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CLASSICAL GOUT*

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"The Gout is so common a disease, that there is scarcely a man in the world, whether he has had it or not, but thinks he knows perfectly what it is."—William Cadogan in *A Dissertation on the Gout*, 1771.

It would seem that custom has sanctioned, and indeed almost imposed, a very definite form of introduction to an address such as this. To this accepted formula I am prepared to subscribe in so far as the usual opening consists in a general confession on the part of the speaker of his unworthiness to be accorded the honour of discussing the subject before so distinguished a body of experts. But I have noticed that there is commonly some passage made from this opening note of humility to a later and slowly emerging atmosphere of omniscience. This transition I shall not make. To the end I shall remain painfully conscious of the fact that upon the very partial knowledge of only a single example I am venturing to generalize upon a clinical condition of the utmost complexity.

We do not greatly value the opinions of a man who, having persuaded some woman to become his wife, assumes that he is the fount of all knowledge concerning the ways of women. We are not in the habit of attaching much importance to the views of any man who, on the strength of possessing a David Cox, poses as a great authority on British water-colours. There is far less reason why regard should be had for the ideas of one who, happening to own a little private gout, assumes that the secrets of that baffling disease are in his special keeping.

As an anatomist I shall naturally be excused from even pretending to any knowledge of the modern developments in the chemistry of the purine bodies; and I shall be pardoned, on the ground of ignorance, from making appeal to the latest dicta concerning the part played by uric acid in human metabolism. I shall therefore content myself by accepting the definition, which I believe to reflect present-day orthodox opinion, that gout "consists in a disturbance of purine metabolism marked by excess of uric acid in the blood." Unfortunately, there does not seem to be complete agreement among authorities on gout that even this definition expresses absolute truths. There are certain other pathological conditions in which hyperuricaemia is a constant feature and yet no signs or symptoms of gout are ever manifested (Cohen, 1948; Mason, 1949). On the other hand, it seems that all that may be said of the association of hyperuricaemia with gout is that "hyperuricaemia is *almost constantly* found" (Cohen) or hyperuricaemia *almost invariably* occurs at some stage in the course of the disease" (Mason). On general principles, it would seem logical to

suppose that, though hyperuricaemia is a very common incident in the course of an attack of gout, it can hardly be regarded as the actual cause of it. Indeed, this appears to be the standpoint of the author of the latest orthodox pronouncement with which I am acquainted. Mason (1949) concludes that "the old saying that gout is a metabolic disorder, repeated in every textbook, is perhaps unfortunate. The more one studies the uric acid problem, the less do such abnormalities as exist appear to be fundamental." We are therefore left with our definition of the disease somewhat the worse for wear; but this is not to say that because it apparently does not express the whole truth it is not a perfectly good definition so far as it goes.

It must be made quite clear at the outset that the only condition I shall attempt to discuss is classical, or regular, gout. This clinical entity I shall not even designate by its accepted name as "articular gout" or "arthritic gout"; for, taking typical podagra as the classical manifestation of the disease, it would seem that it is not necessarily an arthritis, nor even an arthritic condition, in its primary manifestations, but is always and essentially periarticular in its situation. I shall also avoid the adjectival use of the term gout; for a perusal of the literature has persuaded me that the increasing loss of clarity in our conceptions of the essential nature of "periarticular gout" has been due in great measure to the common employment of the term "gouty" for all sorts of conditions that probably have nothing whatever to do with true classical gout. Having thus restricted the field to the discussion of a single but vividly individual entity, I am forced to confine myself, by reason of my lack of clinical experience, to what may be termed the natural history of a complaint the proper understanding of which demands so much more in the way of technical knowledge for its ultimate clarification.

Modern zoologists are at times guilty of discarding as "anecdotal" the observations made by the great naturalists of the seventeenth and eighteenth centuries. But it is to the credit of the physician that he has had more respect for tradition and has never neglected, nor despised, the work of the great writers on the natural history of disease. There is therefore no need to apologize for limiting my remarks to matters of simple observation of overt stages that appear to constitute the life history of a typical attack of periarticular gout.

Precipitating Causes

Whatever may be the underlying disturbance that is responsible for gout there is still needed some factor that fires the train of events witnessed in the progress of an attack. It is perhaps a gain to medical science, and to me it is a great personal satisfaction, that there is a tendency towards rather more charity being extended to the sufferer

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by the physician. To-day there is not the universal condemnation—that “most people who have had the gout severely have deserved it”—which prevailed in John Hunter's time. We hear less of gluttony and of drinking to excess as being the inevitable precursors of an attack, and more charitable views include trauma, fatigue, stress, and allergic and psychic factors among the precipitating causes of an attack.

