feeling instinctively that nothing could be more terrible than the existence of a large hole in a vital organ. And yet a moment's consideration would show him that the dull tympanic note, the absence of vibration in the chest walls, their sinking in, the pectoriloquy, etc., all point to a hard-walled cavity due to a reconstructive action following the destructive process. The formation of this fibrous tissue has shut off the part destroyed from that of the healthy lung; a reparation and cure have been going on, and produced those marked physical signs which he has regarded as indicative of disease. No wonder, therefore, that a mistake in prognosis has been made, and that after condemning a patient who has manifested such physical signs the medical man has been surprised to find him still living years afterwards. I have, indeed, not only seen the mistake made by young and inexperienced men, but by older members of the profession: they have often confessed their surprise on seeing phthisical patients still alive whom they had condemned years before. This case of phthisis is perhaps the commonest example, where the constructive and destructive processes have not been sufficiently distinguished, but we will now take another case where the changes are more complex and where a greater difficulty exists in distinguishing those which may be regarded as destructive and those which are reparative. I refer to cirrhosis of the liver. This organ may be observed during life to be first enlarged and then become nodulated and shrunken; amongst other signs of the disease which the textbooks describe is an enlargement of the veins on the surface of the abdomen. These were thought at one time to be due to some pressure on the vena cava, but this is really not the case, and the appearance is now shown to be due to an enlargement of the epigastric vein in connection with the opening up of the old umbilical vein, which often becomes as large as a goose quill. Through this the portal blood flows and finds a free passage into the vena cava and systemic circulation. Injections may be easily made to pass from one system into the other. The patient in this way is relieved from an engorgement of the portal system and from the consequent dropsy. This enlargement of the veins, therefore, which is simply described as a sign of cirrhosis, should also be spoken of as a sign of a reparative character. And probably this is not all, for when the cirrhotic organ is examined carefully and microscopically, we find that, although the nodules are composed mainly of atrophied hepatic tissue, there has also been an attempt to replace it by a new formation of cells and vessels. New blood vessels may be seen and apparently new ducts, as well as cell growth. Now although these new elements are not perfect enough to take on the function of the liver, there can be little doubt that they do relieve the circulation in a disabled organ. That new hepatic structure may grow under the strain of necessity is seen in those cases where portions of the liver have been destroyed by injury or disease in the same way that one kidney will double its size when its fellow from any cause has become atrophied; illustrating the doctrine of Aristotle that function precedes organisation.

Many other illustrations might be mentioned where a truly morbid process occurs intermingled with the reparative. A common example of this may frequently be found in scirrhus pylorus, where a hard lump is felt in the right hypochondrium. This is composed not only of cancer but of hypertrophied muscular and areolar tissue, and sometimes, if the primary affection has been the cicatrization of a chronic ulcer, the whole tumour is composed of a conservative or reparative material.

In diseases of the spinal cord it is very difficult to place the truly morbid and conservative processes in their right category. I have little doubt that in a case, for example, where a man for 20 years has had symptoms of locomotor ataxy, the sclerosis from which he is said to suffer is the result of a more active pathological change which occurred at the commencement of his malady. At that time the new tissue took the place of the old, and the cord has remained quiescent ever since. As such a patient is able to follow his sedentary occupation and has a fair enjoyment of health, he can scarcely be regarded as the subject of disease, or no more so than a person who has a cicatrix on the skin arising from an old injury, although the tissue may differ from the original healthy one.

If time had allowed me to carry the subject into clinical medicine I could have shown the importance of endeavouring to distinguish physiological processes from the purely pathological ones, such as the salutary hæmorrhage from the lungs in heart disease or the serviceable hæmatemesis and melæna in cirrhosis of the liver; we might also see how diarrhœa is a real safeguard in nephritis, or a profuse sweating in bronchitis. The mode in which organs and functions accommodate themselves to one another or supplement one another requires our most careful study. That vicarious action must go on to a greater extent than we imagine is seen in the fact that when we discover after death the kidneys to be scarcely a quarter of their normal size, we express our surprise at the absence of symptoms indicative of so great a destruction; and the same when we find a marked cirrhosis of the liver where the disease has never been suspected. A study of these relations of organs ought to lead us to more rational modes of treatment, and prevent us from merely making an attack upon symptoms whilst in ignorance of their true significance.

