given square areas of surface above and below the semilunar valves is equal, whether those valves be present and efficient, or be absent. Granting this, how can Drs. Föster and Fothergill refine upon supposed sets given to the current this way, and that as they would reason of water in a closed culvert or in a rain water pipe? Nor again, can I admit the right of the author to find in diminished tension any such explanation as he assumes. Tension does not depend upon the force of the heart-beat, but upon those nervous adaptations to which Dr. Fothergill so eloquently appeals. Moreover, were it not so, he will scarcely deny that tension is equal at any and all points say of the inner surface of the ventricle and of the aorta. This being so, how can the blood thrust itself or be thrust harder into one coronary artery than into another? The most elementary knowledge of hydrodynamics would guard us from so crude a conception as this. I do not hesitate to advance that all elaborate prognoses based upon this conception are worse than wasted.

Once more, does Dr. Fothergill really believe that hypertrophy of a part bears an exclusive relation to the quantity of blood submitted to it? If blood be denied to the part, no doubt it must starve, but it would be too hardy an assertion to say that in aortic regurgitation the blood is shut off from access to the coronary arteries. Surely they have still the same supply that is given to any other artery in the body, and the cardiac muscle can grow still in obedience to the general laws which govern hypertrophy. We know that fatty degeneration of the heart may be detected during life, and the heart muscle nevertheless recover perfectly, if the coronary arteries be normal. Excessive arterial supply, on the other hand, cannot alone give rise to hypertrophy; so far as we know it is the *vis à fronte* not the *vis à tergo* which is herein concerned. We must look, therefore, to some causes of systolic failure in aortic regurgitation other than differential pressures at the mouths of the coronary arteries. The question is of such direct practical importance that for many years I have sought to solve it. Dr. Fothergill will, perhaps, remember that when he lived in Leeds, I showed him notes, preparations, and specimens illustrative of my views on causation of aortic arteritis and valvulitis by bodily effort acting either suddenly or very gradually. In these I frequently found the mouths of the coronary arteries lessened, and sometimes found them occluded by "atheroma".

It was thus possible in many cases to trace fatty change in the muscular tissue supplied by one coronary artery to atheromatous or embolic blocking and so to arrest of blood-supply. To prove that this cause is the only cause of asystole in aortic regurgitation would be difficult, and probably it is not the only cause: that it is a common cause of it I feel pretty confident. The compensation itself is but imperfect at best, and carries in itself no assurance of permanency. However, the purpose of my present criticisms is rather destructive than constructive. After I had committed these criticisms to paper, I consulted Dr. Stone, in order to secure myself against error. Dr. Stone wrote to me as follows on October 2nd, 1879. "Dear Dr. Allbutt,—My views would be entirely in agreement with yours. Indeed, it is quite unmechanical to suppose that the coronary arteries are filled by a 'squirting' action. Neither do I believe that hypertrophy originates in arterial action, though fatty degeneration may depend on coronary starvation. The regulating power must originate in the beautiful balance of motor and inhibitory centres which the arteries obey. Dr. Fothergill is really going back to the obsolete humorism. Dr. Ottomar Rosenbach's experiments, moreover, show conclusively that tension and propulsion, in the closed cell of the circulation, are perfectly separable factors, and that the pressure is maintained unaffected, even when the aortic or mitral valves are artificially broken down. This I worked out in my third Croonian lecture, which I am now preparing for publication."

AN INQUIRY INTO CERTAIN POINTS CONNECTED WITH ALBUMINURIA.

By ROBERT SAUNDY, M.D. Edin.,

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RECENT observations* have established the frequency with which albumen appears in the urine apart from structural disease of the kidneys or any marked deterioration in the general health. It has been long known†

that albumen is present in many grave diseases without structural renal disease or the other symptoms of Bright's disease accompanying it.

In an article published last year, on Albuminuria (*Birmingham Medical Review*, July 1879), I reviewed all these facts, and, having defined albuminuria as "the presence in the urine of a body coagulated by heat or precipitated by neutralisation"; I showed that there were only two albuminous substances which are likely to be met with giving these reactions—viz., 1. Serum-albumen, and 2. Paraglobulin. I am not aware that any researches have been undertaken to show whether paraglobulin ever occurs by itself in the urine; but Edlefsen, J. C. Lehmann, and Senator found it constantly present, together with serum-albumen, in the urine of Bright's disease. The first part of my inquiry was therefore directed to settle this point.