Whatever it is that pulls the trigger, there is no doubt that the attack, and especially the first attack, comes on with dramatic suddenness. In the poor small hours of the morning the patient is awakened on the sudden and at once passes from a condition of normal ease to one of acute disease by a transition which knows no light or shade, nor any stages of gradation. He is awakened by an intense and distinctive pain in his big toe.

Doubtless long familiarity with the condition leads to some forewarning by which the patient learns to recognize under what circumstances an attack is likely to occur. Sydenham said that in his case the “only forerunner was indigestion and crudity of the stomach”; but that, I think, must be regarded as a purely personal matter. It is probably a case of every man to his own choice in what he considers to be a warning of an attack. The overt objective signs of the attack are swelling and redness in the neighbourhood of the metatarso-phalangeal joint of the big toe. Following Morgagni (*De sedibus et causis morborum*), it is logical to make some attempt to ascertain the actual anatomical basis of the swelling and then to determine its cause.

Seat of the Disease

What is the actual seat of the disease? Does gout have its seat primarily in the joint and is it rightly termed an articular disease or an arthritis? All the older physicians apparently regarded the seat of the disease as being *in* the joint. Sydenham constantly alludes to the “affected joint,” and such is the habit of most of the early writers on gout. Even to-day it is customary for British physicians to term the condition “acute gouty arthritis” (Cohen, 1948). From the supposition that podagra is an affection of the metatarso-phalangeal joint of the big toe arises all that literature in which it is accepted that gout is primarily an arthritic condition. It is here that gout, in British medical science, loses its identity, since it has become only one of many forms of arthritis; and gouty arthritis and rheumatic gout have become commonplace and accepted terms in medical literature.

That a typical attack of podagra is manifest in the neighbourhood of the metatarso-phalangeal joint of the big toe

is patently true; but to postulate that the seat of the disease is *in* the metatarso-phalangeal articulation is a mere supposition: and this supposition would seem to have little real evidence to support it. Even Cohen, who consistently alludes to gout as an “acute arthritis,” admits that “radiological changes [in the joint] occur late in the disease and are non-specific.” Williamson (1920) declared, “in nearly two-thirds of all

cases submitted [to x-ray examination] it not only failed to assist in the diagnosis but tended if anything to lead one to arrive at wrong conclusions”; and Mason (1949) states that “radiological changes which are present in about one-third of all cases” are of little diagnostic value. More than that, it is an incontrovertible fact that after repeated attacks of podagra extended over a period of more than thirty years there may be no signs whatever of arthritis or of any pathological changes in the joint revealed by x-ray examination (see Figs. 1 and 2). The dictum of Jaques Sédillot (1935) that “la goutte n'est pas une affaire articulaire” appears to be well justified.

FIG. 2.—The metatarso-phalangeal joint of the big toe of a man aged 56 who has suffered from podagra for 10 years. Taken at the termination of the acute stage of an attack.

The Swelling

The swelling, even early in the attack, occupies a wider area than that immediately overlying the metatarso-phalangeal joint. Commonly the big toe is involved right to its nail-bed, and the dorsum and medial side of the foot share in the swelling for some distance proximal to the joint. The skin is tense and shining, brilliantly red in the early stages of the attack, and the whole foot is notably dry and hot. It is natural to ask what it is that causes the swelling. Apparently all the early writers on gout considered that the swelling of the painful big toe was due to the accumulation of pathological exudates or humours in and around the joint.

Sydenham regarded it as “gouty matter” or “peccant matter.” Oliver, of Bath, was more vivid in his description. “He represents the gouty matter as becoming acrid, tearing the vessels to pieces, curdling the synovia, and turning it into chalk” (quoted by Ring). All were agreed that some very deleterious humour was concentrated in the metatarso-phalangeal joint and that therefore the rest of the body was well rid of it. From such an argument there arose a belief that an attack of gout was a salutary disease, since the “peccant matter” had become separated and localized and one knew in which part of the body it had become distilled and isolated.