I have now, I trust, said sufficient to make my opening statement clear, that, as a teacher of pathological anatomy, I have not paid sufficient regard to the distinction between constructive and destructive processes; that whilst there have been decay and degeneration there have been also growth and regeneration. To study these for the sake of discovering the several influences exerted in the production of each is a matter of great practical import; they also show, as I have already affirmed, that pathology is governed by the same laws as those which exist in every other department of Nature, and therefore may safely take its place among the other sciences.

SOME CASES IN WHICH ARTHRITIS AND ENDOCARDITIS WERE PRODUCED BY DRUGS WHICH DIMINISH THE SOLVENT POWERS OF THE BLOOD FOR URIC ACID.

By ALEXANDER HAIG, M.D.Oxon., F.R.C.P.,

Physician to the Metropolitan Hospital, and the Royal Hospital for Children and Women.

For how many years was the evidence which now seems so obvious of the earth's motion or the blood's circulation overlooked because the facts were regarded through the mist of preconceived ideas and partial ignorance? May not our failure to find the cause of acute rheumatism be due to similar difficulties, our preconceived ideas and our ignorance of the chemistry of uric acid combining to prevent our seeing the real relation between cause and effect?

For years we have been observing that people get their first attack of rheumatism or a relapse later on, under several fairly definite conditions, such as exposure to cold after heat and fatigue, the sudden onset of a febrile disease, or some change in diet, or the administration of some acid or acid-forming food.

But what we did not know was that one and all of these causes affected in a most important manner the solubility of uric acid in the blood, preventing the blood from continuing to hold it in solution, and so bringing about its precipitation or concentration upon the fibrous tissues of the joints, the fasciæ, or the heart; with the result that it set up more or less marked irritation in these structures, and produced the phenomena of acute rheumatism.

We did not know further that a very large number of drugs, metals, salts, acids, and other things which directly or indirectly interfere with the solubility of uric acid in the blood, produce precisely the same results as the above-mentioned causes, and precipitate more or less irritation of fibrous tissues by uric acid.

Further, we did not know that every drug which we had empirically used in the treatment of acute rheumatism, alkalies, colchicum, hot baths and blankets, or salicylates, had this one effect common to them all—that they increased the solubility of uric acid in the blood, and so led to its being taken up from the fibrous tissues and excreted in the urine,

with the result that the irritation in the fibrous tissues quickly begins to subside.

So long as we persist in believing that rheumatism is due to some unknown cause, miasm or microbe, we shall continue to say that the patient had an attack or a relapse with endocarditis, which resulted in serious heart lesion and a crippled life, and shall regard these things as inevitable, much as do the jury who bring in a verdict of "Death by the visitation of God."

But once we realise that these diseases depend solely upon the quantity and solubility of uric acid in the blood, and that, as I have further pointed out, the quantity of uric acid in the blood and its solubility in that fluid are absolutely and completely within our control, we shall pass at one bound from the unknown to the well known, shall see that these deadly diseases are not the result of unpreventable causes, but of our own dietetic follies; and that our children need not be crippled or decimated by them, if we allow them to live according to their own inclinations on milk and garden produce, and abstain from forcing down unwilling throats the stimulating but deadly products of animal metabolism.

For some years past I have been in the habit of asking all rheumatic children I have seen this question: If I offered you a plate of fruit and a plate of meat, which would you take? I have never seen a child that would have taken the meat.

Before giving you my own cases I will just refer to the well known cases of Sir W. Foster, who, with the object of diminishing the amount of sugar, administered lactic acid to two patients suffering from diabetes, with the result of producing severe arthritis resembling acute rheumatism, and in one of the cases six attacks were produced by repeated administration of the acid.

I have every reason to believe that any acid that is absorbed and affects the alkalinity of the blood will produce similar effects if it is given at a time when there is an excess of uric acid in the blood,¹ and the only point about diabetic cases is that they very frequently have an excess of uric acid in their blood as I have elsewhere pointed out.² Not only acid but any drug (and there are many of them) that interferes with the solubility of uric acid in the blood will do the same thing.

Dr. Lauder Brunton speaks³ of cases in which both rheumatism and endocarditis have been produced by injecting acids into the blood, and he also mentions an instance in which drinking the red wine of Southern Europe appeared to keep up a lumbago from which a man was suffering, and that it had often acted similarly in other cases. With regard to endocarditis, he refers to a thesis for the degree of M.D. in the University of Edinburgh presented by Dr. J. A. MacDougall in 1865, and says that he was able, by injecting phosphoric acid into the blood "to produce undoubted evidence of endocarditis."

