A POST-GRADUATE LECTURE

ON

THE INFLUENCE OF THE ARTERIOLES IN
RELATION TO VARIOUS PATHOLOGICAL
CONDITIONS.

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Emeritus Professor of Clinical Medicine, Consulting Physician to
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Her Majesty the Queen.*The Structure and Function of the Arterioles.—Apnœa (Asphyxia).—Anæsthesia by Nitrous Oxide and by Nitrogen.—The Pathology of Choleraic Collapse.—Variations of the Pulse.—The Cardio-vascular Change in Bright's Disease.—Raynaud's Disease.*

THE most important and fruitful addition to our knowledge of the circulation of the blood since the time of Harvey has been the discovery of the muscularity of the middle coat of the arterioles and the stopcock function of these small vessels in regulating and controlling the amount of blood transmitted to the capillaries and veins.

In 1840 Henle¹ described the circular muscular fibres in the middle coat of the arterioles and demonstrated their identity with unstriated muscular fibre in other parts of the body.

In 1852 M. Claude Bernard² published the first of his series of observations on the influence of the vasomotor nerves and the arterioles. His researches, together with those of Brown-Séquard, Waller, Ludwig, and others, have proved that the function of the arterioles is that of regulating the blood supply to the various organs and tissues; while some pathological phenomena to be presently described clearly indicate that the impediment caused by a general contraction of the systemic or the pulmonary arterioles is more than equal to the propelling power of the left or the right ventricle respectively.

My main object in the present communication is to prove that a due appreciation of the action of the arterioles is essential for a correct interpretation of some highly interesting and important pathological phenomena.

APNŒA OR ASPHYXIA.

There are few subjects which have given rise to so many conflicting statements and opinions as that of the immediate cause of death by deprivation of air. The application of the term asphyxia, which signifies an arrest of the pulse, to a condition whose primary cause is impeded or suspended breathing—apnœa³—has been one source of confusion which I will avoid in the present communication.

Dr. John Reid, in his paper "On the Order of Succession in which the Vital Actions are Arrested in Asphyxia,"⁴ has given a very complete account of the various attempts which had been made to explain the phenomena previous to the year 1840, when his paper on the subject was read at the British Scientific Association. He shows that before his own researches began no satisfactory explanation had been given of the impeded circulation through the lungs, and the consequent distension of the right side of the heart in the last stage of apnœa.

By making animals breathe nitrogen Dr. Alison and Dr. Reid proved that the impeded pulmonary circulation "is dependent upon the cessation of the chemical changes between the blood and atmospheric air in the lungs, and not upon the arrestment of the mechanical movements of the chest," as some of their predecessors had supposed, and they suggested that the arrest occurs in the pulmonary capillaries. This explanation, before the discovery of the structure and function of the arterioles, appeared sufficiently plausible. The most interesting original observation made by Dr. Reid was that of the increased pressure in the systemic arteries during the earlier stages of apnœa. This he explained by "an impediment to the passage of venous blood through the capillaries of the systemic circulation." As Dr. Reid's ex-

periments were conducted, the observation of the blood pressure was somewhat interfered with by the sudden variations which resulted from the violent respiratory efforts of the suffocated animals; but Mr. Erichsen⁵ subsequently, in a series of highly instructive experiments, got rid of this disturbing element by dividing the spinal cord as near to the cranium as possible, the animals then being kept alive for a time by artificial respiration. The results obtained by this method of experimenting were in entire agreement with those of Dr. Reid.⁶

It is a well-known fact that newly-born animals bear the exclusion of air much longer than the same class of animals a few days older. Thus Dr. M. Foster says,⁷ "while in a full-grown dog recovery from drowning is unusual after one minute and a-half, a newborn puppy has been known to bear an immersion of as much as fifty minutes;" and he goes on to say: "The cause of this difference lies in the fact that in the quite young or rather just born animal the respiratory changes of the tissues are much less active. These consume less oxygen, and the general store of oxygen in the blood has a less rapid demand made upon it." There is probably some truth in this statement, but the explanation ignores the fact that in these animals for some days after birth, and so long as their eyes remain closed, the foramen ovale is open, so that when air is excluded from the lungs the blood passes directly, as in the foetus, from the right to the left side of the heart, and so escapes the controlling influence of the pulmonary vessels.

