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A FIT OF GOUT (PAROXYSMUS PODAGRÆ): A STUDY IN PATHOLOGY.

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A FIT OF GOUT (PAROXYSMUS PODAGRÆ): A STUDY IN PATHOLOGY.[1]

By GEORGE W. BALFOUR, M.D., LL.D. Edin. and St. And.

"Non fingendum, aut excogitandum, sed inveniendum quid Natura faciat, aut ferat."—BACON.

GOUT as a disease has been known to physicians from time immemorial, yet its most graphic description is of no earlier date than Sydenham, who, in memorable words, has depicted it with all the skill of a physician and all the accuracy of a sufferer. Succeeding writers have but copied Sydenham's delineation of the disease, and have modified his theory of its rationale. This, as we know, was a reversion from the doctrine prevalent in his day, of tartar accumulated in the blood and deposited as chalk stones in the joints, to the still older theory of an effort of the system to expel a peccant humour, the result of imperfect coction. To Cullen's logical mind we owe the differentiation of gout into its several varieties of regular, atonic, and aberrant. And to Cullen also is due the statement that the supposition of any morbific matter is unsupported by any evidence, is quite superfluous, and is useless, "as it has not suggested any successful method of cure."[2] Cullen's own idea was that gout "is manifestly an affection of the nervous system."[3] Modern advances in zoo-chemistry and in morbid anatomy have introduced various modifications of our ideas as to the ultimate pathology of gout. Some of these modifications are more closely allied to the humoralism (Haig, etc.), and others to the solidism (Ord, Bristowe, etc.) of our forefathers; while even those who with Cullen regard the nervous system as the *primum mobile* of this disease (Duckworth, etc.) are forced to recognise the exciting influence of a pre-existing toxæmia.

[1] Read before the Edinburgh Medico-Chirurgical Society, 4th May 1898.
[2] "First Lines," cccexcii. p. 375. [3] *Ibid* cccexciv. p. 381.

With all our increased accuracy in details, it does not appear that our ideas of what gout really is are any clearer or any better defined than those of our forefathers. To the non-professional mind gout presents itself in two aspects—first, as an embodiment of ill-temper, an agonising toe, somebody's specific, and short commons; and, second, as a meaningless epithet, vaguely applied by physicians to all ailments occurring after middle life which are obscure in their origin, variable in their symptoms, and not readily amenable to treatment. As to the professional idea of gout I speak with all humility, having been unable with any certainty to discover what is the prevalent professional conception of this disease; but I presume, subject to correction, that the clearest heads among us regard it as a toxæmia with a uric acid basis, the nervous system as a ruling spirit, and the whole organism as a *campus martius* wherein to exercise. What idea our younger brethren entertain of gout it would probably be hard to imagine, but it must seem to them as a most mysterious complication of life, when they are taught that not only may it hop like a bird from one part of the body to another, but also that such very tangible affections as "hæmatemesis and hæmoptysis are often cured by a regular fit of the disease" (Gairdner). A former member of this society, Dr. Craigie, in his "Practice of Physic" has said that it is in the natural history of gout that its real character and nature is to be sought; and this idea has been repeated and emphasised by the late Dr. Todd in his "Croonian Lectures." This view I myself homologate, and I am persuaded that a clear idea of the true nature of a paroxysm holds the clue to much that seems mysterious in the history of gout, and puts into our hands the means of preventing, if it cannot always enable us with any certainty to cure, its manifestations. The natural history of a paroxysm may not explain all the mysteries of gout, but it gives us a clue that will suffice to guide us through the labyrinth of symptoms presented by this protean malady.

I cannot, I regret to say, show you an illustration of a gouty toe in its paroxysmal condition. So far as I know, no such delineation exists; and he would be a bold artist indeed who should propose to sketch such a toe from nature. A mere fancy sketch would be of no value for my purpose, so I must ask you to recall the simulacrum of such a toe if you have ever seen one; and if you have not, you may still, I hope, be able intelligently to follow me as I point out and endeavour to explain the rationale of the several signs described by Sydenham and others.