Passing for a moment the views of Caverhill (1769), the observations of John Ring (1811) are of considerable importance. He says: “It is the general opinion of ancient and modern physicians that gout proceeds from an accumulation of humours. Some thinking them a morbid secretion, and others considering them to be mere blood. The latter opinion appears more probable, *from the sudden manner in which the complaint comes on.*” This is, I believe, the first expression of the fact that such a dramatically sudden formation of a tense red swelling is more likely to be caused by a sudden change in vascularity than by the accumulation of exuded humours of any kind. But Ring was not a pioneer in supposing that the swelling might be due to distended blood vessels.

Caverhill, in 1769, had given a clear and coherent expression of a highly confused theory which postulated that the

FIG. 1.—The metatarso-phalangeal joint of the big toe of a man aged 71 who has suffered from podagra for 37 years. Taken at the termination of the acute stage of the attack.

cause of the swelling, and of the pain, in podagra was due to a circulatory upset in the metatarso-phalangeal region of the big toe. Sydenham (1683) had already called attention to the fact that, in his own case, in the early stages of the attack, "only a remarkable swelling of the veins appeared as is usual in all gouty fits." Mason (1949) has called attention to the fact that Garrod (1876) depicted a big toe, in an acute attack of gout, with engorged vessels around the metatarso-phalangeal joint, and Cohen (1948) has summed up the situation by saying, "The skin is dry and shiny, of red or mauve tinge, and the neighbouring veins are distended."

There is therefore a fair amount of collateral evidence that locally increased vascularity, with engorgement of neighbouring veins, is at least a regular concomitant of the suddenly developing swelling. Perhaps what seems to be an almost universal modern tendency to allude to the swelling as "inflammation" is rather to be deplored, since inflammation is a term of rather vague connotation.

There is probably some significance to be attached to the observation so often recorded that, at any rate at some stage in the progress of the attack, the area is not only red and swollen but it is also *dry*. Boerhaave had as an aphorism that "he had never known the gout attack anyone while he continues to perspire" in the feet. Of the sufferer he observes, "His perspiration is checked, especially that of his feet: first the feet become dry, then varicose." The combination of an upset in the circulation of the part with a disturbance in the phenomenon of local sweating is probably of some importance in forming an opinion on the physiological factors involved in the causation of the disease.

Hallmark of the Onset of Gout

When we turn to the subjective aspect of the attack there is no doubt about that: intense pain is the hallmark of the onset of gout. But the subject of pain is a difficult one with which to deal in describing the natural history of any disease, and in gout it is one of more than usual difficulty. An observant physician is able to give a very exact and vividly realistic description of the red and swollen big toe, but when it comes to describing the pain that the patient suffers he is under a severe handicap. In the first place he must needs describe it at second hand: "The patient is the only witness we have, and if he says he has a pain there are no grounds on which we can contradict him" (Sunderland and Kelly, 1949). In the second place, though he may not realize it, he has no adequate language in which to describe it, even should he have ever experienced it in his own person. It is possible that it is these almost insurmountable barriers that prevent some modern clinicians from even attempting to describe the pain attendant on the progress of the disease.

The older generation of physicians sought to give a verbal quality to pain, even if they had to rely on the somewhat crude similes instituted by the patient. We may perhaps disregard their efforts as being, at best, mere word pictures of something not actually realized and so of necessity retailed at second hand and often in the language of persons lacking perhaps both imagination and education. But I think it is beyond doubt that there was a certain flavour about the writings of a former generation of physicians who dealt with the natural history of disease that is lacking in most modern and doubtless more rigorously scientific works. "The patient complains of pain" is a poor colourless substitute for the vividly descriptive phrases that some of the masters of medicine employed.

Classical Descriptions of the Pain

For the moment we will dismiss the handicap that pain must inevitably be described at second hand, for there is a far greater disability imposed by the innate methods of human thinking. I do not wish to labour a point to which I have so often called attention in the past: that man from his first using and fashioning tools and weapons, and on to the phases of constructing simple mechanical devices, to his perfecting a myriad of complicated machines, has always possessed a mechanistic and material imagery and so a mechanistic vocabulary and form of expression. We simply have to face the fact, as all biologists have, that we have no language in which to describe biological or psychic processes. This lack of appropriate language is made evident if we take some of the classical descriptions of the pain felt in an attack of gout. It has been described as resembling the "gnawing of dogs" (Sydenham, Ring, and Garrod), and this seems to be the description most often used.