Coming a few years after these remarks of Dr. Brunton's were made, the following case startled me very greatly and made me fear that I had inadvertently caused both rheumatism and endocarditis, and I shall leave my readers to judge for themselves whether this was so.

CASE I.—M. J., aged 30, was suffering from phthisis of the left apex, with some tuberculous troubles also at the left base. In March, 1894, he had some fresh signs of consolidation at the left base, with a temperature of 102°. After a few days in bed the temperature began to come slowly down, and on March 9th, as his appetite was bad and he was a good deal pulled down by night sweats, I gave a mixture containing some nitro-hydrochloric acid and a few minims of liq. strychninæ. On March 10th he complained of pain and tenderness about the left shoulder-joint, increased by movement of the limb; the temperature had risen very decidedly, and on listening over the apex of the heart I was horrified to find a distinct systolic murmur which I had not noticed before. I at once stopped the mixture and gave salicylate of soda in its place, and on March 11th the temperature was lower and the pain in the shoulder had gone; but the systolic murmur remained, and, though I saw him from time to time for six months after this, it never completely disappeared. I have no doubt whatever that I thus caused some arthritis, and I fear also some endocarditis as well.

CASE II.—J. B., aged 7, suffering from chorea, was admitted under my care at the Royal Hospital for Children and Women on October 29th, 1894. The temperature on admission was 99.6°, the pulse 80 to 90. I examined the heart, and, finding no murmurs, put her on a mixture containing mxxv of liq. arsenicalis to be taken three times a day. On October 30th the temperature was nearly normal, on October 31st it rose to

100° in the evening, on November 1st to 100.6°, and November 2nd to 100.4° in the evening. It then kept between 99° and 100°, but on November 6th the pulse had risen to 140 and above, and it remained quick for some time after this; the arsenic was left off. On November 7th the highest temperature was 99.8°, and liquor arsenicalis miiij was given three times a day. On November 8th the highest temperature was 99.8°, on November 9th 98.8°. From this the temperature remained just about 99° until November 21st, when the arsenic was left off. On November 22nd a well marked post-systolic murmur was observed at the apex. From this onward the temperature kept about 99° rising to 100° on December 14th, and 100.6° on December 18th. By this time the chorea, which had improved markedly on the large dose of arsenic given at first, and more slowly afterwards, was quite gone. She was allowed to get up, and on December 27th she went home, the murmur at the apex of the heart remaining well marked to the last.

With regard to the onset of this murmur, I should say that Dr. Huntley, our very able resident medical officer, believed that there was a very slight systolic murmur at the apex from the first; but that it became more marked while under observation. It is quite certain, however, that I heard no murmur on admission, for my rule is not to give arsenic, and certainly not the large dose I gave in this case, if there is any sign of endocarditis.

The fairly marked rise of temperature under the arsenic, and its fall later on when it was reduced and left off, point, I think, to some inflammatory trouble in the body, and there was no evidence of any except in the heart; and then the very marked rise in the pulse rate about November 6th may, I think, be taken as pointing to some fresh heart trouble. I may say that my researches show that arsenic, so long as it does not upset digestion, causes a diminished excretion of uric acid in the urine.