Podagra, you may remember, is the name given by Cullen to the twenty-third genus of the order Phlegmasiæ, class Pyrexiæ. Cullen adopted the name Podagra from Boerhaave and the older writers, because it correctly, if somewhat vaguely, described the great characteristic of the disease (a foot seizure) without com-

mitting him to any definite theory of its nature, explicitly rejecting the term Arthritis as misleading, and as conveying an idea (of inflammation) for which there is absolutely no foundation. Podagra is distinctly a febrile (pyrexial) disease; it has also all the characteristics of a phlegmasia, but of the three characteristics by which an external inflammation is recognised—*calor, rubor, et tensio dolens,* the first is conspicuous by its absence. Moreover, podagra invariably terminates by resolution, and never ends as inflammations so often do, either in suppuration or in gangrene.

Sydenham tells us that on the morning after the commencement of an attack, the part affected—the metatarso-phalangeal joint of the great toe—is found to be swollen, but that up till this time "the only visible swelling had been that of the veins of the affected joint."[1] Turgidity of the veins coming from the affected joint is thus the earliest visible sign of an attack of gout, and this sign persists till convalescence sets in. But venous turgescence is not an accompaniment of inflammation. The veins of an inflamed part undoubtedly carry a larger amount of blood than in health, because the dilated arteries of an inflamed part convey to it a larger amount of blood than usual, and this has to be returned by the veins, but these vessels remain inconspicuous and unnoted amid the general turgidity of the part. In the case of a paronychia, for example, which is an inflammatory affection of the distal extremity of a digit, and in situation, at least, closely resembles a fit of gout, there is redness, heat, and painful swelling, but the veins are neither turgid nor visible. The dilated arteries carry an excess of blood to the part, but the blood path is everywhere free, and the blood is speedily restored through the veins to the general circulation. Should, however, the blood path be choked by an embolus or a thrombus, the case is altogether different. When such a block occurs, in a vessel with many collateral communications, the circulation is scarcely checked and is speedily restored to its normal. But should the block occur in a terminal artery, an anæmic area is at once formed in front of and around the obstruction. When the circulation around this area is carried on through capillaries and small arterioles, no true infarction is formed; but from the sluggish movement of the blood within the anæmic area a stasis occurs in the vessels, which, if visible at all, is most evident in the veins leading from the part up as far as a more or less distant point where they enter a sphere in which normal conditions prevail. Within the anæmic area the augmented tension and diminished vitality of the walls of the vessels, due to the sluggish flow of the blood, cause an accumulation of plasma in the part, accompanied by a diapedesis of the corpuscular elements. Hence there is swelling of this area; and as the red corpuscles part with their colouring matter to the plasma

[1] *Syd. Soc. Translation,* p. 125.

the swelling is of a dusky redness, which gradually deepens till the normal conditions are restored.[1]

Now, if we turn to a gouty paroxysm, and limit our consideration to its essential characteristics, we find that it commences with a sudden attack of agonising pain in the part affected—the metatarso-phalangeal joint of the great toe. But pain is not an exalted condition of nerve function. As Anstie has said, pain is the prayer of the nerve for more blood, for better nourishment. Whatever interferes suddenly and more or less completely with nerve metabolism gives rise to intense pain. Hence the excruciating agony that follows the occlusion of a vessel, whether artificially, as in the treatment of aneurysm by compression; or naturally, as in gangrene following blocking of an artery by embolism or thrombosis, or in many other similar processes. In a gouty paroxysm the excruciating agony comes on suddenly, and is perfectly explicable, on the theory of thrombosis of the small vessels of the joint affected. Moreover, the sequential phenomena are precisely those that would follow the formation of an anæmic area in and around the joint implicated. First of all, before there is much if any manifestation of local swelling,[2] there is visible turgidity of the veins leading from the affected joint up to the dorsum of the foot, till they merge into a sphere where normal conditions prevail. Next, we have swelling in and around the joint, from accumulation of plasma within the anæmic area. And, lastly, this swelling becomes increasingly tense, stretching the skin and giving it a glistening appearance, which is perhaps all the more striking from the deepening dusky redness due to the solution of the colouring matter of the red corpuscles of the plasma.