We all know the story of the medical student who, in the course of an examination, when asked why he had (correctly) diagnosed a swelling as being a varicocele, replied with justifiable confidence: "Because, sir, it feels like worms in a bag." To which the examiner replied with the disconcerting question: "And pray, sir, when last did you feel worms in a bag?" It would be easy to pose the equally embarrassing question to the sufferer from gout: "And pray, sir, when did you last feel dogs gnawing your big toe?" The same criticism applies to the description that the pain resembles a "dislocation of the joint" (Sydenham and Ring), that it is like "tearing the joint asunder" (Garrod), and the many other similes with which the literature abounds. No fault lies with the describer, be he sufferer or physician: the defect is in the fact that, in the absence of a biological vocabulary, recourse must be had to crude physical similes dictated by imagined physical experiences.

Perhaps a nearer approach to truth is the dictum of the Frenchman, quoted by Sir Thomas Watson (1843): "Place your joint in a vice and screw the vice up until you can endure it no longer—that represents rheumatism; then give the instrument another twist and you will obtain a notion of gout." That is certainly the most witty and, in its way, perhaps the most realistic description of gout, but it lacks one thing: it lacks a consideration of the *quality*, as distinct from the *intensity*, of the pain. Gout and rheumatism are as distinct in the quality of the pain that they produce as two pathological conditions can well be. We may well agree with Garrod that "the pain is intense" and with Dyce Duckworth that "the pain increases to positive agony"; but the real secret is revealed in Garrod's further statement that "it is different from other pains." It may be that the first clue to the difference is revealed in the oft-repeated descriptions of gouty patients as being "choleric and irascible" (Ring) or "querulous and irritable" (Cadogan) during the attack: for these expressions indicate in some measure the strangely affective, almost psychical, quality of the pain. As Scudamore (1823) wrote: "The agitation of the mind is much more remarkable [in gout] than in any other inflammatory disease."

A man with sciatica may suffer acute pain, but once he has learned the difficult art of changing position in the manner that produces the minimum of pain he is far on the road to being able to regard the complaint with some equanimity. He dreads the advent of a sneeze; he does not wish to be made to laugh; but beyond that he has no particular anxieties. Sciatica is indubitably a very painful

complaint, but it is one that can be faced with perfect psychic composure.

It is far otherwise with gout: from the first twinge in the big toe a dreadful apprehension overwhelms the patient. He fears lest anyone should come near him and thoughtlessly touch him, or jar him with their footfall, or cause him to move. He dreads the presence of anyone who moves hurriedly, and is terrified lest the fussy person should slip, or drop something, or give him a start of any kind. And in all this it is to be remembered that it is not the actual touch, the jar, or the change of position that causes the pain, it is the apprehension that it might happen. Although there is local pain, enough and to spare, it is the psychic state of dread that characterizes an attack of gout. The initiation of an exacerbation may be caused almost as readily by a mental concept as by physical contact. I do not know the original source of the account of the gouty patient who, having seen a fly upon the ceiling, lay in an agony of anticipation lest it should lose its hold and drop in the neighbourhood of his gouty limb. The picture is doubtless an exaggerated one, and yet it contains a very real element of truth.

Sensation of Heat or Cold

The sensation of local heat that is commonly a pronounced feature of the attack is apparently something altogether apart from the "gnawing" pain. It is not even inevitable that the sensation of heat is present on all occasions, and Sydenham, in describing his own case, said that "the parts feel as if cold water were poured over them." It would seem to be a feature of podagra, as of other conditions that appear to be allied to it, that in the matter of the sensation of heat or cold there are fluctuations during the progress of the attack and differences in individual attacks. Even when the burning sensation is at its height it is obviously a superficial business: it is a cutaneous sensation of heat. The part feels as though the skin were exposed to actual fire and was about to be scorched.

The real and characteristic pain is a deep-seated pain, situated, not in the skin, but deep in the tissues of the affected part. The essential features of this pain are that it is a live, active, and ever-moving pain. As Sydenham said: "Now it is a violent stretching and tearing of the ligaments—now it is a gnawing pain and now a pressure and tightening." One thing only needs to be added to this very realistic description: it is a *radiating* pain. Twitchings, contractions and relaxations, throbbings, pulsations, and flutterings run up and down from the focal point of maximum pain, pulling, tearing, and crushing as they go. Although these feeble descriptive terms are the only ones it is possible to employ, it must be remembered that, though they apparently convey no more than somewhat innocuous connotations, each one expresses a quality that is vividly, even excruciatingly, painful in an attack of gout.