Chemists tell us that paraglobulin is wholly precipitated from its sodium-chloride solutions by saturation with the latter salt or by carbonic acid. On my applying for information on this point to Professor Crum-Brown of Edinburgh, he wrote to Professor Hoppe-Seyler, who referred me to a paper by Hammarsten (Pflüger's *Archiv*, Band xviii, s. 38), in which magnesium-sulphate is recommended; and this method Professor Hoppe-Seyler informed Professor Crum-Brown he had found trustworthy in his own investigations on milk. In the following experiments, all these methods were used, but in many they were combined.

The method of proceeding was to test an urine for albumen by boiling and adding acetic acid, then taking a fresh portion to precipitate it, filter, and test the filtrate as before; the two test-tubes were then compared. The cases were taken as they appeared in the out-patient room, without any kind of selection, and present most of the conditions under which albuminuria occurs apart from acute disease. In all, forty-two examinations were made; two of these being of the same person in one instance only. They comprised cases which may be thus classified: Chronic Bright's disease, 5; albuminuria of adolescents, 16; epilepsy, 7; morbus cordis, 3; amenorrhœa, 3; phthisis, 3; bronchitis, 3; gastric catarrh, 1; scabies, 1. The albuminous cloud diminished after precipitation as above in a large number of these cases, and entirely disappeared in two. One of these was a case of phthisis; the other presented symptoms of dyspepsia, and has been included among the cases of albuminuria of adolescents. On subsequently examining the urine of the same case, the albuminous cloud disappeared partially only. Hence the existence of paraglobulinuria as an independent condition is certainly rare, and cannot be held to explain the frequency with which the reaction of albumen is given by the urines of adolescents and others supposed to be suffering from merely functional derangements of the renal organs.

The next point alluded to in my article already quoted is the doctrine of "food-albuminuria", originated in modern times by Parkes (*Medical Times and Gazette*, 1852, p. 357, and 1854, p. 395). He found that the albumen was increased after food in two cases of chronic Bright's disease, although it was diminished after food in a case of heart-disease. He inclined to the view that the increase after food was due to the passage of imperfectly digested albumen, analogous to that which occurs when albumen is injected into the veins, or to the albumen undergoing some modification in the digestive process, such as its conversion into an acid albuminate, by which its diffusibility would be increased.

Parkes's facts do not seem to warrant this conclusion; for, while the albumen was increased after food in the two cases of Bright's disease, it was diminished under similar circumstances in the case of heart-disease, so that it is necessary to postulate a peculiar inability to digest albumen in the former cases which did not exist in the latter. On the other hand, if we regard albuminuria as a mechanical process, resulting from increase of the lateral pressure within the vessels to such a degree as to permit transudation, we can easily understand why, in the cases of chronic Bright's disease, the stimulus to the circulation increased the albuminuria in already inflamed organs, while it diminished it in the case of heart-disease, where the transudation was due to passive engorgement of the renal capillaries and veins. But Parkes's views have been adopted by many writers. Dr. Pavy (Gulstonian Lectures, *Lancet*, 1863, p. 573) supports Parkes by giving a table of the amounts of albumen excreted before and after breakfast in six experiments, all showing a marked increase. Dr. Pavy further adopted Parkes's view that this might be due to the increased diffusibility of the urine, and supported it by showing that the albumen in some urines is highly diffusible, but did not attempt to prove that the albumen passed after food was more diffusible than that passed before food, or to indicate to what circumstances this increased diffusibility might be due.

Most physiologists deny that unchanged albumen is absorbed into the blood, and the only piece of direct evidence I know to the contrary is the statement of Brücke, that he has found coagulable albumen in the

* Dr. Moxon, *Guy's Hospital Reports*, third series, vol. xliii.; Dr. Dukes, *BRITISH MEDICAL JOURNAL*, November 30th, 1878; Dr. Morley Rooke, *ibid.*, October 10th, 1878; Dr. Lauder Brunton, *The Practitioner*, June 1877; Professor Leube, *Virchow's Archiv*, Band lxxii, Heft 2; Dr. Saundby, *Birmingham Medical Review*, October 1879; Dr. Mahomed, *Guy's Hospital Reports*, vol. xxiv.; Dr. Geo. Johnson, *BRITISH MEDICAL JOURNAL*, December 13th, 1879.

