

complex, however, as may be seen from the result of the researches of Wright and Luff. In *veratrum viride* they found jervine, pseudo-jervine, cevadine, a little rubi-jervine which is the same as Bullock's veratroidine, and traces of veratrine and veratralbine.

I may summarise the medical uses and properties of the drug. It reduces the force without at first lessening the frequency of the pulse, but after a time the pulse-rate falls very much. If exertion be made during this stage of depression the slow pulse will be suddenly converted into an exceedingly rapid one. The slow pulse is sometimes moderately full, but is always soft and compressible. The rapid pulse is exceedingly feeble and small and may become imperceptible. Severe nausea and vomiting accompany or follow the reduction of the pulse-rate. During the stage of depression there are always decided muscular weakness and relaxation. *Veratrum viride* is looked upon as a powerful spinal and arterial depressant exerting little or no direct influence upon the cerebral centres. Its alkaloid jervine lowers the pulse rate by its direct action on muscle and its alkaloid rubi-jervine or veratroidine lowers it by stimulating the inhibitory nerves. Its jervine diminishes the force of the heart beat by a direct influence upon the cardiac muscle, and by producing a general vaso-motor paralysis more or less complete according to the size of the dose. By the action especially of the jervine the spinal motor nerves are directly depressed. Neither the sensory centres nor the motor and sensory nerves are distinctly affected. *Veratrum viride* is used in practical medicine, therefore, to reduce arterial excitement and to quiet spinal spasms.

Remarks.—The case is interesting as one of puerperal eclampsia treated solely by the action of *veratrum viride*, and therefore one is presumably free to deduce that the improvement which followed was due to the action of the drug. The outstanding features in the action are the lowering of arterial tension, the slowing of the pulse-rate, and the absence of convulsive seizures after the dose of the drug was given. Within fifteen minutes of administering the dose there was a perceptible change in the character of the pulse. It had been firm, hard, and bounding, but now became softer, was full and easily compressible. The number of the beats was also reduced from 92 or 100 to 84 and there was slight inequality in the value of the beats. Five minutes later the pulse-rate was reduced to 72; in another five minutes the pulse had become 54 and fifteen minutes later still it had reached its minimum of 52. Afterwards for two hours the pulse-rate was taken at intervals of fifteen minutes and kept varying from 54 to 60 per minute. When taken five hours later it was still 56 and five hours later again it was 60.

Although the reduction in the rate of the pulse is a striking feature it must be remembered that in this case the pulse-rate was never very high, 120 being the maximum—at any rate, after the case was seen by me. The most significant feature, however, was the action of the drug on the arterial tension evinced by changing the firm, bounding, resisting character of the pulse to one of a soft, full, and compressible nature. And this condition of pulse continued fairly well marked for ten hours after the injection of the drug. The influence of the drug in relaxing muscular tissue was shown by the speedy dilatation of the rigid external os. The most objectionable feature in the exhibition of the drug was the retching and vomiting. There was also a considerable amount of salivation which gave trouble by producing cough. The skin of the patient kept moist.

So far the etiology of puerperal convulsions is obscure. The frequent albuminuria with which it occurs gave rise to the opinion that it was the result of uræmia, and although cases occur where there is no albumin present, and other patients with an abundance of albumin escape, it is thought that renal insufficiency is the most frequent if not invariable cause. Traube and Rosenstein refer its causation to acute cerebral anæmia resulting from changes in the blood incidental to pregnancy, the watery condition of the blood being associated with increased arterial tension. Dr. Angus Macdonald, of Edinburgh, found from post-mortem examinations extensive anæmia of the cerebro-spinal centres with congestion of the meninges without oedema. He attributed the convulsive attacks to irritation of the vaso-motor centres from an anæmic condition of the blood produced by the retention in it of excrementitious matters that ought to have been thrown out by the kidneys.