Final Stages

The rest of the story of an attack of periarticular gout is easily told. After a varying interval the pain ceases to be paroxysmal in character and it becomes less in intensity. Some pain may linger for a very long while after the cessation of the acute attack, and after long-continued attacks the normal neutrality of sensation is never restored. Following the cessation of the more acute but non-paroxysmal pain a period of itchiness of the part generally supervenes. This is followed by desquamation of the superficial layers of the skin over the affected area, and it terminates in what can only be described as the "wooden-toe" stage. This final episode consists in the subjective

sensation that the part is hardly a living appendage of the body, but is a more or less insensate extension, the presence of which is realized rather by its annoyingly obtrusive character than by its active participation as a functional member. In the case of larger joints, such as the knee, this subjective feeling of artificiality—and so of unreliability—may persist for years after the initial attack.

So much by way of a personal and probably very biased account of the progress of an attack of acute gout. It remains to be seen if the various phases can be fitted together into a composite and more or less reasonable picture of a definite pathological entity.

Support from Authority

Leaving aside, for the moment, the whole question of the part played by some disturbance of the metabolism of uric acid in the causation of gout, it is worth while to inquire what support from acknowledged authority may be found for some of the other conclusions we have arrived at. There is probably no need to quote further evidence in support of the contention that acute gout is essentially a periarticular and not an interarticular disease and is not primarily an arthritis of any kind. That non-specific articular changes often occur in the course of the disease seems to be a well-established thesis in British medical literature: but that any form of arthritis is primarily, or even essentially, a part of the disease is certainly not a proved fact. In my own case the conclusion that "dans l'accès de goutte du gros orteil, la radiographie montre un interligne metatarsophalangien absolument normal" is certainly correct.

That the swelling is due to a change in vascularity and not to an accumulation of exuded humours or peccant matter has been so often asserted that it may be claimed as being the orthodox view. Sydenham (1683), Caverhill (1769), Ring (1811), and Scudamore (1823) have all testified to the periarticular vascular changes, and de Gennes (1935) has summed up the question by saying: "Sur l'accès lui-même, tout est dit, et nous insisterons seulement sur le signe de la distension veineuse que notre maître, le professeur Vidal, donnait comme le plus fidèle témoin de la goutte." Moreover, two of these observers, Caverhill and Scudamore, formulated theories to account for the curiously localized vascular congestion.

That gout is in essence a disease that affects the nervous system is the whole underlying thesis of Dyce Duckworth's paper published in 1880. In support of this thesis he quotes the previously expressed opinions of Stahl (1737), Laycock (1864), Meldon (1872), Cullen (1872), Liveing (1873), and Paget (1879). He contends "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." In his great *Treatise on Gout*, published in 1889, the thesis is modified to the extent that more importance is assigned to the underlying metabolic change; for he sums up by saying: "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." The nerve-centre involved was postulated by him as being connected with the vasomotor nerves.

Scudamore before him had been a strong advocate for the recognition of the nervous influence involved in the phenomena, but in his time the vasomotor nerves had not been recognized as neurological entities. If Duckworth

may be regarded as being among the pioneers in maintaining the importance of recognizing the neurovascular element in gout, Lancereaux (1910) and, later, Finck (1926) should probably be regarded as pioneers in drawing attention to the parts played by the sympathetic nerves and the vagus respectively in the production of the typical vascular disturbances.

Now that we are free to discuss the part played by the vasomotor nerves, we need not be concerned with the older theories of Caverhill or of Scudamore. But the ideas put forward by these writers are not devoid of interest. Caverhill ignored the knowledge of the capillary system that was available to him and postulated "cohesions" occurring in the course of the "cutaneous arteries" of the big toe. He pictured the breaking down of these cohesions and the stretching of the arteries "above the cohering point" preceding the break as being the causes of the paroxysmal pain. Scudamore recognized the part played by capillaries, but he describes "the pulsatory action of the minute arteries, which appears to be in part owing to the distension of the contiguous parts operating as a ligature." Both these accounts, it must be admitted, are graphic and logical explanations of the observed phenomena in the necessary absence of any knowledge of arteriovenous communications under the control of the vasomotor nerves.

Capillary Circulation

Knowledge of the capillary circulation came rather slowly into the sphere of practical medicine and surgery. Knowledge of the arteriovenous channels, "canaux dérivatifs," or Sucquet-Hoyer canals, is a thing only of yesterday as scientific discovery is reckoned. From the time (1628) when Harvey postulated the existence of the ultimate communicating channels connecting the arterial and venous systems that were beyond his ken, until they were actually seen and described by Malpighi, was a period of over thirty years. Following Malpighi's discovery of the capillaries, almost another century was to pass before Stephen Hales recognized that these minute channels were subject to changes in their diameter in varying phases of the circulation. It was not until towards the end of the nineteenth century that Charles Rouget studied minutely the mechanism of spontaneous capillary movements.