Mr. Erichsen performed some interesting experiments on these newly-born animals, and found that not only did the circulation continue much longer after the exclusion of air than in older animals, but when the chest was opened after death there was no distension on the venous side of the heart, and the amount of blood on the two sides was nearly equal.

The description and explanation of the process of apnœa which had been published by Dr. John Reid and Mr. Erichsen were generally accepted by physiologists, and were repeated in all the textbooks until the year 1867, when Professor Burdon Sanderson, in his Croonian Lecture at the Royal Society,⁸ made the following statement: "It may be concluded that the extraordinary elevation of arterial pressure which has been long known to occur during the second minute in death from apnœa is not due, as was supposed by Dr. Alison and Dr. John Reid, to obstruction of the capillary vessels, either pulmonary or systemic, but to the violence of the respiratory efforts. The cavity of the chest being closed, the force exercised by the respiratory muscles expresses itself in variations of tension of the enclosed air, which are communicated through the intrathoracic arteries to those outside of the chest, producing those violent oscillations of the dynamometer which have been referred to. In support of this inference it was shown that in an animal under the influence of worara⁹—when all respiratory movement ceases while those of the heart are unaffected—the process of apnœa is not only of greater duration, but is not attended by any of those greater disturbances of the circulation which have been hitherto attributed to capillary obstruction. The gradual extinction of the force of the contractions of the heart is indicated by a slow and uninterrupted subsidence of the arterial pressure."¹⁰

In the *Handbook for the Physiological Laboratory*, published in 1875, Dr. Sanderson states that after death from apnœa "it is always found that all the heart's cavities are filled to distension, the quantities in the right and left cavities respectively usually being to each other in the proportion of about two to three."¹¹ The inaccuracy of this statement may be proved by an experiment which need occupy only a few minutes. I have been present on two occasions when the heart of a dog has been examined immediately after death from a ligature on the trachea. In the case of a large dog 2 ounces of blood gushed from the distended right cavities, while 2½ drachms trickled from the flaccid left side. In a small dog the distended right side of the heart contained 5½ drachms of blood, the left a quarter of a drachm.¹²

What, then, is the explanation of the results obtained by Professor Sanderson? Simply this, that the dose of worara which he administered to the dogs—one-tenth of a gramme—was sufficient to paralyse, not only the voluntary muscles,

but also the muscular walls of the arterioles, both systemic and pulmonary, and thus to render them powerless to impede or to regulate the onward movement of the blood.

Professor Rutherford, in a lecture on the circulation,¹³ says that "the dose of curara should be just sufficient to paralyse the voluntary muscles; if the dose be excessive the vaso-motor nerves are also paralysed. The amount given should be 12 milligrammes for a small dog, and 18 milligrammes for a large one." Thus Professor Sanderson's dose of one-tenth of a gramme (that is, 100 milligrammes) is more than five times the amount which Professor Rutherford employs for a large dog. I have at different times witnessed experiments which clearly show that when a moderate dose of curara is injected the results of apnoea are essentially different from those described by Professor Sanderson, while an excessive dose gives results which are in entire agreement with his.

I will now describe the most interesting and conclusive of these experiments.