† Dr. Parkes, *On the Composition of the Urine*, page 186; Dr. W. Roberts, *On Urinary and Renal Diseases*, p. 173; Dr. Quain, *The Lancet*, January 18th, 1879; Dr. Paul Fürbringer, *Virchow's Archiv*, Band lxxi, Heft 3.

lacteals. My attention was first directed to the question whether the albumen is increased after food.

The following experiments were undertaken to decide this point.

CASE.—T. B. Chronic Bright's disease with anasarca. Diet: Milk, bread and butter; fish for dinner; and one boiled egg for tea.

Experiment I.—*Before breakfast:* In bed. Urine faintly acid; 1013; contained one-third of a column of albumen.—*After breakfast:* In bed. Urine acid; 1018; two-thirds of a column of albumen.—*Before dinner:* Up. Urine acid; 1018; half a column of albumen.—*After dinner:* Up. Urine strongly acid; 1024; a whole column of albumen.

Experiment II.—*Before breakfast:* In bed. Urine 1012, faintly acid; one-third of a column of albumen.—*After breakfast:* In bed. Urine 1020, acid; two-thirds of a column of albumen.—*Before dinner:* In bed. Urine acid; 1015; half a column of albumen.—*After dinner:* In bed. Urine faintly acid; 1016; one-third of a column of albumen.

Experiment III.—*Before breakfast:* In bed. Urine 1014, faintly acid; contained half a column of albumen.—*After breakfast:* In bed. Urine acid; 1022; two-thirds of a column of albumen.—*Before dinner:* Up. Urine acid; 1020; three-fifths of a column of albumen.—*After dinner:* Up. Urine faintly acid; 1019; two-fifths of a column of albumen.

Experiment IV.—*Before breakfast:* In bed. Urine faintly acid; 1013; two-fifths of a column of albumen.—*After breakfast:* In bed. Urine 1018, acid; four-fifths of a column of albumen.—*Before dinner:* Up. Urine acid; 1021; nearly a whole column of albumen.—*After dinner:* Up. Urine faintly acid; 1022; contained nine-tenths of a column of albumen.

In all these experiments, the urine was collected for one hour before and one hour after the meals in question. If we add up these quantities, we find that the totals are:

Before breakfast	...	...	1.56 column
After	...	...	2.8 "
Before dinner	...	...	2.6 "
After	...	...	2.6 "

Thus, in spite of the assistance of exercise (except on one occasion) in augmenting the excretion of albumen at dinner-time, and in spite, too, of the more albuminous diet at that period, the after-breakfast urine is not only relatively more albuminous than that passed when fasting, but is also more albuminous than that passed after dinner.

At my request, Dr. A. H. Carter made the following valuable observation on a case of Bright's disease, under his care, in the Queen's Hospital, Birmingham. The quantitative analyses were made by Dr. C. A. MacMunn of Wolverhampton, whose competence and care are a guarantee for their accuracy. The observations were made on the 10th, 11th, 12th, and 13th of March, 1880; but, as one of the bottles was broken in transit, only three days are recorded.

Diet.—Breakfast, 5 A.M., two slices bread and butter, with tea.

Lunch, 10 A.M., " " " " milk, 1 pt.

Dinner, 1 P.M., cooked meat 4 ozs., potatoes 8 ozs., bread and water.

Tea, 6 P.M., same as breakfast.

Supper, 7 P.M., milk half-pint.

Exercise.—Walking exercise twice daily for half-an-hour, at 12.30 and 2.30 P.M.

Date.	Period.	Quantity of Urine.	Albumen in 40 ccms.	Total Albumen.
March 10.....	7 P.M. to 5 A.M.	1620 cc.	.008 grms.	.1296 grm.
"	5 A.M. to 1 P.M.	150 cc.	.016 "	.024 "
"	1 P.M. to 5 P.M.	180 cc.	.01 "	.018 "
"	5 P.M. to 7 P.M.	120 cc.	.008 "	.0096 "
" 12.....	7 P.M. to 5 A.M.	870 cc.	.0069 "	.0601 "
"	5 A.M. to 1 P.M.	180 cc.	.017 "	.0306 "
"	1 P.M. to 5 P.M.	180 cc.	.0141 "	.0254 "
"	5 P.M. to 7 P.M.	150 cc.	.009 "	.0135 "
" 13.....	7 P.M. to 5 A.M.	1410 cc.	.0155 "	.0218 "
"	5 A.M. to 1 P.M.	180 cc.	.020 "	.036 "
"	1 P.M. to 5 P.M.	180 cc.	.0166 "	.0298 "
"	5 P.M. to 7 P.M.	120 cc.	.015 "	.018 "

On each day, the relatively greatest quantity of albumen was secreted between breakfast and dinner. The quantity each day fell as the day advanced, in spite of the meat eaten at dinner and the exercise taken in the afternoon. The total quantity of albumen excreted rose steadily under the influence of meat diet, being three times greater on the fourth day than on the first.