Applying now the recognised physiological actions of

veratrum viride we know that one of its alkaloids, jervine, is able to reduce the force of the heart-beat by its direct influence on the cardiac muscle and that it is capable also of producing a general vaso-motor paralysis; that it likewise reduces the pulse-rate by a direct action on muscle, while the other alkaloid veratroidine by stimulating the inhibitory nerves also reduces the pulse-rate; and both alkaloids—namely, the jervine and the veratroidine—are, in addition, depressants to the motor centres in the spinal cord. We have therefore in *veratrum viride* an agent the physiological properties of which meet the supposed pathological conditions in puerperal eclampsia—namely, increased arterial tension and cerebro-spinal excitement.

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THE PATHOLOGY AND TREATMENT OF GOUT.¹

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SINCE the discovery of a compound of uric acid in gouty concretions by Wollaston in 1787 and the subsequent discovery fifty years ago by Sir A. Garrod of uric acid in the blood of gouty patients the relationship of uric acid to gout, either as a cause or a result, has been universally conceded. Observers and writers on gout have, however, been divided into three schools, accordingly as they held the view—(a) that the uric acid compound only exerted a baneful effect after it had crystallised from the blood and had become deposited in the affected tissues, or (b) that while dissolved in the blood it exerted a true toxic influence, or (c) that the uric acid was merely a by-product in the gouty process—that is, a result of certain changes in the system which by themselves constituted the disease of gout.

The view which regards gout as a disease which is the result of a true toxic action exerted by the uric acid compound dissolved in the blood is untenable for these reasons: (1) there is no experimental proof that uric acid is a poison; (2) a gouty subject just prior to the advent of an attack of acute gout shows no signs of poisoning although the fluids of his body are saturated with a compound of uric acid; and (3) in cases of leucocythæmia and severe anæmia the blood is frequently highly charged with uric acid without the production of any toxic symptoms that could be referred to that body. Again, the various views as to the uric acid being merely a by-product in the gouty process fail to explain many of the phenomena of gout, a subject which I fully discussed last year in the Goulstonian Lectures on the Chemistry and Pathology of Gout.² The remaining view, then, that the compound of uric acid only exerts a baneful effect after precipitation from the blood and deposition in the tissues is, in my opinion, the most tenable one and is the view held by such eminent authorities as Sir A. Garrod and Sir W. Roberts. This view regards the soluble uric acid compound as being destitute of poisonous qualities and as producing no harmful results so long as it remains dissolved in the fluids of the body. When, however, the fluids become over-saturated with this compound a crystalline deposition of sodium biurate occurs which then acts as a mechanical irritant to the tissues and structures in which the deposition takes place.

As all observers are agreed that an abnormal quantity of uric acid is present in the blood in gout, such overcharging of the blood with uric acid must be due to one or more of the following causes: (1) production of the uric acid in the normal manner but insufficient excretion of it; (2) over-production of uric acid while the excretion remains about normal; and (3) diminished destruction of uric acid by imperfect oxidation or by some other means. This last theory may be dismissed at once. There is no proof that the process of oxidation or any other process going on within the organism destroys uric acid; on the contrary, there is proof that uric acid is produced by a process of

¹ A paper read before the Harveian Society of London on Jan. 6th, 1898.

² THE LANCET, March 27th and April 3rd and 17th, 1897.

oxidation. The second theory that there is an over-production of uric acid in the system while its excretion remains normal is untenable, since it is based on the assumption that the kidneys can only eliminate a certain amount of uric acid, whereas there is abundant experimental proof that an increased production of uric acid such as occurs throughout the system in cases of leucocythæmia and severe anæmia does not lead to gout so long as the kidneys remain in a normal condition. In these diseases the blood may be laden with uric acid and the daily excretion of that body may rise to six times the usual amount, yet such a quantity is readily excreted by the kidneys and no development of gout occurs. We are therefore restricted to the first-mentioned views as to the cause of the presence of the uric acid in the blood in gout—viz., that it is due to its being produced at the normal seat or seats of its manufacture, but that it is deficiently excreted. That there is a deficient excretion of uric acid in gout is, I think, proved by the recent accurate estimations of the elimination of uric acid in gouty subjects made by Pfeiffer³ and more recently by myself.⁴