CASE III.—A. S., aged 7, was admitted under my care at the Metropolitan Hospital on February 12th, 1895, suffering from broncho-pneumonia, coming on pretty suddenly with headache and shivering. The temperature on admission was 104°, but it at once began to slope downwards, touching normal on the evening of February 15th. On February 16th it rose slightly above 100° in the afternoon and evening, but up to February 20th it kept between normal and 100°. On admission dyspnoea was the most obvious sign, and percussion was altered over several portions of the left lung, but a few days later there were more obvious signs of consolidation at both bases. There was no murmur over the heart and no increase of dulness. On February 20th, after the temperature had been at 100° or below it for four days, a systolic murmur was noticed at the apex of the heart, and the cardiac dulness was also found to be a little increased to the right. The temperature now continued for some time between 98° and 100°, on February 28th it became subnormal, making, however, daily excursions from 96° to 98°. On March 6th it became more steady and kept nearer the normal, but occasionally ran up to 100°. On March 9th the systolic murmur was still heard at the apex, and reduplication of the second sound at the left base. On March 13th I gave, three times a day, a mixture containing tincture of digitalis miiiss, dilute sulphuric acid miv and some glycerine. I gave this because I was not very certain whether the raised temperature and the apex murmur meant endocarditis or not; and, on the other hand, if the murmur was merely due to dilatation, the digitalis might be of some use. As there was some doubt, however, I told the house-physician to stop the drugs at once if the temperature rose. On March 14th and 15th it rose to 100.3° and 100.2°, on March 16th it rose to 101°, and on March 17th to 101.2°. Accordingly, on March 18th the mixture was left off, and the temperature, though it touched 101° that day, at once began to fall, and did not reach 100° on either the two following days. On March 20th, as the temperature, though lower, was still unsteady, I gave some salicylate of sodium, and the temperature then became more steady, and, with two exceptions, was always below 100° till the end of the month. The systolic murmur remains, and if anything is now more distinct than before, and can be heard outwards in the axilla. The rest of the history is uneventful, the temperature during April and up to his discharge on May 8th kept between 98° and 100°. The systolic murmur continued, and on April 3rd the whole of the first sound was replaced by a murmur.

I have very little doubt that we had here to deal with an endocarditis possibly originating, as I have elsewhere suggested,⁴ in the great strain which falling temperature and its consequent rising blood pressure put upon the valves of the left side of the heart; that this went smouldering on for some time, and accounted for the irregular temperature which followed convalescence, and then blazed up again at once on the acid and digitalis mixture, for the digitalis not only put extra pressure and strain on the valves of the left side, but the sulphuric acid very probably precipitated some fresh urate upon them.

The temperature fell at once when these drugs were left off, but the local irritation went on smouldering as before, though before the patient went out it had been practically normal on several occasions for a few days.

CASE IV.—L. G., aged 45, was admitted into the Metropolitan Hospital on January 22nd, 1891, suffering from gout in the fingers of the right hand. He had gout first in the toes and instep in 1882, and since that in the fingers, toes, ankles, or knees. The pain and swelling in the fingers, which were swollen, tense, and shining, began two days before. Temperature on admission 101°, rose at night to 102°. He was given a mixture containing

¹ BRITISH MEDICAL JOURNAL, 1895, vol. i, p. 640.

² Uric Acid, 2nd edition, Ch. xiv.

³ Proceedings of Royal Medical and Chirurgical Society, April, 1890.

⁴ Practitioner 1891, p. 195.

r. xx of salicylate of sodium four times a day. On January 24th the pain in the fingers had ceased. The temperature fell in the evening to 99.6°. After this he went on well, and the salicylate was left off on February 7th. On February 9th and 10th he suffered pain due to an alveolar abscess. On February 11th the temperature rose at night, and there was some return of the gout in the fingers and in the bursæ of the olecranon process on the left side. On the morning of February 12th the temperature was 100.4°; the stumps were extracted and some salicylate given again, and the temperature soon fell to normal and the pain went.

I have elsewhere⁵ narrated the case of a boy where similar teeth trouble brought about a relapse of acute rheumatism; and I have also seen a case where the fever of an attack of pneumonia produced a relapse of gout in a patient who had had previous attacks.

CASE V.—E. S., aged 36, admitted into the Metropolitan Hospital on December 31st, 1894, suffering from acute rheumatism; had been confined nine weeks before.⁶ The pains began three weeks before admission, first in the head and then in the limbs, shoulders, knees, wrists, and elbows in that order. The joints were all very tender. The temperature was 102°. She was given a mixture containing salicylate of sodium gr. xv and bicarbonate of potassium gr. x, to be taken every two hours. On January 2nd the temperature was 100°, but the joints were still tender. The bicarbonate of potassium was omitted from the mixture. The temperature ran up that night to 102.8° (the highest it had been since admission), and there was a sharp return of all the pains. It fell to 100° the following morning, soon went down to normal, and never rose again.

Now what was the meaning of this sharp and sudden relapse when the drugs were altered? Was it due to the untamable microbe? I believe it is in my power to show that it was simply and solely a matter of the solubility of uric acid, for I have observed the same thing before under similar conditions.

Some years ago I had under my care a patient suffering from subacute Bright's disease whom I had put on admission on a mixture containing citrate of potassium. After she had been on this for several days she got some pain and swelling in several joints, so I stopped the potash salt and put her on salicylate of sodium, and in a few days her pains were very much better, and the salicylate was therefore left off and the potash mixture resumed; here again, just as in the case of E. S., there was a sharp but temporary return of all the joint pains as the result of the change of drugs.

