suitable for gouty cases are Vichy (which may be considered the most typical alkaline Spa), Evian-les-Bains, Neuenahr, Tarasp-Schuls, Vittel, and Royat. Some of these are strictly alkali-saline waters.

Vichy (Allier).—Vichy is one of the most reputed resorts. The waters contain principally sodium bicarbonate, about forty grains in the pint, and vary in temperature from 21° F. to 111° F. The Celestins spring is the most valuable. They are best taken during the intervals between attacks of gout; but, in common with all strongly impregnated waters, whatever be the main ingredients, are unsuitable in cases of atonic gout, and for pallid and weakly patients. Alkaline waters are especially indicated in the case of robust patients who suffer from hepatic troubles connected with gout. Thus, they are useful in glycosuria and in the class of gouty diabetic patients who are often stout.

In gastro-enteric catarrh, with coated tongue and loaded urine, a course of treatment at Vichy, including bathing, often proves highly serviceable. Renal calculi are sometimes passed during such a course. It has often been asserted that the Vichy waters are debilitating. This is denied by M. Durand-Fardel, who has practised there for more than forty years. Garrod believes that tophaceous deposits are liable to be increased by them.

Neuenahr (Prussia).—Neuenahr is in worthy repute for treatment of gouty glycosuria.

Evian-les-Bains (Haute-Savoy).—Evian-les-Bains is a very favourable station for the gouty. It is chiefly resorted to by

French patients. Its position on the Lake of Geneva, 1150 feet above the sea, is all that can be desired. The waters are diuretic and sedative, and of especial value in gouty affections of the abdominal viscera.

Tarasp-Schuls (Engadine).—Tarasp-Schuls, in the Engadine, is also an elevated mountain station. The Lucius-Quelle contains almost as much sodium bicarbonate as the waters of Vichy, but has sodium chloride and sulphate in addition. The temperature is 43° F. Evian-les-Bains and Tarasp-Schuls may be visited after a course at Homburg, Carlsbad, Vichy, or Kissingen, and for such resort, their climates may be sufficiently effectual without recourse to hydropathic treatment.

Vittel (Vosges).—Vittel is available for gouty dyspepsia, diabetes, urinary gravel, and cystitis, and for the same classes of cases as find benefit at Contrexéville. There is less lime and more magnesia in Vittel than in Contrexéville water. Excretion of uric acid is promoted in a very marked manner, as has been proved by Dr. Paul Rodet. Cases of gout with gastro-intestinal, renal, nervous, and atonic manifestations do well at Vittel. Gouty diabetes may be effectually treated there. It is advised that the water should be used every second month for some time. Three or four glasses of the Grande Source may be taken daily, with or between meals.

Royat (Auvergne).—Royat has become an important resort for sufferers from many phases of gout. It is best adapted for asthenic and chronic cases, and for the treatment of gouty skin-disorders, chronic catarrhal pharyngitis and laryngitis. The waters are of the same temperature as that of Buxton, 83° F. Both drinking and bathing are practised.

Contrexéville (Vosges).—Contrexéville is now an important resort, its waters having gained much reputation in the treatment more especially of renal calculi and gravel. Its altitude is 1000 feet above the sea-level, and the climate is sufficiently bracing. The waters issue at a temperature of 53° F., and contain calcium bicarbonate and sulphate, with magnesium sulphate and traces of lithium and iron. They are of especial value in cases of chronic and atonic gout. Their action is laxative, diuretic, and tonic, and, hence, they are better adapted to many phases of gouty disorders than those of Carlsbad or Vichy. Gouty diabetes is efficiently treated here. All forms of urinary gravel, cystitis, and biliary lithiasis may derive benefit at Contrexéville, some of the results being very noteworthy and satisfactory.

Objections are made to the employment of waters containing so much lime salt as is to be found in those of Evian, Contrexéville, Vittel, and others. No full explanation of the benefits derived from recourse to such waters is yet forthcoming, but there can be no doubt of the value of treatment by them, despite the apparent contra-indication of lime salts in calculous disorders originating from uric acid, and in most forms of gout. The fact that large quantities of uric acid sand are passed within a few days of direct treatment by these waters cannot be gainsaid, and I know of no other equally effective method for procuring such elimination as is secured by this method. It is perhaps still more extraordinary, as against preconceived ideas, that alkaline mineral waters, having lime as a base, should prove highly effective in the treatment of oxaluria; but such is the case, as is proved by large experience at Contrexéville.