Rouget's work was the natural sequel to the independent discoveries of Claude Bernard, Brown-Séquard, and August Waller (1851-3) of the existence of specialized nerves—the vasomotor nerves—controlling the terminal radicles of the circulatory channels. With this discovery the ultimate nerve-controlled ramifications of the circulatory system became of clinical importance and many obscure pathological conditions were recognized as being caused by some upset in this neurovascular mechanism. In 1878 Weir Mitchell described his "rare vasomotor neurosis of the extremities," and since that time the medical literature of vasomotor neuroses has become almost unwieldy in its volume.

Apart from Malpighi's capillaries and the universally recognized minute connexions between the ultimate radicles of the arterial and venous systems, other arteriovenous communications were postulated by many writers early in the nineteenth century. These communications were defined as being more direct and more open than the minute and tortuous channels of the capillaries. They were claimed to be pre-capillary arteriovenous communications. Sucquet, in 1862, seems to have been the first anatomist who claimed to have demonstrated the existence of these "canaux dérivatifs" or pre-capillary arteriovenous communications. He described them as being present in many

parts of the body "notamment dans le derme sous-unguéal, la peau des faces plantaire et palmaire et des articulations."

Little attention was paid to Sucquet's work, as it seems to have been rather generally accepted that his observations were erroneous. However, in 1878 Hoyer demonstrated by injection technique that viscous solutions incapable of penetrating the capillary bed could pass direct from artery to vein in those situations in which Sucquet had described his "canaux dérivatifs." It still awaited the work of Masson (1924-37) on the pathology of the so-called glomic tumours before the clinical importance of pre-capillary arteriovenous connexions was widely appreciated. From 1930 R. T. Grant, working in the laboratory of Thomas Lewis, demonstrated, by a very beautiful technique, the physiology of the arteriovenous anastomoses in the ear of the living rabbit. To-day these pre-capillary channels, situated in distal parts, are well recognized as being nervously controlled mechanisms, developed in connexion with regulating the loss or conservation of heat.

Heat-regulating Mechanism

One element of this heat-regulating mechanism is possibly of some importance in the study of gout. The arterial anastomoses around joints are among the commonplaces of topographical anatomy, and in their general disposition they fulfil Hart's law, that "anastomoses are more frequent between arteries in proportion to the remoteness of these vessels from the heart." The anastomoses around the joints become freer and more complex as they are traced distally in the limb, and that in connexion with the big toe is probably the most ample of them all.

Although the periarticular arterial anastomoses are so well known, in consequence of their surgical importance, the corresponding plexuses of periarticular veins receive scant attention in works on descriptive anatomy. Nevertheless, the venous periarticular plexuses of anastomotic vessels are more abundant and more complex than those of the arteries. Moreover, connecting these two complex systems there are the "canaux dérivatifs" described by Sucquet. It is this arrangement that permits each segment of the limb to behave as a self-regulating circulatory unit: arterial blood may be short-circuited into the venous system in the region of every joint by means of the arteriovenous channels.

In very crude fashion the mechanism may be likened to a centrally heated hot-water system, with radiators interposed in the circuit at definite intervals, represented by the joints. The outgoing hot water may pass to the terminal (digital) radiators in full spate and heat may be radiated from the distal points. Or it may be that the terminal radiators are more or less closed down, or that the flow through the more proximal radiators is increased. All these things are made real from homely experiences. The trivial phenomenon of "dead" bloodless fingers slowly recovering and flushing, joint by joint, to the accompaniment of a certain amount of pain as the blood supply returns is familiar. Perhaps a more homely manifestation of this same regulating mechanism is within the experience of everyone, for it is probably a universal grievance that long after the body has got comfortably warm in bed the feet remain persistently cold.

A far graver and more impressive expression of the same physiological phenomenon is the centripetal advance, joint by joint, of the progress of dry gangrene in certain cases. Anyone who has witnessed such a march of dry gangrene in the lower limb of a human subject following ligation of the main artery, or has seen similar conditions in lower animals, must realize that at the periarticular anastomoses

and short-circuits of arterial and venous blood a definite and sharp-cut boundary-line exists between the circulation of one segment of the limb and that lying immediately proximal to it.