It seems then quite clear that when arthritic pains are being relieved by a salicylate it will make them worse to add potash, and, conversely, when arthritic pains are being relieved by potash, it will do harm to add a salicylate. My own personal experience quite bears this out, for when some years ago I had my uric acid under less perfect and complete control than at present, I used occasionally, after severe exercise in hot weather, to get some pain and stiffness in the fascia either of the neck or the lumbar region; and I often found that these troubles were completely removed by a few doses of salicylate of soda, so long as I kept quiet and cool in the house in the morning; but if in the afternoon I went out and got overheated, still continuing the salicylate, my pains returned, and were worse—perhaps much worse—than if I had taken nothing.

Now what is the effect of going out and getting hot? It has been shown by Sir A. Garrod and myself that it produces a fall in the acidity of the urine, or, to put it more correctly, a fall in the hourly excretion of acid in the urine—say the hourly excretion of acid in any individual is equal to 3 grains of oxalic acid, then going out and getting very hot will, other things being equal, reduce it to 1½ or 2 grains in the following hours; that is to say, getting hot is equivalent to a dose of alkali.

But we have already seen that alkalies and salicylates do not pull well together, and we know that uric acid may be excreted in excess under alkali or under salicylate, and it must be extremely annoying to preconceived opinions that giving the two together does not produce the best effect of all; but unfortunately it does not, as can be proved by experiment.

And further there is, as I have for years been trying to point out,⁷ a chemical explanation of the fact, for it appears that salicyluric acid differs from all other compounds of uric acid in being soluble in more or less acid fluids.

Now it is well known that salicylic acid compounds act best in acute arthritis, and better the more acute the case

and the higher the temperature; and it is a matter of the most simple observation that the higher the temperature the higher the acidity of the urine, the greater the hourly excretion of acid, and the less the alkalinity of the blood; therefore salicylates act well when the alkalinity of the blood is low.

It is further known that in subacute cases with but little rise of temperature salicylates may act far less satisfactorily, and I have pointed out that in such cases their activity may be increased by giving them along with substances which raise the acidity or in alternate doses with an acid; therefore salicylates act badly when the alkalinity of the blood is high or undiminished.

It appears, then, that uric acid in the blood may be in solution either with a salicylate or with (potash or soda) alkali, but it cannot be in solution with both at one and the same time; and that if it is in solution with alkali and a salicylate is added this will diminish the alkalinity, and hinder the solubility of uric acid under alkali; but until the alkalinity has been very greatly reduced the uric acid will not pass into solution as salicyluric acid, that is to say, between the two drugs there is a dead point at which the uric acid is not in combination with either, in fact, it is rendered insoluble, and is driven out of the blood into the fibrous tissues of the joints or fascia, producing a relapse of the gout or rheumatism, and a rise of temperature. If the salicylate is continued the acidity rises still further, and then salicyluric acid is formed and got into solution, the uric acid passes freely in this form from the joints into the blood, the temperature falls, and the pains subside once more.

Now, what occurred in the case of E. S. was that she was being given a considerable amount of potash along with some salicylate and her urates were got into solution with potash and little or at all with salicylate; but when the potash was left off the alkalinity fell, the potash lost its hold over the urates before the salicylate gained it, and the urates were for a time driven out of solution into the joints and fibrous tissues, producing a sudden relapse of all her pains, and a temperature of 102.8; later on, the salicylates had it all their own way, and there was no further joint irritation or rise of temperature. This shows, I think, that salicylates control the pain and temperature of arthritis simply by eliminating uric acid; if anything prevents their eliminating uric acid the pains promptly return, and the temperature rises in spite of them.

On the other hand, I have seen salicylates given in various local and traumatic inflammations and in pyæmia, and their effect on the pain and temperature was just nothing, because these were not due to uric acid. Further, I have pointed out⁸ that the compounds of salicin and salicylic acid are powerful in the relief of acute rheumatism exactly in proportion to their power over the solubility and excretion of uric acid; and that those that have least power of elimination (salicin) must be given in much larger doses than those (salicylic acid) that have most. I make bold to say that every drug that has ever been used with benefit in acute rheumatism in the past, and every drug still to be discovered that may be used with benefit in the future will be found to do good in direct proportion to its power of dissolving and eliminating uric acid. But I believe most strongly that, once these simple facts about the causation of rheumatism are generally recognised, prevention will be so simple and so certain that it will soon become quite a rare disease.

