Remarks

ON

THE CAUSES AND TREATMENT OF OEDEMA.

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OEDEMA due to failure of cardiac or of renal compensation, or of the two conjointly, is a common clinical phenomenon the pathology and treatment of which demands renewed consideration in the light of recent investigations on the subject.

In simple cardiac oedema the causal factor is essentially hydrodynamic, its action being mediate. The oedema is not the direct result of back pressure, or, at any rate, only to a limited extent; it is chiefly due to changes in the vascular endothelium caused by the imperfect supply of oxygen and of nutrient material to which the partial stagnation of the blood gives rise. The lack of oxygen and of nutrition impairs the vitality of the vascular walls and makes them unduly permeable; the ensuing transudation is augmented by the low pressure of the blood supply to the kidneys and the coincident retention in the tissues of sodium chloride.

Oedema due to kidney disease is less readily explained. Amongst other causes are changes in the vascular endothelium produced by the toxic action of waste products present in the blood, which, under normal conditions, are removed by the kidneys; the defective elimination of water by the kidneys, and, probably, the occurrence of hydraemic plethora exercise no little influence. During the last few years many suggestive experimental and clinical investigations have been made in relation to the part played by sodium chloride in the development of oedema.

Within the normal tissues a fluctuating percentage of water is retained, the intake and output of which, to some extent, is regulated by the amount of sodium chloride they contain. When much sodium chloride is present, the quantity of water is large; diminution of the salt determines an equivalent dehydration of the tissues. By the administration and the withholding of common salt, Widal and Javal¹ demonstrated in the healthy subject gradations from the physiological hydration of the tissues to those variations which border on the commencement of oedema, and which cannot be exceeded in health, because the kidneys eliminate the excess of salt that is above the normal balance. If an extra amount of common salt is added to the regular diet of a healthy person, an equivalent excess of sodium chloride appears in the urine. It is to be remembered that sodium chloride is almost exclusively eliminated by the kidneys; an insignificant fraction only is discharged with the faeces. In some cases of nephritis there are grounds for assuming that the glomerular epithelium keeps back a considerable amount of sodium chloride over and above the normal balance; if this retention continues day by day, the limit of physiological hydration eventually is exceeded and oedema is the result. Widal and Javal distinguish the early stage of sodium chloride retention as the pre-oedematous stage which by hydration subsequently develops oedema. Halpern² describes three stages of chlorine retention due to renal incompetence: a pre-oedematous stage when apparently no water is retained but only sodium chloride, which is locked up in the tissues; a stage of latent oedema in which, along with the salt, water is retained but not visibly as oedema; a third stage of increased salt retention together with visible oedema. H. Strauss³ assumes that retention of sodium chloride constitutes an indication of the loss of renal compensation and that the chlorine retention precedes and is the cause of the oedema. He further believes that molecules which are free from chlorine do not keep back so much water as those which contain chlorine; so that, in loss of renal compensation, the elimination of sodium chloride and of molecules which contain sodium chloride is interfered with at an earlier period and in a higher degree than that of water and of those metabolic end-products which are free from chlorine.

The kidneys are never absolutely impervious to sodium chloride, and a very decided loss of perviousness is rare; in some cases of Bright's disease in which the elimination of sodium chloride is impaired, considerable fluctuations in the quantity eliminated may occur during the progress of the disease. Koziczowski⁴ believes that sodium chloride

occupies a special position in the elimination of urinary salts, and therefore that it cannot be taken as an indicator for the general saline excretion. He states that in the acute stage of the most severe nephritis the sodium chloride excretion may be absolutely unchanged, and that when it has been much reduced for some time it may be suddenly increased without any other alteration in the symptoms beyond an improvement in the general circulation. When the compensation of the kidneys is lowered or is in abeyance, a normal quantity of urine containing a low percentage of sodium chloride may be excreted; if, under these conditions, an extra amount of common salt be added to the patient's diet the percentage eliminated in the urine remains unaltered, showing that the condition of the kidneys is the cause of the retention. This reasoning, however, is hardly conclusive, as retention due to other than renal causation would have the same effect.