All the events of a gouty paroxysm are thus readily explicable, on the theory of thrombosis of the small vessels of the joint affected, and upon no other theory with which I am acquainted; and all the concomitant phenomena are corroborative of this view.

For example, a gouty paroxysm is commonly ushered in with a rigor followed by a rise of temperature, phenomena of daily occurrence in connection with embolism—with sudden blocking of the circulation. The affected joint presents, we know, many of the indications of inflammation, the *rubor* and the *tensio dolens* are very much in evidence, but the *calor* is conspicuously absent. I have occasionally taken the temperature of the distal extremity of a gouty toe, and found it to be invariably subnormal. Sir Dyce Duckworth[3] tells us the same story. He says he has repeatedly taken the temperature of a gouty toe by putting a thermometer close to it and keeping the two in contact for twenty minutes

[1] *Vide* paper by Dr. M. Litten in the *Ztschr. f. klin. Med.*, Berlin, Bd. i. S. 189; and Cohnheim's "Lectures on Pathology," *New Syd. Soc. Translation*, vol. i. p. 121.

[2] Sydenham, *op. cit.*, p. 125.

[3] "A Treatise on Gout," p. 248. The bodily temperature in gout is seldom over 102°, but has been known to rise to 104°.

with a roll of cotton wool,[1] and has always found it subnormal. On two occasions it was 97° and 95°·5, the corresponding temperatures in the mouth being 100° and 100°·4. I am aware that the temperature of the corresponding toe on the opposite foot has also been found subnormal, but, however we may explain this,[2] it does not lessen the remarkable fact of the distal part of the affected toe, which is tumid, red, and painful, and to the sufferer feels hot, having a temperature two or three degrees below that of the body generally; a state of matters which seems wholly inexplicable on any other theory than that of a stasis of the circulation at the part affected.

After a fit of gout the tissues about the joint and its cartilages are found to be infiltrated with urate of soda, a condition trifling after a first attack, but which increases with each succeeding paroxysm. This condition Garrod regards as of the highest importance as indicating a specific form of inflammation,[3] a form of inflammation which would indeed be not only specific but peculiar and altogether *sui generis*. On the other hand, we know from the researches of Garrod himself that in gout the blood serum contains uric acid,[4] and that this is also found in the serous fluid within the pericardium, the peritoneum, and the subarachnoid space of gouty subjects.[5] The concretions of urate of soda upon the auricular cartilages and on other parts, and the nodosities about the digital joints, known as gouty pearls and Heberden's knobs, are not preceded by any inflammation, but probably follow some slight local injury. At first—if we see them early enough, which is rarely the case—we find a small localised and, as it were, encysted effusion, within which the urate of soda slowly crystallises, the remaining serum is absorbed, and the pearl or knob is left behind. The so-called pearls rarely grow, generally indeed become harder and smaller; but the knobs, by a repetition of the process, do grow, and more and more disfigure the extremities.

Urate of soda is a somewhat insoluble salt, always present in the blood serum of gouty patients; and when the plasma accumulates within the anæmic area of a gouty toe, the salt slowly crystallises out of the extravascular fluid, and gets left behind, infiltrating the cartilages, the cavity of the joint, and the tissues surrounding it, when the serous part of the effusion gets reabsorbed, as the circulation returns to its normal. Each returning paroxysm increases the amount of these extravascular urates, and also the

[1] This method of taking the temperature makes it absolutely certain that it was subnormal, as by covering the bulb of a thermometer with cotton wool we may so prevent the dissipation and favour the accumulation of heat as apparently to raise the perfectly normal temperature of either the mouth or the axilla up to 108° or even higher.