In a perfectly normal human foot in fresh condition and on which no hardening or preservative reagents had been used, injection was made into the popliteal artery. The injected material was a viscous basis composed of gelatin, and in the mass was incorporated a particulate colouring matter (vermilion). Within a few seconds of the arterial injection being started the big toe flushed pink, and during the next few seconds the superficial veins became engorged and the injection mass returned from the veins at the cut surface of the amputated part. The resulting specimen is shown in Fig. 3, and it demonstrates dramatically the

FIG. 3.—Right foot dissected to show the superficial veins of the big toe and median side of the foot. The specimen, derived from an amputated limb, was injected in the fresh condition via the popliteal artery. After only a few seconds the big toe flushed pink and the superficial veins became engorged. The only arterial channels shown in the dissection are part of the dorsalis pedis and the first dorsal metatarsal artery running to the big toe. The arteries are stippled.

freedom of the passage of even a viscous injection mass (carrying suspended solid particles) from the arteries to the veins in the region of the big toe.

It is a simple (perhaps too simple) matter to picture that some vasomotor upset of this very obvious anatomical vascular arrangement normal to the big toe is responsible for the symptoms of podagra, and that the similar conditions prevailing in the periarticular anastomoses of other joints account for the less frequent manifestations of so-called "articular" gout. If this be so, certain other possibilities are opened up. Perhaps periarticular, or classical, gout might find its rightful place among such an assemblage of vasomotor syndromes as Weir Mitchell's erythromelalgia (erythermalgia), causalgia, Raynaud's phenomenon, acrocyanosis, pernio, and the many other conditions in which autonomic nerve involvement in the limbs are the outstanding manifestation.

There is certainly an almost wearying likeness in the descriptions given by patients, and even by clinicians, of the subjective and objective symptoms of these several pathological conditions. "The pain is throbbing, aching, and burning. . . . Usually it comes in the ball of the foot or of the great toe," which is in a state of "mottled redness as if the smaller vessels were always over-distended." The "pain felt, he said, as though barbed wire were being pulled through the foot. . . . Seeing a man on crutches trip over, or seeing two motor-cars nearly collide . . . brought on exacerbations.

The first of these descriptions is taken from Weir Mitchell's account of a case of erythromelalgia, the second from Sunderland's paper on causalgia, and both seem to

evinced a family likeness to the classical descriptions of an attack of gout.

Two Questions

It would indeed seem that Duckworth's contention that classical gout is a form of neurosis affecting the "nerve-centre" of the vasomotor nerves might have a very considerable element of truth in it. If this indeed be so, two questions remain for answer: "What causes the disorder of the vasomotor nerves?" and, "What, if any, is the action of colchicum on the neurovascular mechanism?" The answers to these questions lie naturally without my province; but certain statements in the literature are not without interest.

Duckworth claimed that the "affection of some nerve-centre occurs as a consequence of the altered blood state *ab intra*." By that he presumably meant that the disordered metabolism of uric acid was the cause of the nerve involvement. H. Frédéricq (1923) has stated very definitely that purines act upon the sympathetic system. Finck (1926) strongly supports him and quotes other authorities, such as Palay, who have investigated the effect of purines on the sympathetic nerves. There, unfortunately, the question must be left by an anatomist who has trespassed outside his legitimate field.

As for the action of colchicum, I believe that even an anatomist may have a word to say from the practical experience of having taken large quantities of it. From Garrod (1859) to Cushing (1947) there is complete agreement that colchicum has no action whatever on uric acid metabolism: but with the passage of time there is a curious change of opinion concerning its action on vasomotor nerves. Duckworth sums up the action of colchicum by saying: "The peculiar benefit derived from this drug is not secured in any other form of inflammation, and this is plainly specific. Its cherished action is doubtless exerted upon the vasomotor nerves." He quotes Meldon and others, who, having tried the effect of the drug upon themselves during health, experienced "a general glow at the surface of the body, diaphoresis, throbbing of the blood vessels and palpitation." Bence-Jones in 1867, stated that colchicum "acts on the nerves of the capillaries, stopping the inflammation of gout." Continental physicians held exactly the same views; but apparently no recent authority on pharmacology credits this action to colchicine. It seems possible that there may be some difference in action between the old preparations made from the corm or the seeds of *Colchicum autumnale* (of which some mercifully persist on the market as "patent medicines") and the pure colchicine available during the last twenty years or so.