In 1873 I witnessed a very instructive experiment by Dr. Rutherford when he was Professor of Physiology in King's College. Into the trachea of a large dog, previously anaesthetised, a tube was tied and connected with a bellows for artificial respiration. The voluntary muscles were then paralysed by a moderate dose of curara, and the animal was kept alive by artificial respiration. The sternum and portions of the ribs were removed, and the pericardium opened so as to expose the anterior surface of the heart. One common carotid was divided, and a dynamometer tube connected with a kymograph was tied into its proximal end. Artificial respiration being suspended there was first a steady rise of pressure in the carotid, and soon distension and dilatation of the left cavities of the heart (Fig. 1), then a rapid fall of

Fig. 1 represents the distension of the left cavities of the heart and aorta in the first stage of apnoea (asphyxia). *l.a.*, Left auricle; *l.v.*, left ventricle. Both greatly distended, the former like a smooth india-rubber ball. *a.*, Aorta distended; *p.a.*, pulmonary artery; *p.v.*, pulmonary vein; *r.a.*, right auricle; *r.v.*, right ventricle; *v.c.d.*, descending vena cava; *v.c.a.*, ascending vena cava. The right cavities of the heart, the pulmonary artery, and the systemic veins are in a state of normal fulness. The right ventricle is partly overlapped by the distended left.

pressure, distension and dilatation of the right cavities, with collapse and almost complete emptying of the left side (Fig. 2).

The explanation of the facts is briefly this. Venous blood passing into the systemic arteries excites through the vaso-motor ganglia, nerves, and centres,¹⁴ contraction of the arterioles, with resulting high arterial tension and distension of the left side of the heart from backward pressure. When the deoxidation of the blood exceeds a certain degree,¹⁵ the vasoconstrictors of the lungs impede the passage of the blood, the result being that the left side of the heart receives but little blood, and that of a venous character, the systemic arterial pressure falls, while the right cavities, the large veins, and the pulmonary artery are distended with blood. This unequal distribution of blood, which is invariably found when the chest is opened soon after death from apnoea, was, in the course of Dr. Rutherford's experiment, plainly seen to occur during the lifetime of the animal.

The suggestion that the empty condition of the left ven-

tricle after death from apnoea is the result of rigor mortis is obviously not in accordance with the facts thus demonstrated. And it is evident that in Dr. Rutherford's experiment the increased arterial pressure during the first stage of apnoea was not the result of "violent respiratory efforts."

Some physiologists assert that in the final stage of apnoea the systemic arterioles dilate in consequence of a supposed exhaustion of the vaso-motor centre by deoxidised blood; of this, however, there is no proof, and the assumed condition would not explain the facts. Dr. M. Foster says:¹⁶ "The blood pressure, in spite of the continued arterial contraction, begins to fall, since less and less blood is pumped into the arterial system." The condition of the heart's cavities soon after death from apnoea clearly indicates that the immediate cause of death is primarily the arrest of the pulmonic, and, as a consequence, that of the systemic circulation. It will be seen that there are two distinct stages in the process of apnoea: (1) that of systemic arterial obstruction, with distension of the left cavities of the heart; (2) that of pulmonary arterial obstruction, with distension of the right cavities. The two sides of the heart, therefore, are never fully distended at the same time, since in the final stage of apnoea, while the distension of the right cavities is rapidly increasing, that of the left is with equal rapidity passing away. It is also evident that the contraction of the pulmonary arterioles in the final stage has the double effect of increasing the resistance to the propelling force of the right ventricle while the walls of the heart are weakened by the scanty supply of deoxygenised blood to the coronary arteries.

It has often been noted after death from apnoea that more or less numerous ecchymoses appear beneath the pleura and pericardium. These are obviously the result of back-

Fig. 2 represents the distension of the right cavities of the heart, of the pulmonary artery, and the large systemic veins in the final stage of apnoea (asphyxia). The letters have the same significance as in Fig. 1. In addition, *p.c.* indicates the anæmic condition of the pulmonary capillaries; *b.*, left bronchus. The right auricle and ventricle and the pulmonary artery are fully distended, the auricle having the form and smoothness of a distended ball, while the left cavities of the heart and the aorta are collapsed and nearly empty.

ward engorgement of the bronchial and coronary veins and capillaries which share in the distension of the whole systemic venous system.