Widal and Javal also believe that the elimination of sodium chloride by the kidneys is a specialized function which may be damaged without interference with their other excretive functions—that of urea, for example; so that, in many cases of chloride retention, the usual relation borne by the urea to the sodium chloride of the urine no longer holds good. Castaigne and Rathery⁵ have demonstrated experimentally that a solution of common salt with a cryoscopic index of -0.78°C , has no injurious action on renal epithelium; but when the solution has a higher or a lower density it determines histological changes in the renal cells. Under physiological conditions, Castaigne and Rathery believe that sodium chloride always passes through the healthy renal epithelium at the same osmotic pressure; and that when the renal epithelium is altered by disease the osmotic pressure becomes higher. In nephritis they found that diet poor in salt has a favourable influence both on the albuminuria and the oedema. Achard and Paisseau,⁶ by intravenous injections of hypotonic and hypertonic solutions of sodium chloride (-0.20° to 1.50°C), set up changes in the renal epithelium similar to those produced by Castaigne and Rathery.

It has been observed that oedema may increase without diminution in the daily volume of urine, or with such diminution as is insufficient to account for the quantity of water retained. An attempt has been made to explain this discrepancy on purely physical grounds. Kövesi and Roth-Schulz suppose that the systemic juices of the patient are pervaded by great excess of sodium chloride; and, as the evaporation of aqueous liquids is retarded in direct proportion to their osmotic tension, that the high molecular concentration of the systemic juices determines a material diminution of the insensible perspiration and of the aqueous exhalation from the lungs which, taken collectively, serve to explain the difficulty. The excessive oedema in the case quoted by Kövesi and Roth-Schulz, which called forth this explanation would not be satisfactorily accounted for, however, on this hypothesis, even had the insensible perspiration and the pulmonary exhalation been entirely arrested. It is a common observation in cases of nephritis with oedema, that if the daily consumption of common salt be materially increased the patient will drink more water than before, often without increasing the volume of urine; when this occurs, the excess of water is added to the oedema.

Some recent experiments by Roth-Schulz and de Körösy⁷ are of interest in relation to the question of sodium-chloride elimination. They find that diffusion through parchment-paper follows the same laws as when no membrane is interposed between the saline solution and water, except that the rate of diffusion is slower. If water is replaced by blood, diffusion is much slower, and it does not follow the same laws, inasmuch as some salts diffuse irregularly. If recent animal membrane be substituted for vegetable parchment, further differences are observed; chlorides and carbonates diffuse from mucous surfaces more readily than other salts. The inference is that, apart from the vital function of cells, the nature of the solutions and the physical properties of the interposed membrane exercise a considerable selective influence in the process of diffusion.

The view that oedema is largely determined by primary sodium-chloride retention with sequential retention of water is traversed by several observers. Marischler⁸ inverts the sequence and regards the retention of salt as being secondary to that of water. Mohr⁹ goes still further, and denies that there is any relation between them; he holds that the retention and the elimination of sodium chloride and of water are respectively independent of each other. Achard and Loeper¹⁰ decline to accept the renal causation of sodium-chloride retention in nephritis, and attribute it to extra-renal

influences such as occur in fevers, pneumonia and other acute diseases. The results of a series of chemical analyses of various organs and tissues of the human body made by Rumpf¹¹ tend to support this view. In some cases of nephritis he found that the amount of sodium chloride in the kidneys exceeded the amount present in the blood and other organs; but although, in many cases of nephritis, the amount of sodium chloride in the blood and the tissues was very high, it was much higher in some other diseases—cirrhosis of the liver, tuberculosis, and heart failure. In some cases of chronic nephritis, notwithstanding the presence of oedema, the amount of sodium chloride in the blood and the various organs was below rather than above the average normal. Rumpf also observed, in chronic nephritis, that there was a diminution in the amount of water in the blood and in the tissues; at a later stage of the disease, however, when the excretion of water is frequently diminished, this condition disappears.

The question has arisen as to whether the retention of sodium chloride is a histo-retention or a sero-retention—that is, whether the salt is held back in the tissues or in the juices. Some investigators have found considerable increase in the amount of sodium chloride present in the organs of patients who have died from nephritis with chloride retention, as compared with the corresponding organs derived from those who have died without any preceding retention (Bohne¹², Achard and Loeper¹³ and others). H. Strauss believes that at the commencement of retention the tissues probably play an important part, but that subsequently they contain less than the juices. It appears probable that much of the salt which is primarily held back by the tissues may, later on, be dissolved out by the transudation fluid. A number of analyses, made by Scheel¹⁴ of various organs from patients with and without retention point to the serum and not the tissues as the chief seat of retention. He considers the retention of water to be primary, and the chloride retention to be the result of an effort of the organism to maintain its osmotic balance.

