as to reduce what had been a polycythaemia to figures which were just above those of a normal blood count. Four years before this event Dr. George Graham helped me in giving her continuous inhalations of oxygen. We demonstrated that the blood state of these patients is not improved by this measure.

Haematuria due to Hexamine.

In this series there is only one case of hexamine haematuria. I think there was no doubt about the causation. It was a case of acute encephalitis. A *post-mortem* examination was made eventually. The patient had been given only a moderate dose—20 grains every six hours—and at the end of the third day there was a profuse haematuria.

Unclassified Cases.

There were five unclassified cases, and I have already said that I feel sure that one or perhaps two of them would be put down as "essential haematuria" by those who consider that there is a group of cases to which we might give that provisional label.

One of these unclassified cases was a patient with aneurysm of the thoracic aorta; she had a profuse haematuria four days before death. The second of the unclassified cases might possibly be regarded as a case of calculus or nephrolithiasis. Then there was the case of a boy of 18 whom Mr. Ogier Ward sent to me from St. Peter's. I failed to make a diagnosis, but Mr. Ball was able to say that the blood came from one kidney only, and not from both. We know that in some of these cases of haematuria without obvious cause it is one side only that bleeds. That is no guarantee, however, that if nephrectomy is performed on that kidney the other one may not start a similar condition.

But perhaps the most typical case of what is termed "essential haematuria" was that of a man of 33 (looking up the literature I find that the cases coming into this loose group of haematurias are generally between 20 and 40 years of age), a cycle maker, who, in 1921, passed blood in his urine. He was examined with the cystoscope without anything abnormal being found. In November of that year he had a certain dull, not acute, pain, but nothing was done, because again nothing further was found. In January, 1922, he had a short attack of haematuria with no other symptoms. When he presented himself at the hospital a third time the left kidney was explored, but again with negative findings. He bled a little in February, 1922, and then nothing happened until he came under observation at St. Bartholomew's Hospital in March, 1923. There was no event in his past history or present condition to suggest any reason for this tendency to bleed. Radiology was quite negative, he had no dysuria, there were no signs of arterial changes, no hyperpiesis, no albumin or mucus in his urine. Conveniently for our purpose, he had an attack of haematuria while thus under observation, but the examination of the urine showed merely that there was a good deal of blood, a few blood casts, but no other casts, and the condition cleared up rather quickly; within a week his urine was normal again. I do not know what has happened to him since, but that is what I regard as a type of case which goes by the name of "essential haematuria."

It is easy to say that this is only a group of cases in which we do not know the cause of the haematuria, but it is a striking fact that in some patients bleeding from the kidneys occurs somewhat easily and without obvious cause. The matter has been written about a good deal, not much in this country, although Hale-White¹ has published a paper on it.

I have seen one case of this type in private. The bleeding was so profuse that everybody was alarmed, and said that something must be done; but everything had been done except nephrectomy, and eventually that was done. I failed completely to find anything wrong with the excised kidney, and I was particularly keen on one or two points which had been brought to my attention: one was the study of the pelvis in view of the possibility of minute papillomata, and the other was the so-called naevoid condition in the kidney substance itself.

Hale-White says of these cases that there is no satisfactory explanation; he uses the term "renal epistaxis" for them, and comes to the conclusion that there are two factors, one an undue ease of bleeding from the kidney and the other some toxic state at present unknown.

In my list there does not happen to be an instance, curiously enough, of tuberculosis, or of drugs other than hexamine. The condition has been brought on by use of carbolic ointments and lotions, and there is our familiar friend rhubarb, to which some patients are susceptible. I have also no instance of bilharzia haematuria. I thought, however, it might be useful to take actual cases as a basis for these rather discursive remarks.

An Address

ON

OBSERVATION, CO-ORDINATION OF KNOWLEDGE, AND JUDGEMENT.