We have now to show that when the arterioles are paralysed by an excessive dose of curara or by other substances which are known to have that effect the results are identical with those described by Professor Sanderson.

My friend Mr. Charles James Martin, M.B., B.Sc., who is now teaching physiology in the University of Sydney, while Demonstrator of Physiology in King's College four years since, performed, at my request, some experiments, the results of which throw much light upon the complex phenomena of apnoea. All the animals experimented on were under the influence of anaesthetics, which, while they prevented pain, did not interfere with the physiological results of the experiments.

Into the peritoneum of a cat a decigramme of curara was injected, the animal being kept alive by artificial respira-

tion. A manometer connected with a kymograph was introduced into one carotid. When the respiration was suspended the pressure in the carotid immediately began to fall and artificial respiration was resumed to prevent the arrest of the circulation by cardiac paralysis. The same result followed more than once upon suspension of the respiration. At length after an interval of nearly an hour, suspension of respiration was accompanied by a rise of pressure in the carotid; and the heart having been exposed there was seen to occur the usual distension, first of the left cavities, then of the right, with shrinking of the left. In this experiment the excessive dose of curara paralysed the arterioles and weakened the heart. The suspension of respiration still further weakened the heart and lessened the arterial pressure. After a time a portion of the curara was eliminated, probably by the kidneys, a large amount of urine having been passed upon the operating table, and then the usual results of apnoea, with a moderate dose of curara, were observed.

A moderate dose of curara was injected into a cat. Suspension of respiration was followed by the usual rise and subsequent fall of the systemic arterial pressure. The circulation having been restored by artificial respiration, sulphate of atropine was injected into the jugular vein, after which the arrest of the breathing caused no rise of pressure in the carotid, and when the heart was exposed no unequal distension of its cavities was observed. The arterioles, both systemic and pulmonary, were paralysed by the atropine.

Into the trachea of a small dog, prepared with the chest and pericardium open, and kept alive by artificial respiration, a glass tube was introduced, through which pure nitrous oxide gas was passed into the lungs, whilst the expired gases escaped into the air. As usual in cases of apnoea, first the left then the right cavities of the heart became distended, and in one minute the heart's action had nearly ceased, with enormous distension of the right side. Then without loss of time inhalation of nitrous oxide, mixed with the vapour of nitrite of amyl, was substituted for the pure N_2O , by means of a two-way stopcock, and the result was that almost immediately the distension of the right cavities began to subside, and in two minutes they had nearly regained their normal size. In this experiment we have evidence that the contraction of the pulmonary arterioles—a result of deprivation of air—which caused the distension of the right cavities of the heart was quickly removed by the paralysing effect of the nitrite of amyl on the arterioles, atmospheric air being all the time strictly excluded by N_2O .

Some facts which tend to complete and confirm the theory of apnoea which I have here set forth yet remain to be mentioned. It has been noticed by different experimenters that during the earlier stage of apnoea the resistance resulting from the contraction of the systemic arterioles has a retrograde influence, not merely on the left side of the heart, as we have seen, but also upon the pulmonary veins, and even so far as the pulmonary artery, in which a slight increase of pressure occurs almost if not quite simultaneous with the much greater increase of pressure in the systemic arteries. In the final stage of apnoea, while the pressure in the systemic arteries is falling to zero that in the pulmonary artery is continually rising—an increase which can be explained only by the contraction of the pulmonary arterioles. The anæmic condition of the pulmonary capillaries which is always observed when the lungs are examined immediately after death indicates that the mass of the blood has been arrested before it has reached the capillaries, and when the lungs are exposed during life they are found to become visibly paler during the final stage of apnoea. The extreme collapse of the lungs when the chest is opened after death is a result of the bloodless condition of their minute vessels.