[2] The low temperature of the opposite foot may be due to reflex contraction of its arteries from the intense pain of the podagra. Just as Brown-Séquard and Tholozan found that pinching raised the temperature of the limb pinched but lowered that of the opposite one.—*Gaz. méd. de Paris*, 1870, p. 142.

[3] "Gout," third edition, p. 187. [4] *Ibid.*, p. 84. [5] *Ibid.*, p. 109.

disfigurement of the joint; but their deposit is not the result of any inflammatory action, nor is their presence in the joint ever the cause of any inflammation, beyond the disfigurement; their presence in and around the joint seems to give rise to no disturbance and to be absolutely neutral, so far as the state of the person so disfigured is concerned.

There is nothing, therefore, in the history of a gouty paroxysm which connects it with any form of inflammation, and all the phenomena present are readily—and seemingly only—explicable on the theory of thrombosis of the small vessels of the joint affected, and the formation of an anæmic area around their sphere.

Nay more, the mode of prophylaxis, which has commended itself to many sufferers as a certain preservative from all the ills of an acute attack, as well as one method of cure which, in the hands of a bold practitioner, has proved eminently successful, are not to be explained, so far as I can see, upon any other theory whatever.

Cullen narrates some curious instances of the sudden cure of gout. One man, long confined to bed with gout, when his neighbour's house took fire, ran off as fast as he could. The provoked antagonist of another patient, dressed up as a spectre, and throwing the sufferer over his shoulder, dragged him downstairs, bumping his poor feet against every step; at the bottom of the stair the ill-used man found his legs, and ran upstairs fast enough. While a third, on the first premonition of an attack, took his dog and his gun, and walked it off.[1] Sir William Temple tells us of the Rhyngrave, who was killed before Maestricht in 1676, and was a friend of his own; on the first indication of a paroxysm this nobleman went out immediately, whatever the weather, and walked as long as he was able, "pressing most upon the foot that threatened him; when he came home, he went to a warm bed, and was rubbed very well, and chiefly upon the place where the pain began." If the attack continued or returned next day, the same course was pursued, so that he was never laid up with gout; and he thought so well of his treatment that he strongly recommended it to his son, should he ever be attacked in the same manner. Temple also tells us of one of his brother's gamekeepers very subject to gout, but who never laid himself up. When attacked he busied himself with his deer or his stud, walking about from morning to night in spite of the pain.[2] And the late Dr. William Gairdner of London was acquainted with an active, lively old gentleman of 85, who, when seized with gout, always replied to the remonstrances of his family and his physicians, "I'll walk it off," quaintly adding, "Go to bed with the gout, and it will surely go to bed with you, and be mighty bad company."[3] A remark that recalls the state-

[1] "First Lines," 2nd edition, 1778–84, MS. notes, p. 389, etc.
[2] "The Works of Sir William Temple, Bart.," Edinburgh, 1754, vol. ii. p. 127.
[3] Gairdner, "On Gout," London, 1849, p. 114.

ment by Sir William Temple, that gout haunts chiefly those who treat it as a friend, and not as an enemy, who "carry it to bed with them, and keep it soft and warm, and indeed lay up the gout for two or three months, while they give out that the gout lays up them."[1]