This supports the opinion expressed by Dr. Moxon, that in cases of intermittent albuminuria the after-breakfast urine is most likely to con-

tain albumen. It corroborates Dr. Pavy's facts, but weakens his inference, as it shows that the amount of albumen does not depend upon the quality of the food so much as on the period of the day. In illustration of the last observation, it is worth while calling to mind the well known researches of Dr. Edward Smith* on the diurnal variations of the vital processes. He showed that the pulse, respiration, and urea excretion were each influenced to a much greater degree by breakfast than by any other meal, and that the maximum of all these vital processes occurred at this period of the day, although the food taken at dinner was much more nitrogenous.

These facts are opposed to the view that nitrogenous food increases albuminuria, by passing with the urine as unassimilated albumen. They prove that the maximum albuminuria occurs in that period of the twenty-four hours in which the whole of the excretory functional activities are also at their maximum; and the inference is permissible, that the increased albuminuria depends immediately upon the increased functional activity of the kidney during that period.

This relation of albuminuria to a particular period of the day has never before been carefully investigated; and the want of acquaintance with it explains the incorrect conclusions of Parkes and Pavy.

These observations are not opposed to the conclusions of Drs. Sparks and Mitchell Bruce (*Medico-Chirurgical Transactions*, vol. lxii); there can be no doubt of the general influence of nitrogenous food in increasing albuminuria, but they suggest the probability that this relation depends upon the increase of urea, that is, the increase of the physiological stimulus to the kidney, and they show that this effect is greatest at that period of the day in which the renal functions are most easily excited.

The next point was to determine whether the albumen passed after food diffused more readily than that passed before food. In considering this question, it must be borne in mind that acid albumen diffuses readily, and, as is well known, the urine passed during digestion is usually of higher specific gravity and more acid than that passed at other times. The septum used was vegetable parchment; and the time allowed was twenty-four hours in each case.

The following experiments were made on the urine of the same case.

Experiment I.—*Before breakfast:* In bed. Urine faintly acid; one-third of a column of albumen; no albumen in diffusate.—*After breakfast:* In bed. Urine acid; four-fifths of a column of albumen; a trace of albumen in diffusate.

Experiment II.—*Before breakfast:* In bed. Urine neutral; a trace of albumen in diffusate.—*After breakfast:* In bed. Urine acid; a trace in diffusate.

Experiment III.—*Before breakfast:* In bed. Urine faintly acid; one-third of a column of albumen; a faint trace in diffusate.—*After breakfast:* In bed. Urine acid; two-thirds of a column; a trace in diffusate.

Experiment IV.—*Before breakfast:* In bed. Urine faintly acid; one-third of a column of albumen; a trace in diffusate.—*After breakfast:* In bed. Urine acid; two-thirds of a column of albumen; a trace in diffusate.

Experiment V.—*Before dinner:* Up. Urine neutral; a distinct cloud in diffusate.—*After dinner:* Up. Urine acid; a distinct cloud in diffusate, but less than before dinner.

Experiment VI.—*Before dinner:* Up. Urine acid; half a column of albumen; a cloud in diffusate.—*After dinner:* Up. Urine strongly acid; a whole column of albumen; a dense cloud in diffusate.

Experiment VII.—*Before dinner:* Up. Urine acid; half a column of albumen; a faint trace in diffusate.—*After dinner:* Up. Urine faintly acid; one-third of a column of albumen; a faint trace in diffusate.

In two urines, marked neutral, albumen is stated to have diffused; this may possibly be accounted for by an acid fermentation having taken place. As a rule, the albumen appeared to diffuse in proportion to the acidity of the urine. The diffusate bore no relation to the quantity of albumen present in the urine.