The question next arises whether the uric acid, which in gout is imperfectly excreted, is manufactured in the organs and tissues of the body generally and thence passed into the general circulation, or whether it is produced only in the kidneys, and then, in consequence of imperfect excretion by these organs, the residual quantity of uric acid is absorbed from them into the general circulation. Now if uric acid be produced in the liver or spleen or tissues generally then it follows that it must be conveyed in the blood to the kidneys in order to be excreted, and if such be the case its detection in the blood, if careful search for it be instituted, should be an easy matter. The blood of several healthy individuals has comparatively recently been carefully examined for uric acid by von Jaksch,⁵ by Klemperer,⁶ and by myself,⁷ with negative results in all cases. Sir A. Garrod examined the blood of the ox, sheep, pig, and various birds by the uric acid thread test, but never found a trace of uric acid present, although in all these varieties of blood he always found urea. I have myself examined large quantities of the blood of mammals and birds for uric acid by a new process,⁸ but always with a negative result. On the other hand I always found urea present in all the varieties of blood. This mass of evidence shows that uric acid is never present in the healthy blood of mammals and birds, although the urinary excretion of the latter is almost entirely composed of a compound of uric acid. Such facts inevitably force us to the conclusion that uric acid is normally produced in the kidneys only.

I therefore consider that the first step in the pathogenesis of gout is a failure on the part of the kidneys—from functional or organic mischief—to perfectly excrete the uric acid formed in them and that consequently absorption of the non-excreted portion takes place from them into the general circulation, where it circulates throughout the system at first in the form of sodium quadriurate, and so forms the source from which the gouty deposit is derived. This view is supported by the following facts: (1) in all cases of kidney disease (not associated with gout) in which the blood has been examined for uric acid that body has been found present, showing that when the excretory function of the kidneys is interfered with absorption of uric acid into the general circulation occurs; (2) uratic deposits in the joints are frequently found at the post-mortem examinations of subjects of kidney disease who have never been known to suffer from ostensible gout during life; and (3) kidney mischief is frequently met with at the post-mortem examinations of gouty subjects.

As to the actual formation of uric acid in the kidneys I believe that it is formed in those organs by the combination of urea with glycocholic acid or with one of the derivatives of the latter body. This explains why disorders of the liver constitute so important a factor in the gouty process, since in that organ the antecedents of uric acid are either manufactured or elaborated. The uric acid which is formed in the kidneys is at once converted into the quadriurates of

ammonium, sodium, and potassium, which, if excreted, constitute the amorphous urates of the urine. If, however, any absorption of them takes place into the blood they are entirely converted into the sodium quadriurate, which at first constitutes the sole uric acid compound circulating in the blood. This compound is, however, unstable and after a variable period of time it unites with some of the sodium carbonate of the blood to form sodium biurate. This sodium biurate is much less soluble in the blood than the quadriurate and it therefore, when the blood becomes oversaturated with it, deposits in these tissues which, either on account of having received previous slight injuries or because of their poor vascular supply, specially favour its deposition. Such tissues are structures belonging to the connective-tissue class—cartilages, ligaments, tendons, and the cutaneous and subcutaneous connective-tissues. Although the view has been held for some time past by various writers on gout that uratic deposition is dependent upon a diminution of the alkalinity of the blood, yet I hold the very strong opinion that such deposition is not in any way affected by variations in the alkalinity of that medium. The view that uratic deposition is caused by diminished alkalinity of the blood while increased alkalinity of the blood causes a re-solution of the deposit is, in my opinion, absolutely erroneous and untenable. My reasons for holding this opinion are, briefly stated, as follows: (1) recent researches show that the alkalinity of the blood of gout is very little if at all diminished and that corresponding variations in the alkalinity are frequently met with in healthy individuals; (2) both these views are based on the erroneous supposition that the deposit is uric acid, whereas it is sodium biurate, the solubility of which is not increased by increased alkalinity of the blood; and (3) by a series of experiments described in the Goulstonian Lectures⁹ for 1897 I showed that a diminution in the alkalinity of blood-serum does not hasten or facilitate the deposition of sodium biurate from that medium and does not affect the solvent power of the medium for the biurate. I also consider that the assumption that the gouty properties of certain wines and beers are due to the acid contained in them is erroneous. The acidity of such beverages is mainly due to organic acids, which I have shown to be incapable of facilitating the deposition of sodium biurate. I think it is probable that the gout-inducing properties of such wines and beers are due to the effect they exercise on the metabolism of the liver by increasing the amount of glycocholic acid passed to the kidneys and so causing an increased production of uric acid in those organs. From the results of a series of experiments, which are not yet completed, I incline to the view that the ethereal compounds contained in wines and beers are mainly responsible for the altered metabolism produced in the liver by these beverages. Certainly the effect is not due either to the alcohol or to the sugar contained in them.