Dax (Landes, France).—This is an important station, the old Roman Aqua Augusta, but as yet little frequented by English patients. The waters much resemble those of Bath, Buxton, and Plombières. They are thermal, 162° F., and contain calcic, magnesium, sodium, and potassium sulphates, the chlorides of these bases, also iron, manganese iodine, and bromine. Carbonic acid, oxygen, and nitrogen gases are evolved from them. There are also sulphur and iron waters. The bathing arrangements are excellent, and the climate mild. Treatment may be carried out in winter with advantage, and many varieties of gouty ailments may derive benefit here. The vegeto-mineral mud-baths are of great utility, and hot mud is applied locally to affected joints. Unfortunately, the accommodation for the full comfort of patients is not up to the requirements of the present day, but this will doubtless soon be rectified.

Amongst simple alkaline and slightly saline waters, those of a portable class, much used as table-waters, may be here referred to.

The best of these are the Nassau Niederselters, St. Galmier, Apollinaris, Giesshübel, Kronenquelle, and Vals waters. Some of these are largely consumed, and are of particular value when ordinary drinking-water is either hard, or of uncertain quality as to sewage-contamination. Vals water is somewhat similar to, but weaker in soda salts than, Vichy water, and has a slightly chalybeate taste.

3. Alkaline and Saline Waters.—Of these there are many varieties. The best known are those of Carlsbad, Marienbad, Kronthal, and Brides. Vichy and Royat, already alluded to, are strictly placed in this category.

Carlsbad (Bohemia).—Carlsbad has long been held in repute as one of the best stations for the hydropathic treatment of many phases of gout, and there is ample proof of the value of its waters in such cases. It is properly a resort for those of vigorous constitution whose textures are as yet free from marked degenerative change, and whose gouty manifestations are of a sthenic character. The waters are thermal and mainly charged with sodium salts, the sulphate, chloride, and carbonate predominating. Their action is aperient and diuretic, and the urine is rendered alkaline by them. During the course of treatment an aperient action is not specially sought, nor is such necessary to secure the full benefits of the waters. Uratic deposits are found in the urine. Cases of gastro-enteric disturbance and hepatic derangements, including biliary lithiasis and glycosuria, are suitable for Carlsbad treatment. A careful dietary forms an essential part of the course, which lasts for three weeks, but the German *compôtes* should be avoided. The Sprudel spring is used for baths, with or without peat. Patients commonly lose weight under treatment, and this may occur to a serious extent unless due care be taken. It is proper to follow up the course by a residence at some high or subalpine station, of which many suitable ones may be found in Switzerland, Ragatz being a favourite, also Seelisberg.

Carlsbad water should be taken from time to time after the course, and the best results follow a succession of visits to the Spa. Patients are well-advised, I believe, to resort for three seasons of treatment. In some cases it is desirable to prescribe an early summer and an autumnal course in the same year. The waters may be beneficially taken at home with a suitable dietary and regimen, but it is seldom possible to secure the necessary discipline and attention to details amidst the claims and duties of home-life.

Carlsbad may certainly be pronounced one of the best and most useful Spas for the robust classes of gouty patients.

Marienbad (Bohemia).—The waters of this Spa are very similar to those of Carlsbad. The station has the advantage of greater altitude, being over 1900 feet above the sea, or 600 feet higher than Carlsbad. Forests of pine surround both places. It is not so much frequented for the treatment of purely gouty cases, but the waters of the Kreuz and Ferdinand springs are equally available with those of Carlsbad for many phases of irregular and incomplete gout. Mud (ferruginous peat) baths are used, and whey of goats' milk is much employed in the course. Cases of obesity, hepatic disease with portal plethora, hæmorrhoidal

tendency, and uterine disorders are very efficiently treated here.

KRONTHAL (Nassau).—Kronthal is a favourable station for disorders of mucous surfaces. The waters are cold.

BRIDES-LES-BAINS (Savoy).—This is one of the best mountain stations for summer resort. It is situated 1800 feet above the sea-level amidst beautiful surroundings. The waters are thermal (95° F.), and contain chiefly sodium salts with some magnesium and lime, also a little iron. They are diuretic and slightly aperient. In cases of chronic gout, hepatic congestion, gouty affections of the abdominal viscera, in diabetes and calculous nephritis and cystitis, they are of great service.

Patients may go to Brides with advantage after the course at Aix-les-Bains.

SALINS-MOÛTIERS (Savoy).—Salins-Moûtiers, two miles and a half distant, 1500 feet above the sea-level, over a road commanding magnificent mountain-scenery, possesses invigorating springs. The waters are gaseous, hot (95° F.), and contain much iron and arsenic in the deposits, also small quantities of bromine and iodine. Patients, however, taking the course at Salins usually reside at Brides, where there is better accommodation.

Both of these stations will probably soon become better known and frequented.