Dr. Rose Bradford and Dr. Percy Dean¹⁷ have proved the existence of pulmonary vasomotor nerves, and that the pulmonary circulation is comparatively independent of the systemic, but they say "it is probable that the vasomotor mechanism is but poorly developed as compared with that regulating the systemic arteries." It might with equal reason be suggested that the muscular walls of the right ventricle are "but poorly developed," since they are unable to overcome the resistance resulting from the contraction of the pulmonary arterioles in the final stage of apnoea. That the pressure in the pulmonary artery and its branches can never

equal that in the aortic system is obvious from anatomical considerations alone, but it may be safely assumed that the vessels of the lung, through which all the blood in the body has to pass, have the same regulating and resisting power compared with the force of the right ventricle as that possessed by the systemic vessels in relation to the left ventricle; and I am convinced that much of the confusion and contradiction which appear in the explanation of the phenomena of apnoea given by different physiologists is a result of their ignoring the influence of the pulmonary arterioles.

It is certain that by no experiment that has hitherto been devised can the actual propelling force of the right ventricle or the resisting power of the pulmonary arterioles be even approximately determined; and this for the obvious reason that any impediment to the flow of blood through the lungs, by mechanical compression of the pulmonary artery or its branches, weakens the heart by lessening the blood supply to the coronary arteries. So in the last stage of apnoea, when Mr. Martin has found the pressure in the pulmonary artery to be nearly doubled while that in the carotid is rapidly falling, the heart is weakened, not only by the small amount of blood which reaches its nutrient vessels, but by the fact that even that small supply of blood is entirely deoxidised. The convulsions which always precede death from apnoea when the muscles have not been paralysed by curara, and which tend to increase the distension of the right side of the heart by the pressure of the convulsed muscles on the veins, are the result of cerebral anæmia, the small amount of blood which reaches the brain being also entirely deoxidised. These convulsions are similar to those which result from a copious and rapid hæmorrhage from embolic plugging of the pulmonary artery, from the forcible entrance of air into a vein, or from ligature of the carotid and subclavian arteries.¹⁸

SUMMARY OF CONCLUSIONS RELATING TO APNŒA.

That the immediate cause of death from apnoea is the arrest of the pulmonary circulation is proved by the following facts: 1. When the chest of an animal is opened immediately after death by apnoea, the right cavities of the heart are found enormously distended while the left are comparatively empty. 2. When the heart of an animal is exposed during the progress of apnoea, the right cavities are seen to become distended, while the left cavities, which had been previously gorged, are rapidly unloading themselves. The obstruction in the lungs which causes the distension of the right cavities lessens the blood supply to the left. The two sides of the heart, therefore, are never fully distended at the same time. 3. In the final stage of apnoea there is a continuous rise of pressure in the pulmonary artery, while the systemic arterial pressure is falling. 4. That the arrest of the circulation through the lungs is caused by contraction of the pulmonary arterioles appears to be proved by the influence of agents which are known to paralyse those vessels—namely, nitrite of amyl, atropine, and an excessive dose of curara, the result being that deprivation of air is unattended by distension of the right cavities of the heart and other evidence of obstructed pulmonary circulation, the life of the animal is prolonged for several minutes, and death ultimately results from the toxic action of deoxidised blood upon the cardiac and nervous tissues. 5. It is an acknowledged fact that these paralysing agents act alike upon the systemic and the pulmonary arterioles, but the successive phenomena of apnoea are inconsistent with the idea that the distension of the right side of the heart is a result of systemic arterial obstruction acting backwards through the left side of the heart and the lungs. One result of such a retrograde influence would obviously be engorgement of the pulmonary capillaries, a condition the reverse of that which is found immediately after death from apnoea.

(To be continued.)

NOTES AND REFERENCES.