ILLUSTRATED BY THE CARDIO-ARTERIAL SYSTEM.*

BY

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WHEN the subject of the address you invited me to give had been settled, I decided, after consideration, that the cardio-arterial system would afford all the illustration my theme required. I was strengthened in this decision by some sentences in one of the Gifford Lectures, which Professor J. S. Haldane of Oxford is delivering at Glasgow University. He is reported as saying, "From its beginnings the mechanistic theory of life was embarked on a hopeless task." "What physiology was progressively revealing was the detail of co-ordinated physiological activity." These quotations are applicable to the system we are to consider. Last century we heard much about the heart as a pump, and the arteries as pipes. I propose to look at them as the co-ordinated system of a *living pump and live tubes*, which alter their lumen under a variety of influences, and thereby affect the flow of blood through them.

CO-ORDINATION OF HEART AND ARTERIES.

Beginning with the heart, I ask your attention to the case of a lady who came to Edinburgh a short time ago to consult me.

CASE I.—*Weak Left Ventricle: Constriction of Arteries.*

The patient was a woman, aged 44, the mother of two or three children; she told me that for years she had had a leak at one of her valves. The pulse was 120, regular, small, and cord-like; the brachial artery was in a similar state. There was pulsation between the left sternal edge and the vertical mammary line; the left border of the heart by percussion was beyond that line; the right border was two inches to the right of the sternal edge; the veins above the right clavicle were greatly distended, and showed a ventricular systolic wave. On auscultation at the apex the first sound was faint and impure. Over the area of pulsation and over the adjoining sternum there was a loud blowing systolic murmur. The pulsation was not "a diffuse apex beat," but was caused by, and the murmur was heard over, the right ventricle. The murmur was therefore tricuspid in origin. The brachial pressure given by the sphygmometer was 130 mm. These observations, co-ordinated with our anatomical and pathological knowledge, showed that the left ventricle was seriously disabled; that the right chambers of the heart and the veins in the neck were distended with blood; that the safety-valve action of the tricuspid had come into play; and that the right ventricle was strong enough to produce a loud murmur as blood was driven back from ventricle to auricle. The pulse rate was 120, the brachial and radial arteries were small and cord-like.

When we have to deal with a weak left ventricle it means that the ventricle does not and cannot empty itself. The contractions may increase in number, but each contraction only projects a fraction of the ventricular blood into the aorta; there is, to use Professor MacWilliam's words, "defective cardiac output." The dogma of the

¹ *Quart. Journ. Med.*, 1910-11, iv, 509.

* An address delivered to the Royal Medical Society of Edinburgh.

physiologist is that as a consequence of defective output vaso-constriction occurs, "which tends to keep up aortic pressure." This doctrine of keeping up aortic pressure does not appeal to our clinical judgement when the power of the left ventricle is much lowered. Let me present another view, one which may appeal to clinicians. The aorta and its primary branches constitute a blood cistern,

which is kept full by the contraction of the ventricle lifting the weight on the aortic cusps and emptying its contents into the cistern. It must be realized that the pump, which keeps the cistern full, works from below and at a great disadvantage when enfeebled. The cistern, the fullness of which is essential to life, would undoubtedly rapidly empty did the outlets from the cistern retain their normal size; but they become constricted by virtue of possessing a living and active muscular coat, and the fullness of the cistern is assured—up to a point. I submit that this autonomous co-ordination of left ventricle output and arterial constriction is one of the most beautiful arrangements in the body. This is precisely what happened to the patient—namely, a damaged and weakened pump, and a constriction of the arteries easily recognized by the educated finger, yet her so-called blood pressure was 130 mm. Under treatment matters improved. The distension and pulsation in the veins disappeared, the tricuspid murmur ceased, at the apex a loud blowing systolic murmur appeared, the pulse fell to 70, the arteries were twice as large and had lost their cord-like character, and the sphygmometer reading had fallen from 130 to 105 mm. The left heart had regained power, was shooting more blood into the aortic cistern, and the arteries relaxed in virtue of their autonomous co-ordination with cardiac output. The pulse indicated the free outflow from the cistern, and I ask the reader to note that the sphygmometer pressure fell 25 mm.; and we are asked to believe that these records are blood pressure, and indicate heart power.