Although there appears to be a close relation between the retention of sodium chloride and the occurrence of oedema, there are difficulties in the way of unreservedly accepting the relation of cause and effect; the extent to which the kidneys are responsible for the retention also is debatable. In many cases of chronic Bright's disease with general oedema, the patients being on the same mixed diet and taking the same amount of salt, I have found great variations in the daily output of sodium chloride, ranging between 7 and 12.8 grams, and, moreover, there was no constant relation between the extent and course of the oedema and the amount of salt retained. In some cases in which the oedema was increasing the output of sodium chloride was not far below the average; in others the retention of salt and of water was on parallel lines. Whilst hesitating implicitly to accept the theory that retention of sodium chloride is the exclusive cause of renal oedema, I am satisfied that restriction of dietetic salt in the oedema of Bright's disease tends to promote its dispersion. On the other hand, Ferrannini¹⁵ believes that the withdrawal of salt from the diet of nephritics is of no avail in the treatment of oedema; his deductions, however, being based on the results obtained from the subcutaneous injections of physiological salt solution, are open to criticism.

The treatment of oedema has for its object the removal or the modification of the conditions which give rise to it. First, as to simple cardiac oedema. When the heart alone is at fault, physiological rest is the most potent of all remedial agents. Physiological rest comprises absence of all avoidable calls, mental as well as physical, on the cardiac energy, so that it shall be solely devoted to the nutrition of passive tissues and to the transmission of adequate blood-pressure to the secretory organs. In an imperfectly compensated diseased heart, it has been found that the night urine often exceeds the day urine in amount, which is the converse of the healthy ratio. This excess of night urine is attributable to the greater passivity of the patient, in body and mind, during the night as compared with the day. During the night he sleeps, or, if awake, he is probably more tranquil than throughout the day; he is less frequently disturbed by the administration of food, by attention to his various wants; conversation also is more restricted. The result is that the many duties imposed on the enfeebled heart during the day absorb so much of its energy as to reduce the blood-pressure below what is needful for efficient renal activity; so that the work which should be done in the day is postponed until the more restful night. The low blood-pressure not only limits the output of water, but it also determines retention of

sodium chloride, which probably constitutes an important factor in the production of the oedema. The following by no means uncommon case illustrates the effect of physiological rest in the treatment of oedema due to loss of cardiac compensation.

J. A., aged 61 years, was admitted into the Salford Royal Hospital on May 8th, 1903, with extensive oedema all over the body; the surface was cold and cyanosed. The heart was dilated, and the pulse was quick and feeble with intermissions. A faint systolic murmur was heard over the mitral and tricuspid areas. Basic hypostatic congestion existed at the posterior third of both lungs, and the liver projected about a finger's breadth below the costal margin; but there was no ascites. The tongue was thickly coated, and the temperature was sub-normal. For a week or two before admission the patient passed very little urine. During this time he had got out of bed at intervals, and had gone downstairs to sit by the fire. On admission the patient was put to bed, and was given a little stomachic medicine, but no diuretics, cardiac tonics, nor alcoholic stimulants. On the day after admission he passed only 9 oz. of urine. It was high-coloured, clear, acid; specific gravity, 1.024; it was free from albumin; it contained 3.1 per cent. of urea, but only 4.6 grams of sodium chloride. On the second day he passed 31 oz., on the third 60, on the fourth 63, on the fifth 109, on the sixth 163, on the seventh 220, and on the eighth 214. A considerable portion of the accumulated fluid having now come away, the daily amount of urine dropped to from 60 to 70 oz. a day. When the volume of urine was at its highest the urea only reached 0.25 per cent. The percentage of urea slowly increased; on the tenth day it was 0.6 per cent., and on the twelfth day it rose to 1.3 per cent., and subsequently to 2 per cent. The daily amount of sodium chloride increased with the output of urine; when the urine reached 220 oz. the sodium chloride amounted to 22.7 grams. It remained above 18 grams until the twelfth day, when it dropped to 14.5, and then it settled down to about 12 grams; the daily volume of urine being from 45 to 55 oz. After one month's stay in hospital, the patient was discharged free from dropsy and with restored cardiac compensation, due solely to physiological rest.