¹ *Wochenschrift für die gesammte Heilkunde*, 1840, No. 21. ² *Comptes Rendus*, March 29th, 1852. ³ The function of respiration requires a free access of air to the pulmonary cells and of blood to the capillaries. The exclusion of the former is apnoea, of the latter asphyxia. ⁴ *Physiological, Anatomical, and Pathological Researches*. ⁵ *Edin. Med. and Surg. Journal*, 1815. ⁶ It has been ascertained that the vasomotor centre in the medulla is not the only one, but that there are others along the whole length of the spinal cord and in the ganglia of the sympathetic, which, after section of the cord, may act as centres for the vasomotor fibres in parts

below the division of the cord. See Dr. M. Foster's *Textbook of Physiology*, 5th edition, p. 329; Cohnheim's *Lectures on General Pathology*, New Sydenham Society's translation, vol. 1, p. 113; and Vulpian's *Leçons sur l'Appareil Vasomoteur*, Tome 1, p. 281. ⁷ *Op. cit.*, p. 608. ⁸ *Philosophical Transactions*, 1867; *Proc. Roy. Soc.*, 1867. ⁹ One of the aliases for curara or curare. ¹⁰ *Proc. Roy. Soc.*, 1867, p. 393. ¹¹ For an account of the influence of Professor Sanderson's teaching upon subsequent writers on the physiology of asphyxia see my *Essay on Asphyxia (Apnoea)*. ¹² When the chest of an animal is opened immediately after death caused by a sudden deprivation of air the proportion of blood on the right and left side of the heart respectively is about that of 8 to 1, or ounces to drachms. After a prolonged and complicated experiment ending in apnoea, the heart's walls and the arterioles are enfeebled, and the disproportion of blood on the two sides is consequently less. Care must be taken to avoid wounding a large vein in opening the chest. Such a wound would quickly reduce the amount of blood in the right side of the heart, and the previously distended cavities would be found nearly empty. ¹³ *Lancet*, February 17th, 1872, p. 213. ¹⁴ It has been shown in Ludwig's laboratory that the arteries of an organ that has been withdrawn from all nervous influence contract when venous blood flows through them. Cohnheim, *Op. cit.*, p. 112. ¹⁵ The difference between ordinary venous blood and the blood in the veins and the right side of the heart after death from apnoea is that the former contains some oxyhæmoglobin, as shown by the spectroscope, while the latter contains none. ¹⁶ *Op. cit.*, p. 626. ¹⁷ *Proc. Roy. Soc.*, vol. xlv, p. 389. ¹⁸ See Kussmaul and Tenner on Epileptic Convulsions from Hæmorrhage, New Sydenham Society's collected *Monographs and Essays*, p. 236.

A CASE OF "FILARIAL DISEASE" OF THE LYMPHATICS IN WHICH A NUMBER OF ADULT FILARIÆ WERE REMOVED FROM THE ARM.

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WITH A DESCRIPTION AND IDENTIFICATION OF THE FILARIÆ
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THE following notes of a case of filarial disease are of much interest, and bring to light some new facts regarding the behaviour of these parasites.

T. L., aged 30, a Eurasian, and turner by trade, was admitted into hospital on September 11th, 1893, complaining of pain and swelling of the arm. The patient stated that he had always been in good health until four years previously, when he received a contusion of the left upper arm, which was followed by the formation of an abscess in that part. Since that time he had been subject to slight attacks of fever. The attacks used to come on at night, and last for about twelve hours, and were accompanied by pain running down the spine. They were not severe and never kept him away from work. A week before admission to hospital he received a blow on the left upper arm; not however a severe one. Six days afterwards the arm became swollen and painful.

He was found to be suffering from lymphangitis, affecting the lower part of the inner surface of the left upper arm. The blood examined at night was found to contain embryo filariæ. After suitable treatment the lymphangitis subsided, leaving some thickening extending along the inner side of the arm, from its middle to a point just below the elbow-joint.