I shall now, in conclusion, shortly mention one or two cases in which a relapse of chronic rheumatism was produced by drugs given for other purposes.

CASE VI.—T. P., aged 71, was admitted into the Metropolitan Hospital on January 14th, 1895, suffering from chronic rheumatism (? gout). He had had an attack of sciatica ten years earlier, and, two years later, arthritis in both hands. This he had had off and on ever since, chiefly in the legs, but the hands were a good deal distorted and crippled. He was ordered salicylate of sodium and given some cod-liver oil. He improved steadily, and was soon able to walk much better, and had little or no pain. The temperature varied from 97° to 99°. On February 5th he had some little bronchitis, the salicylate was stopped, and he was given a mixture containing ammonia and senega. As the pain returned the ammonia mixture was stopped and the salicylate resumed on February 9th. On February 13th the pains were distinctly better, but the salicylate was kept on, and, as the previous dose was small, it was increased to gr. xv, and the improvement was steady and continuous. We have here only

⁵ *Uric Acid*, second edition, p. 334; and *Wood's Medical and Surgical Monographs*, 1890, p. 355.

⁶ Parturition generally brings an excess of uric acid through the blood, and some of this was eventually driven into the joints.

⁷ *Medico-Chirurgical Transactions*, vol. lxxi, p. 134.

⁸ *Medico-Chirurgical Transactions*, vol. lxxiii, p. 297.

the patient's word for it, as the temperature showed nothing, but there was no reason why he should have complained of more pain just at the time the drugs were altered.

CASE VII.—H. K., aged 68. This case, very similar to the above, was suffering from chronic rheumatism and sciatica. He was admitted into the Metropolitan Hospital on January 15th, 1895. There was a history of having suffered for nine years. Pains were now chiefly in left knee and hip. He also was put on salicylate, and improved considerably, but he also later on got bronchitis, and on March 20th was given the same ammonia mixture as in the other case. On February 23rd the note says: "Can get about better and pain is, comparatively speaking, gone." On March 20th he got some bronchitis. The salicylate was stopped, and mixt. senegæ cum ammonia ordered. On March 26th the pains were decidedly worse again, especially in the knee. He was ordered to have the salicylate mixture as well as the ammonia, and the pains soon got better.

I may explain that urate of ammonium is one of the more insoluble urates, and that ammonium raises the acidity of the urine and increases the hourly excretion of acid. It would therefore increase gouty or rheumatic pains when given alone, but would not interfere with the action of salicylates; on the contrary would aid it.

I could easily give a number of similar cases; indeed, a week never passes without my seeing one or more of them. Almost every time a uric acid headache is cured some joint pains are produced, and in gouty and rheumatic cases the well-known effects of changes of weather can almost always be translated into changes in the alkalinity of the blood, secondary to the effects of the weather on the excretion of the skin.

Thus a patient recently seen gives me the following history of an attack of gout. He lives in India, but occasionally comes home to England, and after a previous visit to England, where he had been "feeding up," he felt the heat very much in the Red Sea on the way out, and then in Bombay was exposed to comparatively cold March winds, and had a violent attack of gout. Can anything be more simply and easily explained than this history? We have (1) the ingestion of a lot of uric acid, (2) its solution in the blood (uricæmia) as the result of heat, and (3) its precipitation on the joints by the rising acidity produced by cold winds.⁹

Similarly in patients who suffer from mental depression, pains in the joints are often looked upon as the harbingers of better things; and in women, when menstruation with its depressing influences causes a rise in the alkalinity of the blood and an excess of uric acid in that and the urine, mental depression or headache are generally worse, while rheumatic pains are to some extent relieved.

It is not my object in this paper to lay any great stress upon the cases quoted above, which I have been able to collect in a few months, but I hope, by bringing them to the notice of clinical observers, to induce them to regard their cases of arthritis and endocarditis in the light of what is now known about the solubility of uric acid; and if I am successful in doing so, I feel sure that they will very soon see hundreds and hundreds of similar cases, which only the mist of an erroneous pathology has prevented their seeing before, and they will not only see, but will be able to prevent and control, these disease processes to an extent which was previously impossible.