In most cases of failure of cardiac compensation rest alone is insufficient to restore it. Of medicinal remedies digitalis stands apart from other heart tonics in respect to efficacy. Few medicinal preparations, however, vary so much in activity as do those of digitalis; this is owing to the wide differences in percentage of the active principle yielded by the foxglove leaves in different years, and the time of year when they are gathered, together with the treatment to which they are subsequently submitted. Keller¹⁶ found variations of from 0.26 to 0.62 per cent. of digitoxin in the leaves grown in various years, and he prepared a tincture which was three times richer in digitoxin than a commercial tincture of the same year. Strophanthus is much used as an alternative to digitalis. Fraenkel¹⁷ found that the preparations of strophanthus vary still more widely in activity than those of digitalis—from 100 to 6,000 per cent. My own experience leads me to attach a much higher value to digitalis than to strophanthus; it is not to be denied, however, that much of the ordinary tincture of digitalis is inert, or thereabouts. Amongst the active preparations are Nativelle's granules of digitaline and a form of digitoxin recently discovered by Cloetta, which is soluble in water, to which the name "digalen" has been given.

In some instances the judicious administration of alcohol is of great efficacy in tiding the heart over a critical period; but its effects require close observation. When the kidneys are diseased alcohol is to be avoided unless it be absolutely necessary.

Renal incompetence is usually more difficult to overcome than simple cardiac incompetence. When the kidneys are in fault the efficiency of cardiac tonics is limited by the degree of impairment to which the renal epithelium has succumbed; and the diuretic action of salines, to say the least of it, is uncertain. Fortunately, the resources of renal therapeutics have been materially added to by the introduction of a comparatively new class of diuretics. Diuretics of this type are purin derivatives, being methyl substitution products of xanthin or dioxypurin. They comprise caffeine or trimethylxanthin, theobromine or dimethylxanthin, and its isomer theocine, the methyl group in theobromine being in the 3.7, and in the theocine in the 1.3 positions. Theocine (theophylline) was first obtained from tea leaves, by Kossel, in 1888; but until Traube in 1901 succeeded in producing it synthetically it was too costly for therapeutic use. These closely-allied substances are selective in their therapeutic action. Caffeine stimulates the heart, and has only a limited direct action on the kidneys. Theobromine acts much more powerfully on the renal epithelium; it also stimulates the heart, but not so powerfully as caffeine. Theocine also is a less powerful cardiac stimulant than caffeine, but it is more active as a diuretic than either of the other two. Theobromine is often given as a double salt with sodium salicylate, under

the designation "diuretin," and more recently with sodium acetate, as "agurin." Theocine is also given combined with sodium acetate. Full doses of theocine may cause nausea and even vomiting, therefore the dose should not exceed 6 or 7 gr. All these diuretics, but especially theocine, increase the elimination of the solid urinary constituents as well as of water. Dreser¹⁸ compared the depression of the freezing-point of urine with its electrical conductivity, and found, after the administration of theocine, that the aggregate number of molecules, especially of electrolytes (salts) is increased as well as the volume of water. On the other hand, when diuresis is solely due to an increased intake of water, the excretion of non-electrolytes, such as urea, predominates over the electrolytes. This special activity, by which the excretion of salts is promoted, places theocine amongst the most efficient of diuretics in cases of oedema with retention of sodium chloride. The diuresis produced by these xanthin derivatives is frequently delayed for twenty-four or more hours, and sometimes, when the renal epithelium is profoundly disorganized, it fails to occur at all. It is often necessary to give digitalis along with theobromine and with theocine in order to develop diuresis.

When the incompetent kidneys fail to respond to the stimulus of diuretics, the skin affords an alternative route for the elimination of water and also of the solid urinary constituents. Kövesi and Roth-Schulz found that the dissolved substances in sweat, under ordinary conditions, cause it to have about the same cryoscopic index as blood (-0.4° — -0.6° C.); but not infrequently a much higher molecular concentration, equal to more than 1° C., is met with. Sweat free from albumin yields from 0.1 to 0.45 per cent. of nitrogen and from 0.09 to 0.56 per cent. of sodium chloride, so that an amount equal to about half of the solid constituents of the urine may be removed by the sweat, with a corresponding reduction in the molecular concentration of the blood. Thus the hot-air bath and the vapour bath place at our disposal a very efficient auxiliary means of reducing oedema and at the same time of promoting the elimination of nitrogen and of salts, especially of sodium chlorides. During the administration of the bath the patient should not be allowed to drink copiously of water, as is frequently prescribed with the object of promoting the perspiration; the water thus drunk is added to that which is already retained, and therefore in proportion to its amount it neutralizes the remedial action of the bath.