As it was thought probable that the lymphangitis had been set up by the presence of filariæ, it was decided to explore the parts in the hopes of discovering and removing the parasite. Chloroform was therefore administered, an incision made and the whole of the thickened tissue removed. The wound healed by primary union, but some thickening formed at its lower extremity. The blood was examined after the operation, and found still to contain embryo filariæ. Eighteen days after the first operation another similar one was performed and the rest of the thickened tissue removed. The second wound supplicated slightly, and was followed by considerable thickening around the cicatrix. The blood still contained young filariæ. The patient was discharged on October 25th, as he was anxious to return to work.

The tissues removed on these two occasions were macerated in a mixture of nitric acid and chlorate of potash, and examined by Dr. Bourne, of the Presidency College, who found

in the lump first removed seven specimens of the adult *filaria sanguinis hominis*, and in the second lump one filaria. Three of these were males and the remainder females. Those first discovered were coiled up together in a mass, and appeared as if lying in a lymphatic channel, but owing to the maceration of the tissues it was difficult to be certain of this.

The operations on this patient were undertaken with the express purpose of removing the parasite, and by this means getting rid of a probable source of further mischief. Although the result shows that all the worms have not been removed, yet the fact that it was possible to diagnose the presence of the parasite and remove a number of them should, I think, encourage us to undertake operations of a similar nature in other cases.

In the *Indian Medical Gazette* of October, 1891, I suggested that operations of a similar nature might be undertaken in cases of lymphangiectasis, and I published the notes of a case in which such an operation had been performed with great benefit to the patient. This patient was cured of the attacks of inflammation to which he had previously been subject, and the embryo filariæ disappeared from his blood. At the time that the case was published, two years and a-half had elapsed since the operation, and the patient remained well, but I have lately heard that although he still remains in comparatively good health, yet a new swelling has developed on the opposite side of his body, and filariæ are again to be found in the blood. Since 1889, ten operations for the removal of lymphangiectasis have been performed in the Madras General Hospital. In some of these cases filariæ were found in the blood, in some they were not found, but all the cases were considered to be of filarial origin. One of these patients returned to hospital two years and a-half after operation suffering from elephantiasis of the leg. In five of the cases the operation has been so recently performed that no opinion can yet be formed as to the result. Unfortunately none of the other cases have been heard of since the operation.

An interesting point in the case just related is the great number of the parasites that were found, and also the fact that they appeared all to be intimately associated with one another. So far as I know, such a large number of these parasites have never before been found in one individual, nor have they been found coiled up together in one mass as a number of these were. This arrangement is, however, found in the cases of other varieties of filariæ, and therefore it is probable that it is not an unusual one in the case of the parasite under discussion. Another point of interest in the case lies in the situation occupied by the worms. There is still some uncertainty as to the exact part of the lymphatic system usually selected by the parasites for their habitation, and whether they remain permanently located in such situation or not. There is, however, strong reason to believe that in the great majority of cases they lodge in the distal lymphatics of the limbs.

The case furnished another proof, if such were wanting, that the lesions produced by the adult parasites themselves are not such as result in elephantiasis and lymphangiectasis. Although this small colony of filariæ had managed to set up local irritation and inflammation of the lymphatics, yet the main circulation of lymph throughout the limb was in no way interfered with.

DESCRIPTION AND IDENTIFICATION OF THE PARASITE BY DR. MANSON.

[Along with the foregoing interesting and important communication, Surgeon-Major Maitland sent two slides containing specimens of the parasites referred to; these were handed to Dr. Manson, who reports as follows]:—

Of the two slides one contains the greater part of two female *filaria bancrofti*; unfortunately the tail end of both worms is missing. The other slide contains three complete male and one complete female *filariæ bancrofti* in a fair state of preservation. The parasites are mounted in balsam which has caused considerable contraction and, here and there, actual shrivelling of the structures; on the whole, however, and by supplementing what is invisible or obscure in one worm by what is distinct in the others, a fairly complete idea of the coarse anatomy of *filaria bancrofti* can be obtained.