I direct your attention to Fig. 1, which represents a contracted brachial artery; and to Fig. 2, which is a bit of the same artery relaxed (the *post-mortem* appearance of a normal brachial artery). Fig. 1 represents the tightened up artery present in the patient I have spoken of. That that contracted, hypertonic muscular tube, with its thickened wall and much lessened lumen, offers no more resistance to the pressure of the armlet than the artery of Fig. 2, to which, of course, has to be added a normal living degree of tonicity, is a strange belief, and seems to have sprung from the traditional doctrine of the maintenance of aortic pressure even when the left ventricle had lost a large part of its power. Years ago the late Dr. Janeway, in conjunction with Park, experimenting in America with the artery of a bullock, the size of a human artery, came to the conclusion that when hypertonically contracted it required over 60 mm. of pressure to occlude its lumen.

HYPERMYOTONIA IN UNTHICKENED ARTERIES.

I now ask your attention to further clinical manifestations of hypermyotonia in unthickened arteries.

CASE II.—Hypermyotonia due to Acidosis.

A man had consulted me when his pressure was 120 mm. Some months later I saw him in the country. He was suffering from insomnia and air hunger; the heart action was irregular, and the arteries were tightened; the sphygmometer pressure was 160 mm. He was given sodium bicarbonate in considerable quantity, and 20 drops of paraldehyde instead of his usual hypnotic. When I saw him the following morning the air hunger was gone, he had slept well, and the pressure had fallen to 135 mm. I suggested that the alkali might be continued as the pressure would probably come down 10 to 15 mm. more. This was clearly a case of hypermyotonia due to acidosis.

FIG. 1.—Contracted Brachial Artery.

precordial pain, and he had had to stand still two or three times before he reached my house. His pressure was 158 mm., and his vessels were tightened up. This was clearly the kind of thing he had been describing to me but which I had not seen. He went into a nursing home, where, with rest and some simple treatment, the pressure stabilized to 120 mm. A day or two before he left the home he was sitting up in bed, and offered to show me building plans in which he knew I was interested. This he did, explaining them very clearly and in detail. When he had finished he complained of precordial pain: the pulse was tightened up, and the pressure, which had been 120 mm. before this effort, had risen to 150 mm. Here was an illustration of mental and some emotional stimulation leading to hypertonic arterial constriction and acting back on a myocardium with little reserve of power. I advised him to give up all his country activities, and to confine himself to office and administrative duties. He was an abstainer from alcohol and tobacco. His diet was regulated, a mild laxative was used, and a myocardial tonic taken. He was to avoid everything which threatened to bring on an attack. He has followed out these directions for nearly three years, and has been able to do a great deal of most useful work; but he has still to walk slowly on level ground, and when faced by a slight incline still more leisurely.

FIG. 2.—Same Artery as Fig. 1 relaxed.

These three cases illustrate three causes of arterial constriction in persons with unthickened arteries: (1) lowered cardiac output; (2) blood composition; (3) nervous or emotional influence. In all, when the hypertonia or constriction passed or was overcome, and the left heart recovered, the readings fell; and we are asked to regard them all as blood pressure only.

HYPERMYOTONIA IN SCLEROSSED ARTERIES.

Sclerosed arteries are common. Their characteristics are investigated most easily by examining the lower part of the brachial artery, where it is often superficial and its pulsation visible. The finger shows it to be hard and thickened, and often tortuous. The degree of sclerosis, of course, varies in different persons; a tightened up, and therefore hard, normal artery must not be confused with sclerosis.

In Fig. 2 the *post-mortem* thickness of a normal brachial artery is shown; but Figs. 3 and 4 represent advanced sclerosis. It will be noted that the thickening is due entirely to the thickening of the middle coat—that is, the muscular coat. This is the kind of artery which, during life, may be felt as hard as a pipe shank; and it was looked upon as a rigid tube. As to its resistance to the pressure of the finger, there was never any doubt until physicians were led away by the claims of the sphygmomanometer, or, as it was first named, the haemodynamometer. This thickened muscular coat is hard; but in many cases it is not a rigid tube, for it can contract and relax, although it has not the range of movement the normal artery has.