Cathartics, often of great service in the treatment of Bright's disease, avail little in the dispersion of oedema.

When the kidneys are in fault, the diet in cases of oedema is of great importance. The indications are—to avoid excess of proteids and of such dietetic substances as are likely to irritate the renal epithelium, or to increase the retention of water, and at the same time to afford a sufficiency of nutrient material for the systemic requirements. Milk is almost universally held to occupy the first place in the dietary of nephritis, and of all foods milk is the least harmful to the kidneys; its exclusive use, however, is not without objection. A large quantity of milk needs to be drunk daily in order to furnish the requisite nutrient material; this keeps the vascular system unnaturally replete, and consequently the heart is liable to become dilated. It is quite true that in some conditions—in diabetes, for example—the heart does not suffer from the imbibition of large excesses of liquids; but in these conditions the kidneys are active—even super-active; they speedily remove the redundant liquid, whereas in kidney disease with dropsy diuresis is tardy and restricted. Again, the imbibition of much liquid tends to increase the amount of water already retained in the system. Except in acute nephritis, when the advantages of a milk diet outweigh the disadvantages, an exclusive milk diet is not to be recommended.

In the oedema of chronic Bright's disease, the balance between the dietetic indications is best adjusted by allowing the patient to take a moderate amount of solid food, special attention being paid to the quantity of proteids and of sodium chloride contained by it. A diet rich in proteids is to be avoided, and any lack in calories must be made good by carbohydrates and fat. It is also advisable to eliminate from the dietary those food stuffs which contain a high percentage of extractives; purins, creatinin, and other bodies classed as extractives, are irritating to the renal epithelium. For example, according to the analyses of Walker Hall,¹⁹ the percentages of total nitrogen and extractive nitrogen of beefsteak are both high; whilst in codfish, although the total nitrogen is equally high, the extractive nitrogen is low. In

mutton, the percentages of total nitrogen and of extractive nitrogen are both considerably lower than in beefsteak; in chicken, the percentage is slightly above that of mutton. Glandular organs such as liver, kidney, and sweetbread, which are rich in purins, should be forbidden; also all ragouts and "made dishes." As regards total nitrogen, the differences between fish, flesh, and fowl are not great, and they may be equalized by carefully restricting the intake of such kinds as are of high percentage. Proteids may be replaced by milk puddings and other farinaceous dishes, cream, butter, bread, potatoes, and sweets, together with eggs, and weak tea and coffee. If soups are allowed, they should be simple and home-made, without salt, or with but a sprinkling; concentrated meat extracts, which abound in creatinin, should not be permitted. As the eliminative activity of diseased kidneys varies considerably within short periods, it is well to estimate the twenty-four hours' excretion of nitrogen from time to time, and, if defective, to further restrict the allowance of nitrogenous food, which should not exceed from two to three ounces daily.

The quantity of liquid allowed to dropsical patients with Bright's disease should be determined by careful consideration of the conditions involved. The plan frequently adopted of causing the patient to drink copiously of liquids in order to promote the elimination of end-products is fallacious on two grounds: First, the kidneys are incompetent to excrete urine of normal molecular density, consequently it is poor in solid constituents; secondly, the fact that oedema is present indicates a tendency to retention of water which lays under contribution the whole of the liquid imbibed, leaving but a small proportion of the excess to act as the vehicle for carrying away effete products in the urine, so that the oedema is increased in place of the tissues being flushed out. On the other hand, undue restriction of liquids impedes the output of a certain proportion of solid constituents which the kidneys are still competent to eliminate, provided that the volume of urine be not arbitrarily checked. When the volume of urine is ample, the intake of liquid usually needs no restriction; with scanty urine, the intake should be adjusted by comparing daily the intake with the weight of the patient; increase in weight would suggest restriction of intake. It is better to aid diuresis by a sufficiency of water than to attempt to induce it by copious imbibition.