These are sufficiently remarkable statements, based upon equally remarkable facts, yet we are told that the idea of attempting to abort a fit of gout could not occur to anyone "who regards it from a proper pathological standpoint."[2] To myself it seems quite the other way about, and that it is only to those who have a correct idea of the pathology of gout that it could legitimately occur to attempt to abort an attack. We are told that "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] But we never hear of any evil results following the successful aborting of a fit by active exercise; Dr. Gairdner's old friend carried it out to the end of his life. A friend of Mr. Apperley (the Nimrod of old times), after a skinful of wine and other good things, always finished the evening by walking off the soles of his pumps before retiring to bed, in order to ward off gout, and this he did successfully and without damage to the end of his days. Cullen, it is true, tells us of one man who, after warding off many attacks, was suddenly laid on his back by a severe fit,[4] but his failure to check this one could scarcely be attributed to his having successfully overcome previous attacks. Both Cullen and Duckworth, regarding gout as largely an affection of the nervous system, look upon the good results that follow this method of fighting off a fit as an example of the curative effect of "great mental emotion,"[5] or as an instance of the "remarkable influence exerted by a vigorous nervous system on the peculiar phenomena of a gouty fit."[6] But no mental emotion, however great, nor any power of will, exerted even by the most vigorous nervous system, can produce any effect upon a gouty digit, unless it be accompanied by movements of the joint sufficiently energetic to dislodge any thrombi, break up the stasis, and restore the circulation in and around the joint affected. On the other hand, we know that manipulation and friction, carried out with or without the will of the patient, are sufficient to restore the circulation and to cut short an attack without any ulterior bad result. It is true we hear but little of this method of treatment in medical literature, yet it must have been occasionally employed, as when Boerhaave, in his 1275th aphorism, under section 8, speaks of curing gout "*exercitio magno, continuato, equitationis in aere puro, tum frictionibus, motibusque partium saepe iteratis*,"[7] the con-

[1] Temple, *op. cit.*, p. 128.
[2] Duckworth, *op. cit.*, p. 348.
[3] Duckworth, *loc. cit.*
[4] *Op. cit.* p. 392, MS. note.
[5] Cullen, *op. cit.*, p. 389.
[6] Duckworth, *op. cit.*, p. 349.
[7] Boerhaave, "Aphorismi de cognoscendis et curandis morbis," Lugd., Batavorum, 1727, p. 212.

text shows that he speaks of these as approved methods to employ in the cure of an acute attack, and not merely as means to be used for the removal of the rigidity of gouty limbs during intervals of comparative health.

It may be readily understood that, however efficient this method of treatment may have proved itself to be, it is not likely to have been often or lightly resorted to. When the trifling succussion caused by merely crossing the floor to the bedside is liable to provoke intolerable agony, he must be a bold practitioner who would venture to propose to apply massage to the offending digit itself. Yet this has not only been proposed, but also successfully carried out with the most astonishing results.

In the first half of this century we had in Edinburgh a well-known practitioner—Dr. William Balfour[1]—who had a great reputation for his successful treatment of rheumatic affections, by the various methods we now comprise under the term massage. Occasionally he attacked more acute ailments with a boldness and a success which were truly remarkable. In the *Edinburgh Medical and Surgical Journal* for October 1816,[2] Dr. Balfour relates several cases of acute gout, treated by compression, friction, and percussion. These cases are all remarkable, and all perfectly successful. I shall quote only one, as that is sufficient to show his method and its results:—

Case.—Mr. J. O., solicitor, æt. 47, of a cholerico-sanguine temperament and full habit of body, woke in the morning of 22nd July with violent pain in the balls of both great toes, particularly at the flexures of the joints. "When I visited him at eleven o'clock forenoon, the parts were swelled, red, tense, and shining. The inflammation and pain were confined almost exclusively to the balls of the great toes, except that in the left, which was by much the worst, the pain reached upwards about halfway along the metatarsal bone. The patient experienced continual excruciating pain, even when at perfect rest; his countenance indicated considerable distress, and he complained of sickness, general uneasiness, and oppression; pulse, 82.

"When I entered my patient's room, he told me that, if I intended to apply friction to the parts affected, he was afraid he could not suffer

[1] William Balfour was the younger son of Alexander Balfour of Tillery, Kinrossshire, where he was born in 1769. He graduated at the University of Edinburgh in 1814, and shortly afterwards, as is stated in an obituary notice in the *Lancet* of 9th May 1846, he "commenced a career which, under whatever aspect it is viewed, has seldom been exampled in the medical world." His paper entitled "Observations on Adhesion, with Two Cases demonstrative of the Powers of Nature to restore parts which have been by Accident totally separated from the Animal System," was both original and acute, and characterised by perspicuous and forcible language, qualities by which his style was uniformly distinguished. This paper was transferred to every scientific periodical of repute, and at once made him famous. He subsequently turned his attention to the treatment of rheumatism and gout, and speedily acquired a large and lucrative practice. He died in 1846 at the age of 77.