4. Bitter Acidulated Waters.—These are used as medicinal agents, and their sources are not resorted to.

OFEN OR BUDA GROUP OF BITTER WATERS (Hungary)—RUBINAT (Spain)—PÜLLNA (Bohemia).—The water of Epsom in this country is the type of these. The best known are those of Hunyadi Janos, Friedrichshall, Æsculap, Rubinat, and Püllna. All of these are now used in domestic practice, or employed at other Spas for their special aperient and depurative effects. They are best taken with an equal quantity of hot water early in the morning.

5. Saline Waters.—Of these a large variety is at command. Some of the best included in this class contain other than saline ingredients, such as sulphur and arsenic.

AMELIE-LES-BAINS (Pyrenees).—Amelie-les-Bains is 700 feet above the sea-level, and has thermal waters of sulphureous sodic character. It is frequented by patients suffering from cutaneous and respiratory disorders.

BADEN-BADEN (Duchy of Baden).—Baden-Baden is an attractive station, in high repute for all classes of arthritic cases, and especially for those of gouty and rheumatic origin. The arrange-

ments are very perfect, and the waters are thermal, varying from 110° F. to 161° F., containing chiefly chlorides of sodium, potassium, and magnesium, with calcic sulphate and a little iron. The Fettquelle and the Murquelle springs also contain lithium in larger amount than is found in any other mineral water. Cases of chronic and atonic gout derive benefit. The climate is very hot in summer, and a more bracing station should be sought after a course of the waters.

Bagnères de Bigorre (Hautes-Pyréneés).—Bagnères de Bigorre is a favourite bathing-station, 1750 feet above the sea-level. The waters are thermal, saline, arsenical, sulphureous, and ferruginous. It may be visited in winter, but the high season is from June to September.

La Bourboule (Auvergne).—La Bourboule possesses effervescent saline arsenical waters of high thermality, and is of great service in gouty cases with skin-disorders and neurotic manifestations. Diabetics are well-treated here. The waters are portable, and may be used at home. The bathing arrangements are now very complete. Chlorides and carbonate of sodium are largely present in the water, and each litre contains the equivalent of twenty minims of Fowler's solution of arsenic. It has been called "*l'eau arsenicale par excellence.*" La Bourboule is also as valuable a resort for strumous as for gouty patients, and children derive great benefit from the waters.

Ems (Duchy of Nassau).—Ems, though a favourite station, is little frequented by gouty patients. The climate is relaxing, but elderly patients may be benefited here. The waters are found useful in cases of migraine due to uric acid disturbances, palpitation, and some gouty skin-affections. For gouty bronchitis and asthma it is certainly one of the best stations. Lithiasis, cystitis, and diabetes are also well-treated at Ems, as also cases of congestive dysmenorrhœa, menorrhagia, and chronic uterine catarrh.

The waters are all rich in alkaline chlorides, and thermal. All forms of baths and inhalations are available.

Harrogate (Yorkshire).—Harrogate is one of the best and most widely useful of our English Spas. I refer here to the saline sulphur waters, which owe as much of their virtues to their saline as to their sulphureous properties. These are non-thermal, like all Harrogate waters, but are artificially heated for use. Barium, strontium, iodine, bromine, and calcium chloride are found in small amounts, while calcium sulphate is in but small quantity.

These waters are valuable in chronic gout, hepatic congestion

with constipation, and bronchitis. In chronic eczema, prurigo, and psoriasis they rank second to none in usefulness. Gravel and lithiasis are well-treated here. As an inland station, with a bracing climate in summer, Harrogate has strong claims for attention for many phases of gouty disorder, and the arrangements are excellent and attractive.

ISCHL (Austria).—Ischl is situated 1600 feet above the sea-level amidst charming surroundings. The waters are cold, saline, and sulphureous. All varieties of baths are available, and the classes of cases suitable for treatment include disorders of the digestive organs, chronic skin-diseases, uterine ailments, and nervous derangements.

HOMBURG (Hessen-Nassau).—Homburg-les-Bains is one of the Spas now most largely frequented for all purposes. The waters are saline, acidulous, and ferruginous, richest in sodium chloride, and contain magnesium and calcic chloride and carbonate, with some sodium sulphate. The arrangements are as complete as possible, and the place is attractive, bracing, and salubrious.

Treatment at Homburg is of especial value for atonic gout with hepatic and digestive disturbance. Peat, pine-extract, and other baths are given. Garrod recommends a course at Homburg, either before or after one at Wildbad or Aix-les-Bains.