Finally, as it might be presumed that the albumen met with frequently in dyspepsia and other functional derangements might be a modified form of albuminuria, I submitted the albuminous urines of twenty-one out-patients to diffusion, and found albumen in the diffusates of nineteen. There was no special frequency in any particular class of case, but diffusion to a moderate extent was plainly the rule. In one case, the diffused albumen disappeared after precipitation by magnesium sulphate. The twenty-one cases comprised six cases of chronic Bright's disease, in all of which diffusion occurred; one of sub-acute nephritis, diffused; eight of albuminuria of adolescents, of which only one did not diffuse; phthisis, one; morbus cordis, one; epilepsy, one; rheumatism, one; scabies, one; eczema, one, no diffusate.

* *Health and Disease.* By Edward Smith, M.D., etc. London: 1861. Pp. 27-48.

These observations appear to me to be opposed to the doctrine, that albuminuria is ever due to the transudation of a modified or more easily diffusible form of albumen.

I have shown that the coagulation temperature, as determined by Dr. Lauder Brunton (*St. Bartholomew's Hospital Reports*, vol. xiii) and Mr. D'Arcy Power, cannot be relied upon to afford any distinction between different forms of albumen, if such there be, as it depends upon the quantity of albumen and salts present. "The urine of a case of subacute nephritis, pale, faintly acid, specific gravity 1008, showed signs of coagulation when the thermometer, with its bulb immersed below the surface of the urine, marked 112° Fahr. Coagulation was looked for by the side of the bulb. The urine contained three-fourths of a column of albumen. Diluted five times, the coagulation point rose to 120° Fahr.; diluted ten times, to 130° Fahr.; twenty times, to 145° Fahr.; forty times, to 170° Fahr.; eighty times, to 180° Fahr. The same quantity of fluid and the same apparatus were used on each occasion."*

These facts appear to warrant the following conclusions.

1. The body present in urine which is coagulated by heat or precipitated by neutralisation is almost invariably serum-albumen, rarely paraglobulin.

2. The presence of paraglobulin alone in the urine is still somewhat obscure, and has not been correlated with any clinical group of cases.

3. The maximum amount of albumen excreted by the kidneys is during the after-breakfast period, and this relation is maintained in spite of the more albuminous character of the midday meal.

4. While there can be no doubt that nitrogenous diet increases albuminuria, this is not due to the absorption of undigested albumen, but to the increase of nitrogenous excreta, and the consequent stimulus to the usual functions.

CASES OF SYSTEMIC ARTERIAL AND PULMONARY EMBOLISM.

By W. VAWDREY LUSH, M.D., M.R.C.P.,

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CASE I. *Embolism into the Right Axillary, Left Brachial, and Right Middle Cerebral Arteries: Recovery.*—In December 1873, I saw in consultation a gentleman forty-six years of age. He had had varicose veins for some time, and was then suffering from an inflamed condition of the external saphena vein of his left leg. Subsequently, the right leg became similarly affected. In both, the pain, tenderness, and swelling gradually subsided, and the patient, being apparently convalescent, was left to the care of his usual medical adviser. His pulse was then 68. He was in no pain. His tongue was clean. But, although alkaline remedies had been administered, his urine after his dinner still remained turbid and acid.

On the evening of December 24th, the patient was sitting up in bed rubbing his left leg, the one which had been first affected, when he felt pain in his cardiac region, followed by some difficulty of breathing. His medical attendant was sent for, and examined his heart, also his lungs anteriorly and posteriorly, but could detect no morbid sound; but the pulse had risen from 68 to 98 in the minute. This increased frequency continued for two days. On each day, the patient sat up in his room.

About half-past seven in the evening of December 26th, he was carried by his wife and son from his chair across the room to his bed, supporting himself with his arms round their necks. He stated that, as they laid him on the bed, he felt a little palpitation, then slight giddiness, and then lost the use of both his arms. I saw him with his medical attendant at 10 o'clock. There was no pulse to be felt in either wrist; there was none in either brachial artery; there was none in the right axilla; but pulsation could be felt below the right clavicle, and also in the left axilla and in both carotids. The patient was perfectly conscious; he had no difficulty of breathing. He said he did not feel very ill, but complained of most severe pain in his right arm. There was no loss of motor power. He perspired freely. At one time, neither cardiac pulsation could be felt, nor cardiac sounds heard, but the patient revived on being laid down and given some brandy and water. The pain in the right arm continued for some hours, and then gradually subsided. The passage of an induced galvanic current through the arm seemed to afford relief. He was given five grains of carbonate of ammonia and ten drops of laudanum with some ether every two hours.