Disease by the visitation of God, or the work of that Jupiter Tonans of modern pathology, the nervous system, will give place to disease the result of folly in diet; the straightforward and easily verified chemistry of uric acid will replace the hypothetical microbe, and gout and rheumatism will be regarded as mere modifications of one and the same disease—arthritis due to uric acid.

⁹ See BRITISH MEDICAL JOURNAL, 1895, previous reference.

THE MEDICAL PROFESSION IN HUNGARY.—According to a statistical report recently issued by the Hungarian Minister of the Interior, the total number of medical practitioners and lower grade Surgeons (*Wundärzte*) in Hungary in 1894 was 4,280, being an increase of 106 as compared with the previous year. The distribution of practitioners throughout the country is very unequal: thus there are counties where 20 per cent. of the population are without a doctor. The average proportion of medical practitioners to population in the towns in 1894 was about 11.1 in 10,000. In Buda-Pesth there was 1 doctor to every 545 inhabitants, the lowest proportion in any town being at Hód-Mező-Vasárhely, where it was 1 in 3,460. In rural districts there was on an average 1 medical man to every 4,992 inhabitants.

OBSERVATIONS ON THE PATHOLOGY OF ENLARGED OR HYPERTROPHIED PROSTATE.

By REGINALD HARRISON, F.R.C.S.,
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IN advocating what I would speak of as the muscular theory in explanation of hypertrophy of the prostate I am not aware I ever denied that the prostate was dissociated from the genital function. I have referred to it as "a muscle containing a tolerably large proportion of glandular or secreting tissue embedded in it," and though failing to see evidence showing that it is engaged in elaborating the secretion of the testicles I adopt the view of Dr. Handfield Jones¹ expressed in the following words: Its function relative to the sexual act is "in supplying a vehicle which enables the fecundating fluid to act with greater certainty over a larger area whilst at the same time it supplies a muscular buttress against which the ejaculatory muscles of the urethra may advantageously act in the emission of the semen." It is therefore, I believe, to this extent a compound organ, and I am not aware that anyone has brought forward arguments contravening this limitation of duality.

I regard senile enlargement of the prostate as an example of a muscular hypertrophy analogous to other similar kinds of overgrowth, and arising out of the muscular functions in which the part is unceasingly engaged. It is of the first importance that its muscular action should be clearly established, for unless this can be done the whole of my argument necessarily falls to the ground. The difficulty that has hitherto arisen in fully recognising this explanation is in some measure due to the isolated manner in which the prostate has been studied.

It has been urged that though the prostate is in some measure involved in the process of micturition, the fact that it is used for this purpose half a dozen times or so in the twenty-four hours is insufficient to account for its overgrowth. This, I would emphasise, is merely the casual part the organ plays in what we generally regard as a voluntary act. Such a limited view as this implies is in a large measure due to the habit we have acquired of taking our ideas of the living organ from the condition it presents after death when removed from the body in a more or less contracted state, somewhat resembling a Spanish chestnut in shape. This is no more like the living prostate than the dead heart resembles the organ in full vigour and activity. The prostate assumes, I believe, no such appearance during life except on the rare occasions when the bladder is absolutely empty, but, on the contrary, the muscular fibres of which it is so largely composed are spread out like a funnel so as to furnish a contractile support for the bladder and its varying contents. This disposition is probably best appreciated by examination by the rectum when the patient is in the erect and semi-erect positions with the bladder empty as well as in various degrees of repletion. Though the existence of a prostate is not limited to man, I am not aware that in any animal it undergoes a change analogous to the senile enlargement observed in the human species. Hence, though enlargement of the prostate and the habit of maintaining the erect position may be mere coincidences, this fact cannot be allowed to pass unnoticed.

As the normal prostate is in this funnel-shaped manner spread out, so is it when it is enlarged. We are often surprised to find after death, or by a suprapubic incision, how much smaller the prostate turns out to be as compared with what it was when examined during its functional activity by the finger in the rectum. Under an anæsthetic an enlarged prostate usually ceases to oppose the introduction of an instrument into the bladder. Unless this continuous muscular action of the prostate in supporting the contents of the bladder in accordance with the degree required, as well as in minimising the effects of shock applied to this part of the body, are fully appreciated, it is impossible to understand how the functions of the viscus can be discharged without some other provision than that afforded by a mere fibrous floor.

It was not until I recognised the funnel-shaped manner in

¹ London Medical Gazette, vol. v, 1947.