At this stage let me point out that the resistance offered by such a structural thickening as is represented in Figs. 3 and 4 cannot be removed; all that remedial measures can effect is to check hypermytonia, and the power to do this is illustrated in the following cases.

**CASE IV.—Moderate Sclerosis:
Hypermytonia 75 mm.**

A man, aged 58, while seated at his writing table was seized with inability to write, and he could not speak. He was taken home and seen by his doctor soon after arrival. His brachial pressure was 225 mm. He was given a tablet of erythrol tetranitrate. The following morning the pressure was 180, a fall of 45 mm. I saw him two days after his seizure. Speech had improved but was still blurred and it was difficult to make out what he tried to say. The right hand and arm had improved. The radial artery was small and hard, the brachial pressure was 175 mm. The heart sounds were faint. The apex by percussion was in the mammary line. He was given sodium iodide and bicarbonate and tincture of squill. A suitable laxative was suggested. I saw him again seven days later, when the pressure had been 160; my visit put it up to 180. Speech was much improved, but not quite pure, and he was able to write. Thereafter the pressure varied from 125 to 190, but it was promising to stabilize about 150. At that level it could be left, as a certain amount of sclerosis was assumed. Hypermytonia was responsible for at least 75 mm. There was in this patient a history of habitual constipation, prolonged worry, and considerable tobacco smoking.

**CASE V.—Marked Sclerosis:
Hypermytonia 30 mm.**

A man, aged 68, complained of being drowsy and lethargic, and fit for nothing. I saw him in November, 1923. The brachial artery was hard, thickened, and tortuous; the pressure was 210 mm. He had been taking each morning a purgative which gave him one or more loose motions daily. He was given a laxative which was to be used so as to give one formed motion daily; his diet was regulated, and he was advised to abstain from wine. Two weeks later I saw him again. He had carried out instructions. His unpleasant symptoms had disappeared, he felt quite well, and the pressure was 180 mm. Iodides had no effect in lowering this figure, so they were not persevered with. I saw him last in February, 1924. He had continued well, and the pressure had remained at 180 mm. His death was recorded in August, 1926; but I do not know the cause.

It will be noted that a fall of 30 mm. removed the symptoms complained of, while the structural changes in the arteries made further efforts at reduction unwise.

CASE VI.—Marked Sclerosis: Hypermytonia 60 mm.

A man, aged 54, sent to me at the end of February, 1924, complained of headaches, irritability, and somnolence—he fell asleep when he tried to read, and what he read he forgot in

a few hours; he was unfit for work. The pulse was languid and poor. The brachial artery was visible at the bend of the elbow; it was hard, thickened, and tortuous, and the pressure was 240 mm. The heart sounds, including the aortic second, were faint. The urine, acid, specific gravity 1018, contained no albumin or sugar. I had seen this patient ten years before, when the brachial pressure was 170 mm. The outlook now seemed very hopeless, but I suggested cascara and taraxacum as a daily laxative; squill and spirit of nitrous ether; and spare meals. He was a teetotaler.

He was sent to me again early in the following May, and I was surprised at the improvement. The drowsiness had gone, and memory had been recovered. The pulse was 84, regular and good, the myocardium had improved and the radial artery had lost its segmented character. The pressure was 180 mm.—a fall of 60 mm., and only 10 mm. above what it had been in 1914.

I need not multiply cases, but there are two others of such great significance that they must be briefly alluded to.

**CASE VII.—Suppression of Urine
after Prostatectomy.**

This was a patient with high pressure, who remained well when his pressure was kept about 160 mm. After removal of his prostate he had suppression of urine, and was steadily sinking into coma. The pulse was 140, difficult to count, and felt like a small firm thread. The pressure was 220 mm. With the approval of the surgeon I gave him pituitrin and erythrol tetranitrate—the former to counteract the spasm of renal vessels, which I assumed

to be the cause of the suppression, the latter to counteract the constrictive action of pituitrin on the systemic arteries. Within a few hours renal function was re-established; the threatening coma was removed; the pulse was large, slow, and regular; the pressure was 160 mm.

Here there was a fall of 60 mm., with relaxation of arteries and a steady good pulse wave.