At 8.30 the following morning, the patient was free from pain, except at the tips of the fingers of the right hand. He was quite conscious

and composed, and related explicitly the circumstances of his attack. He had slept about twenty minutes. At 9.45, the patient appeared faint, and a twitch of the face was noticed. His voice, which had been hitherto clear, became muffled. He was partly comatose. The pulse in the temporal arteries was 84. Respiration was somewhat stertorous, 16.

December 28th. The tongue was pointing decidedly to the left when protruded. The cardiac pulsations were 92; respiration 16. He was taking nourishment well.

December 29th. The cardiac pulsations were 96; respiration 16; temperature 100°. Feeble pulsation was felt in the left radial artery.

December 30th. There appeared to be complete paralysis of the left arm and leg. The tongue still pointed to the left. Hearing was more acute than natural.

December 31st. Pulsation was felt in the right axilla; also in the left wrist.

January 1st, 1874. The pulse counted in the left radial was 84; the heart's pulsations were 84.

January 3rd. The pulse was felt in both wrists.

January 4th. There was perhaps slight movement in the left leg. On the 18th, he flexed it.

From this time, he gradually recovered. Upwards of five years have elapsed, and he is still living, can walk with the aid of a stick, has some power in the left arm, and is able to attend to his business and other duties.

I do not think there can be any doubt that this patient was attacked with arterial embolism. In my opinion, a thrombus had formed in the left auricle or left ventricle, had passed up into the aorta and divided, one portion passing into the right subclavian artery and another into the left, lodging in the one case below the clavicle, and in the other below the axilla. The next day, a portion of this clot was shot into the right carotid, passed into the right middle cerebral, and was followed by left hemiplegia. Eventually, the circulation became re-established, and there was partial recovery from the paralysis, that in the leg being as usual much more complete than that in the arm.

What was the cause of this blood-clotting? I believe, the excessively fibrinous or gouty condition of the blood. We know how prone gouty persons are to attacks of phlebitis; and throughout this patient's illness, his urine, in spite of alkaline remedies, was acid and deposited lithates.

Some who have heard of this case have supposed that the dislodgment of a clot from the veins of the leg would account for the symptoms; but a moment's reflection would show that such a clot would not pass through the pulmonary circulation to the left side of the heart, and be of sufficient size to block an axillary artery. In my opinion, the clot formed in the heart itself, and was thence carried into the arteries.

This case may be contrasted with that of a lady who no doubt suffered from three attacks of pulmonary embolism.

CASE II. *Three Attacks of Pulmonary Embolism in One Individual: Recovery.*—Some years ago, I was summoned to London to a near relative, who was dangerously ill. This lady, fifty years of age, had for some time suffered from profuse menorrhagia, associated with an uterine tumour, probably of a fibroid character, and she had had some swelling of her left thigh and leg. One day, just after a profuse catamenial period, she was attacked with pain in the chest, which became intense; her lips and nails became blue; she was apparently dying. On my arrival, she was somewhat better, but her pulse was rapid. There were most painful attacks of dyspnoea, the patient being obliged to sit up in bed, thirsting for air, as Trousseau describes it. This continued night and day; there was phlegmasia dolens of the left leg, which was swollen to a frightful extent. The patient's sufferings were most intense. During nine days and nights, she only once obtained two hours' consecutive sleep. Opium was of no avail, and a dose of chloral-hydrate caused her to leap out of bed.

At first, auscultation revealed nothing; but, on the seventh day, a pleural friction-sound was detected on the left side. She was then seen by my good friend and teacher Dr. James Andrew of St. Bartholomew's Hospital, who diagnosed embolism of one of the branches of the pulmonary artery. At his suggestion, five-minim doses of tincture of digitalis were given, and the patient was supplied with abundance of fruit, so as to lessen the liability to coagulation of the blood. The correctness of the diagnosis will be proved by the sequel. Finding opiates of no avail, I totally discontinued them, and sleep followed.

The phlegmasia of the right leg gradually subsided, and then the left became similarly affected. At length, the patient recovered sufficiently to be wheeled on a chair into an adjoining room, where she lay on a couch and went to sleep. In the evening, she was wheeled back to her bed; and, not wishing to disturb her attendant, endeavoured to get into bed herself, giving her leg a jerk in doing so. Im-

* *Birmingham Medical Review*, 1879, p. 227.