KISSINGEN (Bavaria).—This is justly a very favourite resort, possessing five springs, mostly rich in sodium and magnesium chloride and magnesium and calcium sulphate. The temperature is between 60° and 70° F., and the waters are warmed before drinking. Their action much resembles that of Homburg waters, and is available for the same classes of cases. The district around is salubrious and attractive, and the general arrangements excellent. The baths are of great value, especially the Soole water, peat, and gas (carbonic acid) baths. Diabetic patients are as well-treated here as at Carlsbad, and cutaneous disorders also derive much benefit. After the course the Rakoczy water may be continued for some time. It is portable, and may be taken at home.

CAPVERN (Hautes-Pyrénées).—These waters are cold, and contain chiefly calcium sulphate. They are especially useful for calculous disorders, and are much resorted to by patients from the South of France. They much resemble those of Vittel.

POUGUES (Loire, France).—This station has an altitude of 780 feet. The waters are highly carbonated, calcic, magnesium, sodium, and iron bicarbonates, with calcic and sodium sulphates, magnesium chloride being also contained in them. The arrange-

ments are very complete. Various dyspeptic ailments are well-treated here, and the place is highly appreciated by French patients.

CHATEL-GUYON (Auvergne).—This is a new station, sometimes called the "Kissingen of France." The waters are thermal, 95° F., gaseous, and contain sodic and magnesium chlorides, calcic, sodium, iron, lithium, and potassic bicarbonates. Patients sometimes resort here after a course at Royat. The action of the waters is aperient and diuretic. Digestive disturbances, hæmorrhoidal tendency, and cases of headache and melancholia are benefited here.

WIESBADEN (Nassau).—This Spa is largely resorted to, and has a deserved reputation in the treatment of many phases of gout. The springs are thermal, 160° F., rich in chlorides of many bases, also in calcic carbonate, but essentially saline. Their action is diuretic and slightly aperient, and is best adapted for languid patients with crippling, who present no marked signs of visceral decay. The baths, taken under strict supervision, are of great use.

HAMMAM R'IRHA (Algeria).—This is a station of importance, especially because it is available in winter. The climate and scenery are charming. There are two springs, one thermal, one containing calcic sulphate, 105° F., and the other cold, impregnated with bicarbonate of iron, 45° F. The arrangements are good, and living is not costly. An inspection of the baths and system pursued here led me to form a high opinion of this place. There are several similar establishments in Algeria.

LEAMINGTON (Warwickshire).—This spa has several powerful waters. The Old Well water is impregnated with sodic and calcic chlorides, sodic sulphate, and is charged with carbonic acid. The saline chalybeate water is more powerful.

CHELTENHAM.—This station has a variety of waters, saline, saline aperient, and iodurettted and sulphur springs.

LLANDRINDOD (Radnorshire).—(*a.*) Saline, (*b.*) sulphureous, (*c.*) chalybeate. The old saline spring at this Spa is of undoubted usefulness in many forms of arthritic ailment, and has been employed for two centuries. It contains 440 grains of mineral constituents in the gallon, consisting chiefly of sodium, magnesium, and calcium chloride, with potassium salts. The water is athermal, laxative, diuretic, and alterant; and being portable and aërated before being bottled, may be drunk at home, either with milk or as a table-water. It much resembles Homburg saline water. There are also sulphur and chalybeate springs.

SARATOGA (New York State).—Saratoga is one of the best of the American Spas. The waters are cold, and have the merit of

being very palatable. Saratoga is (as I can testify after several visits) salubrious and sufficiently attractive. The waters are slightly aperient. The dietetic discipline necessary for gouty patients is, perhaps, hardly enforced as it should be, but, as a matter of fact, there are not as yet many truly gouty patients in the United States to make use of the course.

URIAGE (near Grenoble, France).—Uriage possesses one of the best-ordered thermal establishments in France. The waters are saline, slightly arsenical and sulphureous, with a temperature of 81° F. Their use is adapted to the class of cases for which Harrogate is available, skin-disorders, eczema, herpes, and psoriasis being well-treated here. The place is largely resorted to, bathing being vigorously carried on.

ST. CLAIR SPRINGS (Michigan, U.S.A.).—There are two springs here, and a good establishment in the Oakland Hotel. The alkaline chloride is most employed, but the sulphur chalybeate is also available. These were discovered 1000 feet down while boring for petroleum. The springs are largely resorted to by American and Canadian patients. An inspection of them in 1886 led me to form a high opinion of these waters.

6. Sulphureous Waters.—The main indications for the employment of sulphureous waters in gout are chronicity and want of tone. Skin-affections commonly derive great benefit, especially psoriasis, dry eczema, prurigo of all varieties, and acne. Muscular pains, stiff joints, cramps, and many aches of the gouty, often called "rheumatic," are oftentimes remarkably amenable to treatment by sulphur, both externally and internally. Portal congestion, constipation, and hæmorrhoidal tendency are also markedly under the influence of sulphureous treatment, hydrotherapeutic or otherwise.