When it is deemed desirable to reduce the ingestion of salt, the dietary for a time should be limited to substances which are poor in salt, such as milk, cream, unsalted (or washed) butter, eggs, bread, potatoes mashed with cream, rice, a couple of ounces of beef or of breast of fowl, and weak tea or coffee. In the first instance, no added salt should be allowed; a limited amount may be permitted subsequently, when the oedema is subsiding. If the oedema is slight, it will be sufficient to limit the quantity of salt to one-half of what would be taken under ordinary conditions. In this way it is easy to reduce the daily excretion of sodium chloride in the urine from 14 or 15 grams to 6 or 8 grams; by further limitation of intake it may be brought down to 3 or 4 grams. Most patients with Bright's disease can excrete as much sodium chloride as will yield 0.5 per cent. in the urine; on this datum Kövesi and Roth-Schulz recommend that the allowance of salt should not exceed $\frac{1}{2}$ gram for each 100 c.cm. of urine, so that with an output of 400z. of urine, the daily allowance of salt would be about $1\frac{1}{2}$ dr. The object to be fulfilled is to maintain the normal balance of sodium chloride without trusting to the eliminative capacity of the kidneys to remove an inordinate excess.

The progress of the oedema is ascertained by periodically weighing the patient, whose weight diminishes as the oedema subsides. One example of the effect of a limited intake of table salt will suffice.

C., male, aged 23 years, who had been several months in hospital suffering from chronic parenchymatous nephritis with progressively-increasing oedema, was put on a diet which contained about 8 grams of salt daily, with the following result:

				Weight.
January	16th	...	...	208 lbs.
"	27th	...	...	201½ "
February	3rd	...	...	194 "
"	10th	...	...	189½ "
"	17th	...	...	187½ "
"	24th	...	...	183½ "

During this period the daily excretion of sodium chloride ranged from 7 to 13 grams. The urea kept close upon 1.1 per cent. From 0.7 to 2 per cent. of albumin was present. Part of the time the patient took 6 gr. of theocine twice a day;

subsequently he took 8 gr. of theobromine twice daily. Whilst taking the diuretics the patient passed from 60 to 80 oz. of urine in the twenty-four hours; previously it was below 50 oz. The oedema visibly diminished, and the condition of the patient improved.

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ON THE TREATMENT OF PURULENT CAVITIES.*

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To judge by recent and current medical literature some confusion of thought exists concerning the principles underlying the treatment of purulent collections. One result of this confusion is that methods of treatment found suitable in particular cases are presumed to be applicable universally. Thus in every textbook I have seen, whether on general or departmental surgery, it is considered that the principle of drainage, so successful in dealing with soft-tissue abscesses, is a prime factor in the cure of chronic purulent collections inside fixed walls.

I shall attempt, among other things, to show that simple drainage is of small value in such cases.

In every soft-tissue abscess the tissues which surround the abscess have been by its growth distorted from their natural position, to which place they always tend to revert. The force of this reversion, or, what is the same thing, the pressure of the pus, is determined as to its degree by the following three factors: First, the rapidity of the growth of the abscess; secondly, its situation; and, thirdly, its size.

As regards the first, a rapidly-growing collection pushes the tissues more rudely aside, it gives them less time to adapt themselves to its presence; their natural elasticity is therefore less impaired than in a case of a slowly-growing one.

Secondly, the situation in which the abscess grows. If the surrounding tissues are soft and lax, the tendency to revert will be feebler and the pus pressure correspondingly lower than when the tissues are denser and tougher. By surrounding tissues is not necessarily meant only those directly bordering on the abscess, but all structures which tend to be distorted by its presence. Fibrous-tissue planes, though remote from the wall, may help it enormously in its reversion.

When we come to consider the influence of the mere size of the abscess on its pressure, we find the question is not quite so simple as it might first appear. It might be thought, since the bigger an abscess grows the more its surroundings are distorted, that the pressure inside would in all cases increase proportionately, but there is an important factor which interferes with this. In cases where we may regard the force of reversion, or pus pressure, as being due to tension in the sac wall, the pressure varies, according to a well-known physical law, not only as the tension per unit of length but also inversely as the radius of curvature.

$$P = \frac{T}{R}$$

Therefore if the sac wall tension remains the same, the larger the sac, the lower will be the pressure inside and conversely. Now in a growing abscess the tissue fibres at the sac wall are always, except at the start, on the point of giving way and are in other words at their maximum tension. Therefore I think we may say that the tension per unit of length of the sac wall is constant or nearly so, for a given abscess, no matter what its size so long as it is confined to the same tissue.