**CASE VIII.—Thick Hard Artery; no Radial Pulse; Brachial
Pressure 110 mm.**

A man was admitted after a severe attack of pneumonia. He was collapsed and apathetic. There was no pulse at the wrist. There was no heart sound audible anywhere. On examining the brachial artery at the bend of the elbow it was visible, tortuous, and hard; and the pulse could be felt as a mere tap. A pressure of 110 mm. was required to stop the tap. On auscultation no sound was heard as the pressure was applied. Cardiac stimulants were administered, and when I saw him the following morning the pulse at the wrist was easily counted. The pressure was 125 mm.—a rise of 15. How many of the 15 were required to drive the pulse wave from the bend of the elbow to the wrist I do not know.

SUMMARY.

This last case provided a clinical demonstration, more convincing than many experiments, of the positive resistance of a sclerosed living artery, and of the unreasonableness of regarding sphygmometer readings as an index of heart power. Years ago I tested a sclerosed brachial artery, obtained soon after

death, and found that it offered a resistance of 100 mm. to compression.¹

The cases I have reported are examples taken from many. The observations were made in the course of ordinary routine examinations. I have shown constriction of a normal artery, and also the pathological changes which are present in the hard and thickened arteries known as arterio-sclerosis. I have given examples of the tightening up of sclerosed arteries, and of the removal of that tightening,

FIG. 3.—Sclerosis of Brachial Artery.

FIG. 4.—Sclerosis of Brachial Artery.

and of the concomitant fall of sphygmometer pressure varying from 30 to 75 or more millimetres. To me the judgement to be pronounced is clear, plain, and inevitable.

CAUSATION.

As to causation, let me refer for a moment to the term "hyperpiesis," introduced to define cases of raised sphygmometer readings in persons without arterio-sclerosis and with no evidence of kidney lesion. They were therefore inexplicable, and were labelled with this name. I have seen many cases of this kind, but I never saw one without palpable constriction of the brachial artery. But the users of this word, and the exponents of what is called "primary and essential hypertension"—hypertension meaning sphygmometer readings—do not recognize that a constricted normal artery, with its living and thickened wall, and lessened lumen, materially affects sphygmometer pressures. I have stated that Janeway and Park gave over 60 mm. for hypertonic contraction in an unthickened artery; the first three cases recorded here gave relaxation readings of 25, 30, and 25 mm.—and that figure after the left ventricle had improved. In Case IV, after the initial fall of 45 mm., the extreme sensitiveness of the arterial wall gave sphygmometer readings which varied from day to day between 120 or 130 and 190 mm. If heart power was being recorded in this case the readings would have indicated an erratic variation of heart power of from 90 to 100 mm. without any indication that the left ventricle was responsible.

As to the influences invoked to account for high pressures, Professor MacWilliam² in a recent lecture says:

"While it is obvious that some powerful agency is at work, the search for definite pressor bodies (from any source) in the circulation has proved to be singularly inconclusive. Clifford Allbutt said: 'Such catch phrases as intestinal toxins, kidney inadequacy, endocrine disorder, and the like are but dust in our eyes.'"

As a clinician who for many years has watched and measured the constriction and relaxation of the arteries examined for clinical purposes, I have learnt the great sensitiveness of this arterial function. The physiological conception that it is mainly, if not wholly, to maintain or regulate blood pressure is much too restricted a view. The arteries respond not only to cardiac output and to nervous influences, but to influences from the alimentary tract, including excess of protein food, wine, tobacco, insufficient removal of hepatic excretion, constipation, and so on. The subtlety of the chemico-vital composition of the blood in different individuals is a fact of which the observant physician has abundant proof, although the laboratory may not be able to explain it or define it by analysis. Biochemistry has done much; but can chemical analysis of the blood determine minor degrees of what is known as acidosis? Yet the clinician removes symptoms, and relaxes hypertonus, by the administration of sodium bicarbonate. So also with treatment directed to biliary secretion and suitable colon emptying; in other cases to the reduction of protein food; in others abstention from wines, and so forth. In all these classes the only feasible explanation is that there is a chemical substance present in the blood which acts directly on the arterial wall. Clinical medicine cannot wait for the advance of biochemical analysis for the confirmation of treatment results. Supposing that large protein feeding acted by increasing the secretion of adrenaline, could biochemistry show its increase in the systemic blood? The same question can be asked with regard to other possible constituents. As a clinician, with some understanding of chemical pathology, I entirely repudiate the doctrine that "such catch phrases as intestinal toxins, kidney inadequacy, endocrine disorder, and the like are but dust in our eyes." Any one of them, or any combination of them, can, and does, cause arterial constriction; and that, as we have seen, means a rise in sphygmometer reading.