Sulphur is held in but feeble combination in the various sulphureous mineral waters in the form of sulphides of calcium, magnesium, and hydrogen. In Germany sulphur has long been used as an anti-arthritic remedy, and, in precipitated form, is still prescribed to be dusted in the shoes, whence it distinctly impregnates the system.

The best of the sulphureous waters contain saline matters, to which, as well as to the sulphides and free sulphuretted hydrogen, much of their good effects are due. Harrogate is the type of such waters.

AIX-LA-CHAPELLE (Rhenish Prussia).—Aix-la-Chapelle has a well-established reputation, but is now more frequented for the treatment of venereal than of gouty ailments. This notoriety

has, for no sufficient reason as regards the unquestionable value of the waters, debarred of late years many gouty patients of both sexes from having recourse to their advantages. The arrangements leave nothing to be desired. Obstinate chronic arthritis, sciatica, and gouty skin-disorders are well treated here both by bathing and water-drinking.

Aix-les-Bains (Savoy).—Aix-les-Bains is now one of the most frequented thermal stations for all forms of gout, and its waters are of great service. The place is attractive in many respects. For obstinate joint-affections, skin-diseases, sciatica, and in atonic gouty arthritis, the waters are of great value. They are of high thermality, 112° to 114° F., and contain calcium and magnesium carbonates and sulphates, sodium chloride, hyposulphites, and sulphuretted hydrogen gas. The system of bathing, douching, and shampooing is carried out with great care and skill. The course is often better preceded by one of water-drinking at Carlsbad or Homburg, especially if the patient be plethoric. The waters of Marlioz and Challes, in the vicinity, are often employed during the bath-treatment. The former of these is rich in sodium sulphide, and the latter contains iodine and bromine in large amount, together with sulphur.

After the course, patients should resort to some alpine station, as Ragatz or Brides-les-Bains.

Strathpeffer (Ross-shire).—This station possesses some of the most powerful sulphureous waters known, and is available for all ailments in which these are indicated. The accommodation and arrangements will probably be improved as this Spa becomes more frequented. There are two springs, the upper one containing more sulphuretted hydrogen than any other British mineral water. Sodic and calcic sulphates are the chief saline ingredients.

Moffat (Dumfries-shire).—There is a sulphur spring here containing sodium chloride. It is of great value in some forms of irregular gout.

Luchon (Pyrenees)—Barèges (Hautes-Pyréneés).—Luchon is available for such cases as may be expected to derive benefit from thermal sulphureous waters, and the same applies to Barèges, where the springs are alkaline as well as saline and sulphureous, and the altitude great, 4200 feet above the sea-level.

Harrogate (Yorkshire).—This station supplies sulphuretted waters of great strength, and may be resorted to by patients requiring treatment of this kind.

Baden (Switzerland, near Zurich).—At this station is a thermal sulphureous spring, much frequented, and of value in chronic and

atonic varieties of gout. The temperature is 119° F. The water is used chiefly for baths, but also for drinking. There are present in it sodium chloride and calcium sulphate and carbonate.

SCHINZNACH.—Not far from Baden is Schinznach, which has stronger but athermal sulphureous waters, containing less lime than the Baden water, and a moderate amount of sodium sulphate.

7. Bromo-Ioduretted Waters.—WOODHALL (Lincolnshire).—The athermal water of Woodhall Spa best represents this class. It has proved undoubtedly beneficial in removing the results of gouty arthritis, and is taken internally, and employed both as baths and in the form of douches. It is portable, and may be taken in doses of four or six to thirty ounces daily before meals.

MARLIOZ (Savoy).—This station has been already referred to. Its sulpho-sodic and bromo-ioduretted waters are of proved utility.

CHELTENHAM (Gloucestershire).—Cheltenham possesses an ioduretted and sulphuretted chalybeate, also ioduretted saline and magnesium saline waters.

8. Ferruginous Waters.—The number of chalybeate springs is legion. They are not generally available as remedial agents in gouty disorders, and such waters are only of service in small quantities in cases of atonic gout with anæmia. The tendency of iron to injure the digestive processes, and to induce retention of uric acid, must be borne in mind in prescribing a course of iron waters. In spite of this, many patients derive benefit, provided that the action of the bowels is maintained. Those waters of this class are best which contain saline ingredients in addition.