THE TREATMENT OF GOUT.

In the first place it should be borne in mind that no routine treatment can be adopted which is suitable to all cases. The nutritional condition of the patient, his habits, surroundings, and mode of life constitute factors that must necessarily modify the treatment of individual cases. The treatment of gout should have for its aims the following objects: (1) the treatment of the gouty paroxysm in cases of acute gout and the relief of the pain as speedily as possible; (2) the treatment of the subacute or chronic condition and the prevention of the recurrence of an attack, which may be effected by the promotion of the elimination of uric acid, by checking any excessive formation of uric acid that occurs in some subjects, and by careful attention to diet and general hygiene; and (3) treatment of the affected joint or joints, with the object of removing the uratic deposit and of preventing permanent deformity. It is useful before commencing treatment, and from time to time during treatment, to know the amount of uric acid that is being daily eliminated in proportion to the body-weight of the patient. This determination of the amount of uric acid eliminated must be made on a sample of the mixed urines of twenty-four hours. The process that I always employ for such determinations is the Gowland-Hopkins process which is a very accurate method for the estimation of uric acid in urine.

TREATMENT OF THE GOUTY PAROXYSM.

The limb should be placed in the horizontal position, or slightly elevated above the level of the body and a cradle

³ Berliner Klinische Wochenschrift, 1892, p. 418.

⁴ THE LANCET, March 27th, 1897, p. 860.

⁵ Deutsche Medicinische Wochenschrift, 1890, Band xxxiii., p. 741.

⁶ Ibid., 1895, Band xxi., p. 655.

⁷ THE LANCET, March 27th, 1897, p. 863.

⁸ THE LANCET, March 27th, 1897, p. 861.

arranged to take the weight of the bedclothes off the affected part. To alleviate the severe pain felt in the affected joint warm packs should be arranged round it consisting of cotton-wool saturated with a soothing lotion and then lightly covered with oil-silk. I have found the following lotion most useful in relieving the local pain: three drachms of carbonate of sodium, two ounces of liniment of belladonna, one ounce of tincture of opium, with water to eight ounces. A small portion of the lotion should be mixed with an equal quantity of hot water and poured on cotton-wool previously arranged round the joint. For the internal treatment of acute gout colchicum is one of the most valuable drugs that we possess. It should be especially used for acute gout and for subacute attacks supervening on chronic gout. If used continuously tolerance is apt to be acquired and then the drug ceases to act. At the commencement a large dose of from thirty to forty minims of colchicum wine should be given, followed by a mixture containing from ten to twenty minims of the wine with from forty to sixty grains of citrate of potassium, which should be administered three times a day. Colchicum reduces the gouty inflammation, relieves the pain, and shortens the attack. From four to five grains of blue pill should be given the first night, followed by a dose of Epsom salts in the morning. In my opinion it is advisable in the treatment of gouty patients to avoid the use of saline purgatives owing their efficacy to salts of sodium on account of the undoubted power possessed by all sodium salts of diminishing the solubility of sodium biurate. For the first day or two of an attack of acute gout the patient should be restricted to a milk diet.

TREATMENT OF SUB-ACUTE OR CHRONIC GOUT.