The clinical observation which it is most important for us to train ourselves to make is the tightening up of the arteries we examine for clinical purposes. Arterio-sclerosis is the result of more or less continued tightening up in persons whose hearts readily respond to extra strain. It takes years to develop; and it can be checked if tightening

is recognized and means are taken to eliminate the factor which is the prime cause. The heart hypertrophy is the response of the heart to the extra strain of driving blood through constricted channels. Kidney changes develop of an interesting and informative type. The renal artery outside the kidney shows the type of change seen in Figs. 3 and 4, whilst the divisions of the artery inside the organ show an irritative and progressive endarteritis. This goes on to the "gouty kidney" of last century, the arterio-sclerotic kidney of a later time.

REFERENCES.

- ¹ W. Russell: *The Sphygmometer*, Baillière, Tindall and Cox, p. 79.
- ² Professor MacWilliam: *BRITISH MEDICAL JOURNAL*, January 22nd, 1917, p. 125.

THE HISTOLOGY OF THE PERIODONTAL SULCUS.

BY

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(With Special Plate.)

THE importance of the periodontal sulcus as being the seat of origin of pyorrhoea will be patent to all who have grasped the significance of dental sepsis, yet it is a fact that there are no definite data as to its intimate structure. The chief point at issue has been how is epithelial continuity provided for in the normal condition? Does the gum epithelium attach itself to the tooth, or is there normally a breach of epithelial continuity where the tooth projects? In the latter case the apex of the sulcus would be a serious defect in our defensive armament.

For many years I failed in my efforts to get a section of the normal periodontal sulcus, owing to the dissociation of the tissues which occurs during decalcification and sectioning. A few years ago, however, I hit on the plan of embedding in celloidin before decalcifying, and have been able to get some very useful sections. Having no access to the dead-house I have had to rely on the forceps for my material. Obviously the forceps destroys the lingual and labial parts of the periodontal sulcus, so that my observations relate only to the interstitial part. In many cases a piece of the interstitial gum comes away attached to the tooth, probably more or less dissociated, but possibly intact. I have had a large number of such specimens sectioned, and have been led to the conclusion that interstitially at least there may be, possibly should be, no periodontal sulcus. I have found a considerable number of cases in which the gum is attached directly to the tooth-crown, and the attachment has been firm enough to resist disruption at the moment of extraction. Some of these cases show that there was no possibility of a sulcus (Fig. 1), the gum epithelium being prolonged on to the tooth-crown and fusing with Nasmyth's membrane; others leave a possibility that there was a shallow sulcus, not, however, at the neck of the tooth, but in a position halfway along the crown (Fig. 2). Some sections, again, suggest a potential rather than an actual sulcus (Fig. 3).

The question of epithelial continuity may be definitely answered. There should be no breach until the dentine of the tooth is exposed, or the neck denuded. The gum epithelium may spread over the tooth-crown, much as the skin epithelium over a nail (Fig. 1), becoming continuous with Nasmyth's membrane over a wide area; or it may be continuous with Nasmyth's membrane only at the edge, leaving the fibrous gum to take direct attachment to the tooth-crown (Fig. 5); or for a varying distance it may be split away from Nasmyth's membrane, taking a vital attachment only as the neck of the tooth is approached (Fig. 6); or it may take attachment only to the neck of the tooth—that is, to the cementum (Fig. 7). The first and last two cases leave a possibility of a periodontal sulcus which would develop as the gum epithelium split away from Nasmyth's membrane, possibly as the latter became less vital owing to ageing, or as the gum epithelium peels off