SPA (Belgium), PYRMONT (Waldeck-Pyrmont), ST. MORITZ (Engadine), CHELTENHAM, TUNBRIDGE WELLS (Kent), SCHWALBACH (Hessen-Nassau), BOCKLET (near Kissingen), and HARROGATE are amongst the best known and frequented chalybeate springs. Recourse may be had to some of these stations as an after-cure, following a course at other Spas.

As a general rule, however, it may be laid down that sufficient iron is found in most of the waters in repute for successful treatment of gout and gouty states.

ST. NECTAIRE (Puy-de-Dôme, France).—The waters are mixed alkaline and ferruginous, bicarbonated, both cold and thermal, chiefly resorted to by French patients.

OREZZA (Corsica).—This spring furnishes gaseous chalybeate waters, which are largely exported.

Sea-Bathing.—This is rarely advisable, and of doubtful value after the age of fifty. In sthenic gouty cases between paroxysms,

and in young subjects, it may be practised with prudence and in moderation, if found to agree. For older persons and in asthenic gout, warm sea-water baths and douches are of undoubted value. In cases of blended struma and gout, recourse should be had to the seaside for some weeks each summer. The waters of Salins-Moûtiers, already referred to, constitute, amidst the Alps of Savoy, "une véritable mer thermale," likely to be of great value in such cases.

Electricity in the Treatment of the Gouty.

As a restorative of muscular, nervous, and general debility in chronic gout, benefit may be gained from electrical baths. Amyotrophy, due to arthritis, is powerfully influenced for good by voltaic currents applied daily, together with shampooing of the parts. Neuralgia and neuritis in the later stages sometimes yield to this method, and post-zonal neuralgia may be thus treated with expectation of benefit.

Friction in the Treatment of Gout.

The adage of Sir William Temple, to the effect that "no man need have the gout who could afford a slave to rub him," is, I believe, true. As this diplomatist was our Minister at the Hague when Boerhaave was a youth, it is not unlikely that the latter may have heard of the recommendation, and so been led to advise friction in the treatment of gout.[1]

There can be no doubt of the usefulness of regulated friction in promoting a more active interstitial and serous circulation, and in dispersing stases of peccant material. At the present time, friction is much in vogue, and is reduced to a strict therapeutic method by trained persons. Like all other plans of treatment, it is open to abuse, and too much has been claimed for it. It has a place in the management of chronic gouty cases where crippling is threatened, and where other forms of exercise are impracticable. When all pain has passed off in recently affected joints, friction and shampooing may be practised with benefit. At many of the best-regulated Spas this plan is adopted both in and out of the bath, sometimes alternating with the latter. It is especially useful in winter months, when the skin is inactive. Properly conducted, it causes slight fatigue, but promotes better

[1] "Exercitatio magno, continuato, equitationis in aëre puro, *tum frictionibus*, motibusque partium soepe iteratis."—*Aphorisms*, 1275.

appetite and digestion. It must not be overdone. Friction to the extremities is very useful for goutily disposed persons, especially before any debility sets in. The feet should be daily washed with soap and water, and the parts well-rubbed afterwards. The socks and shoes should be changed twice daily, and in some cases each digit, or at least the great-toe, may be provided with a distinct encasement by "thumb" socks. The boots should be easy, and in winter provided with an extra cork-sole. Wet boots should be changed as soon as possible, and great comfort is attainable by having many pairs, so as to vary the pressure peculiar to each.

Travelling in the Treatment of Gout.

Much of the benefit derived from treatment at the various Spas depends on the varied influences inseparable from travelling. Dry, temperate, hill and mountain inland air commonly suits best; but exceptions are met with, some patients finding benefit from marine influence. Where sea-voyages are well-borne and enjoyed, great good may come from them. The unfavourable elements in these arise from the tendency to over-eat and to take insufficient exercise.

If the winter and spring seasons are spent in England, some sheltered, but not too relaxing, place should be selected. It is easier to find suitable stations beyond these islands. The Riviera presents many available resorts. Egypt is too relaxing. Algeria is very favourable in chronic cases, especially with renal complications, and so is Morocco. For patients who are not crippled and not too feeble, great benefit is derivable from a voyage to the Cape of Good Hope undertaken in the early months of the year, or from one to India begun in October. The cool season spent in healthy northern parts of India presents, in my experience, about the best climate obtainable anywhere. Mexico and Southern California also afford brilliancy with invigorating air.