If it should ultimately prove to be the case that classical or regular gout is in fact a syndrome connected with some disturbance of the vasomotor nerves controlling the periarticular plexuses, a further interest attaches to its sex incidence. That it is almost exclusively associated with masculinity is an observation dating from the time of Hippocrates. So much importance has been attached to this fact that at one time castration was recommended for its cure. On the other hand, Raynaud's phenomenon, which in so many of its features is the antithesis of classical gout, is (apart from industrial vibratory trauma) essentially a syndrome manifested in females. The study of the inheritance of these two conditions obviously opens up an interesting field to the geneticist; and some scattered facts are already available to form the basis of such a study.

Conclusion

I have tried hard to keep the vow with which I prefaced my remarks, and I have been careful to trespass as little

as possible outside the sphere of my own experience. As a consequence I am well aware that I have not discussed the general problem of gout. I know well that the condition that I have been discussing under the name of regular or classical gout is not the only disease passing under that name. I have not spoken of arthritic gout, or tophi, or chalkstones, or deposits of urate of soda. The clinical entity that is distinguished by these characteristics seems to be a lineal descendant of the rheumatic gout of the early nineteenth century that was so graphically portrayed by Adams.

The relationship of the arthritic condition to the spasmodic vasomotor disturbances manifested in classical or periarticular gout can be determined only by the clinician. Since all scientific nomenclature depends upon the question of priority, it seems clear that the vasomotor syndrome may rightly claim the name of gout; and some other name should be given to the gross destructive arthritic condition—unless it can be clearly shown that this is the definite sequela of a previous periarticular vasomotor disturbance.

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SIGNIFICANCE OF HAEMOGLOBIN LEVEL
IN THE PUERPERIUMITS RELATION TO BIRTH WEIGHT AND PRODUCTION
OF BREAST MILK

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There have been innumerable studies of haemoglobin in pregnancy, and some have been continued into the puerperium. Very few, however, have included an attempt to assess quantitatively the value of high or low levels, and the advantages of a high level have usually been assumed without extensive proof. One such attempt was made by Reid and Mackintosh (1937a) in their analysis of data obtained in Stockport. They placed about 1,000 women in two groups according to their economic circumstances, and found that when a low level of haemoglobin during pregnancy was associated with poverty it apparently led to an increase in the neonatal and infantile death rates and in the amount of illness in the first year; the period of breastfeeding was also likely to be curtailed. A low level of haemoglobin did not seem to bring similar disadvantages to the higher-income group.

The present work consists of an analysis of the results of about 1,500 haemoglobin estimations made on women who were delivered in the Landesfrauenklinik der Rhein-Provinz, Wuppertal, between June and December, 1946. The estimations were carried out on the seventh day after delivery, and always by one or other of two hospital laboratory assistants. The Sahli method was used, and the colour was assessed in an optical haemoglobinometer; 100% on the scale of this instrument was said to represent 14 g. of haemoglobin. The nutritional status of the women is discussed in full elsewhere (Dean, 1950), and in the same place it is reported that in the years when the food supply was reduced—which included 1946—the children were smaller at birth than children born in times of plenty, and mothers had much more difficulty in establishing lactation. There was no rise in the incidence of any of the diseases of pregnancy or the puerperium, and the children appeared to be normal in every way except size. The mothers were not given iron during pregnancy. Their diets contained large quantities of vegetables and therefore adequate amounts of vitamin C. There were no signs of vitamin deficiencies and none of the women had oedema of nutritional origin.

Unfortunately, estimations of haemoglobin had not previously been done as a routine at the Landesfrauenklinik, and it was therefore impossible to make comparisons with the pre-war years. Cases of multiple pregnancy, caesarean section, and intercurrent illness have not been included in the analyses; cases of post-partum haemorrhage have been considered separately in the sections dealing with lactation but have otherwise been omitted. It was the custom to weigh, and not merely to estimate, the blood loss at delivery.

Results

General.—The distribution of women with high and low levels of haemoglobin is shown in Table I. If the women who lost 500 g. of blood or more at delivery are omitted, more than 20% of the total number were in the range of haemoglobin up to 77% and less than 80% were in the higher haemoglobin range.

The Queen's Institute of District Nursing has issued a pamphlet entitled *Houses for District Nurses*, describing the type of house and district room recommended by the institute as most suitable for district nurses and midwives. It has been prepared in response to inquiries from a number of medical officers of health and nursing associations. The plan and appearance of the house are depicted, accompanied by a short description. Copies may be obtained for 1s., post free, from the institute at 57, Lower Belgrave Street, London, S.W.1.