Promotion of the elimination of uric acid.—This may be effected by medicinal treatment, and by diet and regimen. Citrate of potassium is employed as a diuretic which increases the volume of the urine and at the same time diminishes its acidity. The use of the citrate of potassium may with advantage be pushed until moderate alkalinity of the urine is produced, as by such means the quadriurates are rendered more soluble and more stable than they are in an acid urine and so the tendency to the deposition of uric acid or sodium biurate in the kidney tissues is removed. Free diuresis should also be encouraged by the drinking of sufficient quantities of water. A patient suffering from gout should avoid the use of common salt at table owing to the power it possesses of diminishing the solubility of sodium biurate and thereby both augmenting the precipitation of that body and also interfering with its removal from the system. On account of the results that I have obtained with the mineral constituents of vegetables, to which I shall briefly refer later, I am inclined to suggest that a table salt composed of the ashes of certain of the vegetables should be freely used by gouty subjects in place of common salt.

Means of checking the excessive formation of uric acid.—These consist in careful attention to diet and regimen, in the promotion of the metabolism of the liver, so as to check the excessive production of the antecedents of uric acid, and in the relief of congestion of the portal system, which can be effected by keeping the bowels open at least once a day. In addition to colchicum, which acts as a stimulant of hepatic metabolism, guaiacum may very usefully be administered as an alterative which stimulates the metabolism of the liver and also affords relief to the portal system. From five to ten grains of guaiacum resin should be given in cachets two or three times a day, according to the effect on the bowels, since guaiacum generally acts as a laxative. If constipation occurs a sulphur and guaiacum tablet or a dose of compound liquorice powder should be taken at night. An occasional dose of blue pill followed by a purge of Epsom salts will be found useful.

Diet and general hygiene.—A rational mixed diet is the one best suited for gouty patients, care being taken to avoid excess. The assumption that a purely vegetable diet is best for the gouty is erroneous, since the production of uric acid depends on the ingestion of proteid matter, and it makes no difference whether the proteid matter be of animal or vegetable origin. At the same time it must be borne in mind that since animal food is so much richer in proteids than a vegetable diet the amount of the former taken by the gouty should be strictly limited. Moreover, I find as the result of a lengthened series of investigations that whereas the mineral constituents of meat exercise a marked effect in diminishing the solvency of a gouty deposit the mineral

constituents of most vegetables exercise a marked power in increasing its solvency. The vegetables whose mineral constituents I find are most efficacious in this respect are spinach, Brussels-sprouts, potatoes, cabbage, and French-beans. At the same time it must be borne in mind that with certain patients some of these vegetables may tend to produce some form of dyspepsia and I cannot too strongly urge that in the dieting of the gouty no hard and fast rules can be laid down, but the idiosyncrasy of each patient to various articles of diet must be made the subject of careful observation and study. The following plan gives an indication of the diet to be recommended:—

Morning.—A pint of hot water flavoured with a slice of lemon peel should be slowly sipped immediately on rising.

Breakfast.—A selection may be made from the following articles of diet according to the taste of the patient: porridge and milk, fresh fish, fat bacon, eggs cooked in various ways, dry toast, tea infused for three minutes and then strained from the leaves.

Lunch and dinner.—No soup should be taken at either meal. The varieties of fish most suitable to the gouty are whiting, sole, turbot, and plaice. Meat should be taken at only one meal and then in moderate quantity. Two vegetables should be taken and in abundant quantities. The vegetables that, in my opinion, should be avoided by the gouty are asparagus, tomatoes, and green peas. Stewed fruits or baked apples or pears may with advantage be taken every day at one meal and a milk pudding made with rice, sago, or tapioca at the other meal.

Night.—A pint of hot water flavoured with a slice of lemon peel should be slowly sipped before retiring to bed.

Alcohol.—As regards the employment of alcohol each case must be individually and carefully dealt with. If the gouty person be of robust habit of body then total abstinence is undoubtedly the best for such a patient. If, however, the cardiac action be weak and failing then moderate quantities of alcohol should certainly be given. In cases of chronic gout a moderate amount of alcohol may be necessary for the promotion of digestion. The best form of alcohol for the gouty is a tablespoonful of matured whisky freely diluted with salutaris or with plain water and taken towards the end of lunch or dinner. If any wine is taken by the gouty the one which is least open to objection is a good claret. Ale and stout should be avoided.