It must always be borne in mind, however, that no climate is by itself helpful to the gouty, unless the habits of life are sufficiently conformed to the requirements of the individual patient, both in respect of exercise and diet. The effects are to be gauged by the condition of general bodily nutrition and nervous vigour secured in each instance. If the traveller is not happy and free to enjoy his new environments, he is not likely to derive benefit. It is sometimes very difficult to induce gouty patients to submit to the varied discomforts of travel. They are prone to

prefer their home-routine, and to court ease and wonted pamperings. It is often well to break in on this, and to stir up dormant energy by the jostle and variety inseparable from any form of locomotion. In suitable cases, it is certain that much benefit comes from occasional travelling, and that the general health is so far restored as to render the system much less prone to suffer from gouty manifestations. Habits of routine and perpetual search for comfort too commonly induce decay of power, and foster textural degeneration. New scenes, and the efforts necessary to reach them, often avail much to rouse latent energies that would otherwise run to decay.

Change of climate sometimes acts in a remarkably beneficial manner on patients with gouty proclivity, causing paroxysmal phases to disappear, and many forms of irregular gout to subside. The most noteworthy results commonly follow resort to inland stations on the Continent. The factors which favour these desirable effects are not known with certainty. I am disposed to think that removal from sea-influences counts for a good deal. The quality of the air, as favouring the action of the skin, and the quality of the water, may also have much to do with the matter. The dietary is often lighter in quality, less strong, and less "English," and much is, doubtless, due to altered habits of life in respect of mental distraction, exercise, freedom from cares and the daily "grind" of home-life. The more brilliant sky, the greater sun-power and general brightness also avail much, in contrast with the prevalent dulness and moisture of the British islands. It is certain that obstinate neuralgias, sciatica, and gouty eczema are thus very favourably influenced by prolonged residence in high and dry stations on the European Continent.

CHAPTER XXIV.

LIFE-ASSURANCE IN RELATION TO GOUT.

A CAREFUL consideration of the importance of gout in the family and life-histories of persons offering themselves for life-assurance, cannot be evaded by those who are called to determine the fitness of such candidates. Opinions differ widely as to the measure of gravity to be attached to gout and gouty history in these cases. It is obvious that no hard and fast line of action can be taken in respect of this, yet it is common in many Societies to affix in a routine fashion an extra premium amounting to three years on such lives.

Each individual demands special consideration in respect of his gouty heritage and his personal gouty manifestations.

Such of the latter as occur in early life must be taken note of, and no less must the phases of incomplete or irregular gout in the adult be duly regarded.

It may be broadly laid down that attacks of frank, regular gout, occurring after forty years of age, are altogether less grave than those which break out before thirty.

In the latter case, there is probably strongly inherited proclivity, and a consequent enfeeblement generally of the constitution. Persons thus early affected are apt soon to become goutily cachectic, and to evince signs of renal or cardiac failure.

Where frank, paroxysmal, articular gout occurs at intervals later in life, the disorder is less apt to lay hold of the constitution, and to lead to degenerative visceral and vascular changes. The relative "weighting" of these two classes of cases should surely be very different. Some persons in whom gout supervenes early in life are not safe for assurance on any terms; while in most of them the existence of the dyscrasia in this form demands a heavy addition. The points demanding attention relate especially to the condition of the kidneys, the heart and vascular system, the liver, and the digestive system. It is especially necessary not to

be misled by the absence of articular symptoms, which may be marked in cases which are otherwise of more serious import in respect of the condition of the organs just mentioned. Care in auscultation and urinary examination, as now practised by medical officers to life-assurance societies, commonly avails to determine the gravity in any case, but it is possible that the fact of associating many minor lesions with gouty taint may dispose the observer to be lenient in his views, and unduly considerate in estimating the probabilities of life.

The association of alcoholic excess in varying degree with gouty habit is another element to be fitly gauged in each instance, and in both sexes; and where met with in obvious extent, should determine against acceptance of the life.

As has been pointed out, some of the worst phases of gouty habit may supervene in perfectly temperate persons, who may nevertheless claim no higher consideration for assurance purposes than the habitually intemperate.

Attention to the specific gravity of the urine in the absence of albuminuria will aid, with other clinical features, in determining the presence or absence of chronic interstitial nephritis, which is so common an attendant on, or rather concomitant of, gout.

The state of the arteries in respect of hardness or brittleness, together with plethoric tendency or the reverse, will avail to guide opinion as to tendency to cerebral or other hæmorrhagic outbursts.

No case of gouty cachexia should be accepted for life-assurance. Sufferers from chronic gout should be declined, if signs of visceral and vascular degeneration are detected. Cases with albuminuria, glycosuria, hard and tortuous arteries, with tense pulse, should be rejected. Emphysema, with bronchitic tendency and cardiac dilatation, are also inadmissible, the liability to suffocative bronchitis and pneumonia (almost always fatal in such cases) being borne in mind. Any degenerations with an associated obese condition are especially bad. Lean and wiry subjects admit of better prognostication under similar circumstances. The peculiar vulnerability of the goutily cachectic should not be forgotten.