This law of the pressure varying inversely as the radius only applies to cases where the pressure may be regarded as being due to tension in the sac wall, and where that tension is constant. In the other class—namely, where the pus pressure

is due to influences remote from the sac, it is by no means true.

If for simplicity's sake we suppose a small abscess situated in the middle of a much larger and not very resistant structure, which is in turn confined by a tough capsule, a relatively large increase in the abscess will only cause a relatively small stretching of the remote capsule, which will not be distorted beyond its limit of elasticity; its tension will be increased, and the pressure internal to it, and therefore the pus pressure increased. Hence we arrive at the interesting result that increase in the size of an abscess causes a fall of pressure if the resistance to its growth is immediately outside the sac, while if this resistance is some distance from the sac it results in a rise.

This is really only another way of saying that as long as the tissues, whether near or remote, to whose tension the abscess pressure is due, are not distorted beyond their limit of elasticity, increase in the size of an abscess is followed by a rise in its pressure, while if that limit be passed it results in a fall.

There is one possible case in which this is untrue, and though it does not occur in the human body, it may be mentioned for sake of completeness. The case would arise if the tissues were so elastic that the increase of tension due to stretching was more than counterbalanced by the increase in the radius of curvature.

It need hardly be said that though both these varieties exist in their pure form, yet most abscesses consist of a combination of both, in which one or another element predominates.

From the foregoing it only needs a little consideration to show that if the cavity is large enough and the surrounding tissues lax enough, or if the abscess is sufficiently slow in its growth, the tendency after incision to spontaneous evacuation of its contents may be so feeble as to be unable to overcome the effect of gravity if the opening is not near the lowest point; while if the abscess is moderate in size and the surrounding tissues firm, the influence of gravity, especially in acute cases, may in turn be quite negligible.

It is in the former class of case that drainage from, or close to, the most dependent point is so important; for it is clear that the abscess will never heal if the head of the pus between the drainage opening and the lowest point of the sac is sufficient either to force the fluid further through the tissues, or to prevent the contractility of the tissues from extruding the matter. Examples of this are to be found in every part of the body—abscess beneath the scalp will occur to everyone. A chronic abscess burrowing through the thigh, especially when the wasting of the muscles from inaction has relaxed the deep fascia, is also a case in point; but perhaps the most common region is in and around the abdomen where the general looseness of the tissues favours its occurrence.

As an example of the latter class—that is, where the influence of gravity may be disregarded, a tonsillar abscess may be cited. The most convenient place for puncture is in the soft palate, and this is rarely the lowest point, yet the healing is never retarded on this account, for the abscess is generally acute, the quantity of pus is never very great, and the surrounding tissues are never very lax.

If the conditions are favourable even large abscesses may empty themselves in spite of the influence of gravity. I have seen two cases of deep-seated abscess at the root of the neck, which from the necessities of the cases had to be opened in front of the trachea low down, at a place which in the upright position must have been quite the highest point, and even in the recumbent position could not have been very far from it. From each of these a quantity of pus escaped on incision, which showed it must have come from deep within the chest, and yet in spite not only of the influence of gravity, but the perhaps more important negative pressure of the thorax, healing took place rapidly. In these cases the abscesses were situated in the connective tissue, and the extrusion of the contents was not so much the result of the contractility of the walls as of the reversion of the deep fascial places.

This negative pressure of the thorax leads us to consider the part it plays in the empyema of the pleural cavity.

The case of the pleura differs from any we have yet considered in that the outer concave wall is fixed or nearly so. Healing can only take place through obliteration of the cavity by union of the parietal and visceral walls. Now, this could only be accomplished rapidly by expansion of the lung in opposition to the negative pressure, a thing which the lung has no inducement whatever to do since the negative pressure of the thorax is in fact due to the contractility of the lung. So

* Read before the Surgical Section of the Royal Academy of Medicine, Ireland, on March 24th, 1905.

† For the case of a cavity having a single curve. If the wall is curved in two directions the formula is $P = T \left(\frac{1}{R} + \frac{1}{R_1} \right)$ where R and R₁ are the radii of curvature.