Mineral springs.—The use of a mineral water, so far as its employment with the object of removing the gouty deposit is concerned, lies solely in its watery constituent, and does not in any way depend on the mineral salts dissolved in it. As a matter of fact the salts dissolved in a great many of the natural mineral waters are directly harmful in gout both by encouraging deposition of the sodium biurate and by checking solution of the gouty deposits. The flushing of the system of a gouty patient with abundant quantities of water is undoubtedly beneficial, since it dilutes the blood for the time and so tends to prevent uratic precipitation and at the same time promotes diuresis and encourages the elimination of urates. The question, however, naturally arises whether if the water of a mineral water be its only beneficial constituent for effecting removal of the gouty deposit the sending of gouty patients to spas presents any advantages over their drinking ordinary water at home. If the conditions of the life of the patient at home and at a spa were the same there would be no such advantages, but among the special benefits to be derived from residence at a spa must be reckoned the almost undistracted attention that is given by the patient to treatment, the careful dieting that is frequently observed, the change of surroundings, the absence of business or home worries and the opportunities for the use of thermal baths for the external treatment of articular gout. It should, however, be carefully borne in mind that owing to the undoubted fact that sodium salts are directly detrimental to the removal of the gouty deposit those springs should be avoided which owe their activity to those salts where the removal of the deposit is the main object sought for. The springs which contain no sodium salts or traces only are the ones suitable for such cases—such as the waters of Buxton, Bath, and Strathpeffer in this country; in France the waters of Aix-les-Bains, Contrexéville, and Vittel; in Switzerland the Pfäfers water; in Austria the Gastein water and the Sauerling spring at Carlsbad; in Germany the Wildbad water. I wish it to be clearly understood that I am by no means condemning the very proper use that mineral waters containing sodium salts can be put to in the treatment of many gouty affections of the viscera and other structures,

but I wish to emphasise the point that when the system is flushed with a mineral water with the object of dissolving and removing gouty deposits then it is undoubtedly advisable to select a water as free as possible from sodium salts.

TREATMENT OF THE AFFECTED JOINTS.

Careful massage and gentle exercise of the stiffened joints should be resorted to, but only when convalescence is fairly established. The thermal baths of Bath, Buxton, and Aix-les-Bains are useful in the treatment of cases of chronic articular gout and successful results have been obtained by the localised application of very hot dry air which appears not only to relieve the pain and congestion of the joints but also to disperse the gouty deposit. In cases of chronic gout with painful affections of the joints or subcutaneous tissues a mixture containing ten grains of iodide of potassium with from five to ten minims of tincture of iodine is frequently beneficial, but such a mixture is contraindicated if advanced kidney disease be present. After convalescence as much exercise as possible short of fatigue and discomfort should be taken in the open air. Cycling is an excellent form of exercise for the gouty, as the body-weight is borne by the machine, and good muscular movement in the open air is obtained without the gouty joints having to bear the weight of the body.

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IMPRISONED TOOTH: AN OBSCURE CAUSE OF CERVICAL ABSCESS.

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ALVEOLAR abscess, whether in the acute stage or at a later period when a discharging sinus in greater or less proximity to the offending tooth is the source of complaint, although apt to be overlooked by a casual observer rarely gives rise to any real difficulty in diagnosis. The situation of the abscess, the position and the direction of the sinus, the presence of a carious tooth which is more or less painful or tender to pressure, will generally sufficiently indicate the nature of the case. I have met with two examples in which the diagnosis was rendered difficult by the presence of unusual conditions, and although they belong to a class with which dentists and surgeons are by no means unfamiliar they are sufficiently uncommon to be worthy of record.