[2] P. 432, "Observations, with Cases, illustrative of a New, Simple, and Expeditious Mode of Curing Gout."

it; for he had attempted that himself, but might as well have applied 'living fire.' I told him I knew that friction was quite inapplicable to his case as it then stood, but that I would, notwithstanding, employ mechanical means for moderating the paroxysm, and I hoped with complete success. I therefore grasped the ball of the toe in my hand, applying pressure gradually, to a very considerable degree. Slight pressure gave pain; heavy pressure none. In this manner I compressed the parts, sometimes with one hand, at other times betwixt both hands, for about ten minutes, now and then interposing gentle percussion; and then applied a thin flannel bandage. Ordered a brisk purgative of senna and sulphate of magnesia."

In the evening the patient said he "had considerably less pain, or rather none at all, from the moment I left him; but he had a good deal of cold shivering during the day;" his pulse was 100. The swelling and redness of the parts had much abated, and motion of the affected joints was practicable. Compression and percussion were reapplied, and were much better borne than in the earlier part of the day. Next morning the patient was found to have slept all night without interruption; his pulse was 80; pain, swelling, and redness of the parts almost entirely gone, and he could suffer them to be handled with the utmost freedom; he walks very well. Compression and percussion were applied in the morning, in the evening, and again next morning, after which the patient went abroad in the prosecution of his business—on the third day of his attack.

Such a case—even if it were a solitary one—would be truly remarkable, but as one of a series it is more than remarkable, it is instructive. The case and its cure are so graphically detailed as to leave no doubt as to the nature of the one or the reality of the other. Dr. Balfour points out that, as the roll of the waves does not at once cease when the gale drops, so also the assuaging of the local ill was not at once followed by the cessation of the symptomatic fever, which indeed increased for the first few hours, but speedily thereafter passed away. The general oppression, the sickness, and the dyspeptic symptoms were also relieved, though this, as the doctor wisely remarks, was no doubt largely due to the purgative remedies employed, and not altogether to the cure of the joint affection; the latter he rightly ascribes to the restoration of the circulation in the part affected.

As we have seen that all the phenomena—objective as well as subjective—of a gouty paroxysm are apparently only explicable on the theory of thrombosis of the small vessels of the part affected, so now we find that a gouty paroxysm can be readily and rapidly cured by measures which can only act by restoring the circulation through the part affected, and which are well calculated to affect this result. But, however delightful it must be to be cured of a bad fit of gout by one or two séances of a practised masseur, I fear there would be a difficulty in getting any one to submit to an operation which seems to bring with it the possibility of such agony. And I am equally persuaded that there are few,

if any, who possess sufficient nerve and skill to carry out effectively so delicate an operation.

On a future occasion I hope to return to the subject, to point out the rôle of thrombosis in relation to all those various affections we term gouty, to endeavour to expiscate the causes of thrombosis in gouty subjects, and to show how they may best be warded off; at present I must content myself with having directed your attention to what seems to be the true cause of a gouty paroxysm, which, as Craigie and Todd have told us, is the first step to a clear understanding of the true nature of gout itself. And I cannot conclude better than in the words of Sir William Temple, who quotes from a Dutch friend who had long lived in the East Indies, that there "the general remedy of all that were subject to the gout was rubbing with hands; and that whosoever had slaves enough to do that constantly every day, and relieve one another by turns, till the motion raised a violent heat about the joints where it was chiefly used, was never troubled much, or laid up by that disease."[1] I can so far corroborate this that the few cases I have persuaded to employ massage of the extremities night and morning, have since then kept free from any severe attack.

[1] *Op. cit.*, vol. ii. p. 127.