This subject has received careful attention from Dr. Symes Thompson,[1] who suggests that in gouty cases, as described above, the existence of any of these degenerations demands more serious attention than is often given to them; and he has expressed the opinion that such indications require, for the protection of the

[1] Med. Times and Gazette, vol. i., 1879, p. 64.

societies, not less than an addition of twenty per cent. to the premium demanded.[1]

The medical officer must do his duty to the Society and to the client, but it not infrequently happens that the latter declines to submit to the increased impost, and seeks admission to some other and less strict assurance company. For the credit of all concerned, it were well that some measure of agreement were established in respect of marking the true significance of gout and gouty indications.

It sometimes happens that clients are unfairly judged because too much importance is attached to minor tokens of gouty habit. Thus, an intermittent pulse may cause undue anxiety. It may be temporary in a gouty person, or, if permanent, of no real gravity. Its true significance is to be gauged by other signs, and these require for their detection skill and experience of such cases. Again, palpitation may cause unnecessary alarm, and excite fears of severe organic heart-disease which is non-existent. Such cases should be referred for a few weeks or months, and re-examined.

[1] Dr. Alexander Davidson, of Liverpool, has recently adduced evidence which confirms in all particulars the opinions here stated. "On the Medical Selection of Lives for Assurance." *Liverpool Medico-Chirurgical Journ.*, July 1889, p. 243.

In the *Medical Handbook of Life-Assurance*, by Dr. James Edward Pollock and Mr. Chisholm, F.I.A., will also be found confirmatory opinions. (This excellent work was issued while this volume was passing through the press.)

CHAPTER XXV.

PROGNOSIS IN GOUT.

In framing a prognosis in any disease, regard must naturally be had to all the factors concerned in its production, and no less to the inherent vital power and resistance manifested by the person affected. In short, the prognosis is for the individual, and not for gouty patients as a class. The same holds good in any disease, and similar considerations arise in discussing the prospects in cases of phthisis, heart-disease, diabetes, or nephritis. The questions are: What is the particular significance of the disorder in the individual, and what degree of resisting power does he possess against its influence? In gout, a special question arises with respect to the habits of life as to diet, exercise, and control of the appetites. The family history avails much to aid in determining prognosis. Thus, even with gouty ancestry, longevity is a powerful and favourable factor. The prospects in the case of one descended from a robust stock are vastly better than those of one who comes of a frail and unresisting one. Regard must be had to the influence of blended diathesis, as struma, and of ancestral intemperance. Where any diathesis is marked, malign results may be distant if it has not passed on to become a cachexia. Where gouty paroxysms have proved sthenic and sharp, and long intervals have occurred between the attacks, little if any curtailment of life is likely. The reason for this opinion is not far to seek, since in such cases there is usually absence of visceral complications and degeneration of texture. The gout is regular, and that is always a favourable sign. Where visceral degeneration prevails, the prognosis is unfavourable. Rank of life, and ability to ward off the ill-effects of attacks, count for much in the determination. Amended habits and self-control count for no less. If gout be associated with struma, and the patient live over forty years, the prognosis is not bad, unless

strumous tendency predominates, especially if care be taken, and the habits are prudent.

In persons with proclivity to vascular disease and degeneration (vascular diathesis), the prognosis is not favourable for longevity, especially if the patient is otherwise of feeble constitution. Where nervous features prevail, as more often seen in women, the prognosis is not unfavourable, provided the nervous element is not highly predominant. As associated with obesity, unless in excess, the prognosis of gout is also not unfavourable.

The varieties of gout were affirmed by Laycock to be dependent on varieties in constitution.[1] He taught that all the diatheses were liable to gouty affections, but that each modified its course and symptoms. The typical English form he regarded as the sanguine; hence, the "sanguine arthritic" or "John Bull" type, with prevalence of sthenic and regular paroxysms. In another type, less common in this country, the "bilious arthritic," he was of opinion that the disease was apt to be asthenic, and to develop at an earlier age.

In the category of the "nervous arthritic" he recognized two forms of gouty disease, and believed that it might complicate either the sanguine or the bilious type, or be combined with struma: (*a.*) *Neuro-arthritic proper*, with tendency to affection of cerebro-spinal axis, with its nerves and their investments; and (*b.*) *Neuro-vascular* or *vaso-motor*, in which the blood-vessels of the nerve-centres were involved by reason of alterations in the vaso-motor nerves.

[1] These views were taught to his class by Laycock, but never published by him. By the kindness of his son, Dr. George Laycock, who lent me for perusal some of his father's manuscript lectures, I am enabled to record them here.

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