CASE 1.—A woman, aged twenty-five years, had for several years before she consulted me suffered much from pains in her left lower jaw. In 1891 she had one of the molar teeth extracted and shortly afterwards an abscess formed which discharged itself through the cheek at a point midway between the symphysis and angle. This abscess had been in the habit of closing, refilling, and bursting at varying short intervals ever since. Some months after the extraction of the first tooth all the remaining teeth on that side, including two which were quite sound, were removed, but still no improvement in the recurring abscess was effected. On the occasion of my first seeing the patient—viz., on March 14th, 1893—the sinus was discharging and by means of a probe bare bone was discovered at its distal end. Within the mouth the edentulous gum looked and felt firm and sound, affording no indication of disease within. The patient having been placed under ether I cut down upon the face of the jaw making the sinus the centre of the incision. On exposing the jaw a minute hole was discovered in it which, enlarged by means of a gouge, was found to communicate with a cavity of considerable size and in the cavity, completely surrounded by bone, lay a bicuspid tooth whose crown was partly destroyed by caries and one of the roots of which was thickened and rough. This was removed, the cavity was drained and the wound was sutured. In the course of a few weeks all was soundly healed.

CASE 2.—A man, aged forty-five years, was brought to me on Oct. 9th, 1897, by Dr. Foster, of Shipley, from whom I obtained the following history. Early in July the patient was attacked with pain all over the left side of his face and in the ear; the pain was so severe as to put a stop to work and to destroy sleep and appetite. Towards the end of the month a swelling formed extending from the ear to the chin on the outside and inside obscuring the angle of the jaw, the pillars of the fauces, and the tonsil. An incision into

the internal swelling directed towards the lower jaw evacuated a small quantity of pus and gave a degree of relief. For a week or two pus continued to ooze from the incised wound and then it ceased. Subsequently a swelling formed in the neck and well below the angle of the jaw, and here on Sept. 2nd an abscess burst. This was followed by other points of suppuration, two of which were incised. At the time of my examination a cluster of sinuses disfigured the patient's neck all leading outside the ascending ramus of the jaw in the direction of, but not quite reaching, as far as, the upper alveolus. By outward inspection and examination with a probe I was unable to determine the origin of the suppuration. There was no stiffness of the jaw, the mouth could be well opened, and an internal examination afforded no further clue. All the molars of that side, both upper and lower, had been extracted or had otherwise disappeared some six years before. There was no appearance of any of the wisdom teeth and he was unable to tell me whether or not there ever had been any. Suspecting a concealed tooth I examined both alveolar ridges very closely. That of the upper jaw struck me as being wider, thicker, and more substantial than natural; whilst the lower was sharp, firm, and compact. This observation led me at the operation, which was performed a day or two later under ether, to attack the upper jaw first, which I did somewhat extensively by means of a gouge with the result, however, of extracting only healthy bone. Upon this I was on the point of giving up the search after obtaining Mr. Moynihan's and Dr. Foster's assent to the improbability from its appearance of there being any fault in the lower jaw and trusting solely to the laying open and clearing out of the sinuses; but unwilling to leave any possible source of mischief unexplored and calling to mind the comparative frequency with which difficulty and pain and inflammation attend upon the eruption of the lower wisdom teeth I plunged a gouge into the site of the lower dens sapientiae—viz., the point of junction between the horizontal and ascending rami—and at once I became conscious of having struck the marble-like surface which the crown of a tooth presents as contrasted with the dead, dull, wooden sensation conveyed by impinging upon ordinary bone. By a little manipulation I soon disintombed a full-sized wisdom tooth with just enough caries on its enamel and corrosion of its roots to satisfy me of its having been the originator of all the mischief of the preceding months. After the removal of this tooth the discharge from the sinuses at once ceased and in a few days they had entirely closed.

Difficulties, irregularities, and complications attendant upon teeth eruption are of common occurrence and are usually brought under the observation of the dentist. In both the instances I have related the opinion of the dentist had been sought and in the latter a throat specialist also had been consulted. Both, however, I think, may be claimed as belonging to the realm of surgery rather than to that of dentistry or any other specialty, and both, I venture to think, present points of sufficient interest to make it unnecessary for me to apologise for bringing them before the notice of the readers of THE LANCET.

Leeds.

VITALITY.¹

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X.—THE INTERSTITIAL CIRCULATION IN ALL LIVING THINGS AND ITS CAUSES (*continued*).

It is desirable to consider whether the results of observation and experiment justify us in proceeding yet further in our endeavour to discover some reasonable explanation of the continual flow of fluid holding nutrient and other substances in solution towards and into the very substance of every particle of living matter in nature. This movement of

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