An Address

ON

ARTERIAL SCLEROSIS AND HYPERTONUS IN THEIR RELATION TO DIET AND TO THE DIGESTIVE SYSTEM.

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PERMIT me at the outset of my remarks to indicate to you the meaning I attach to the term "arterio-sclerosis." Clinically, the word means a thickening or induration of the arterial wall, perceptible to the sense of touch, the ordinary clinical method of examining vessels.

My own observations¹ on persons who have shown the existence of this condition during life, and whose bodies I have examined after death, have led me to the conclusion that the changes present are (1) a hypertrophy of the middle or muscular coat, and (2) a fibrous hyperplasia of the internal coat. These changes are present in the radial arteries, and, more or less, in the arteries in all the more important situations in the body. The thickening in the radial arteries is very commonly spoken of as atheroma; but it differs from atheroma as entirely as other morbid processes differ from one another.

TONUS.

In the next place you must bear with me while I deal with certain elementary phenomena connected with the arteries. We all know the word "tone" as applied to the radial pulse. Tonus, or tone, may be used as indicating, or as including, the actual degree of contraction of the muscular coat. There is an average or normal tonus which in its sustained rhythmical response to the rhythmical flow of the blood resembles elasticity, such elasticity as is manifested by the walls of the pulmonary alveoli in stretching during inspiration and retracting during expiration. The arterial movement, however, is not a mere mechanical one of that kind; but is, on the contrary, presided over by a tissue—involuntary muscle fibre—which has the power of active contraction and relaxation, and even initiates these movements under the influence of stimuli of one kind or another. This continuous and unceasing rhythmic movement of involuntary muscle is only seen in heart and vessels. Emphasis has to be laid on this property in arteries because of the tendency to regard circulatory phenomena from the purely physical side—the heart a pump and the vessels a mere system of elastic tubes. While this purely mechanical conception is applicable, as I have said, to the vesicular movements in respiration, it leads to serious misapprehension if undue prominence is given to it when dealing with circulatory conditions. In the consideration of circulatory manifestations—manifestations which lie at the very foundation of such a large part of our professional work—it cannot be too strongly insisted on that this active and sensitive responsive power of the living muscle fibres of the arterial wall must be given a place second only to the driving power of the left ventricle.

While an average tonus may be postulated, the average is a variable measure, varying within considerable limits, varying from day to day, and even from hour to hour. Some of the influences which determine the tonus will be considered presently.

The next point is that the degree of tonus affects the lumen of the vessel. Any over-tonus—hypertonus as I have called it elsewhere—necessarily means diminution of lumen. You cannot have the muscular coat contracting without a corresponding constriction of the tube it embraces. This further point may, I think, be accepted as incontrovertible.

BLOOD PRESSURE AND HEART POWER.

We turn now to the question of blood pressure or arterial tension, and the effect which alterations of tonus have upon it. Whenever the question of blood pressure or arterial tension is raised the driving power of the left ventricle at once becomes the factor of supreme and determining importance. In this connexion the heart of average vigour has a fairly wide margin. The existence of this reserve of force is fully recognized as enabling the average organ to respond to the strain of sudden and of varying degrees of physical effort. This margin of reserve, however, diminishes with the advance of years in the vast majority of people. This is the explanation of the breathlessness which impedes and finally limits within

narrow bounds the physical activities of advancing years. You must not regard my insistence on this as the assertion of a mere platitude, for I shall show you presently that it has a practical significance and application which is by no means universally recognized.

THE RELATION OF TONUS AND TENSION.

As to the relation between tonus and tension, there is what may be called the physiological aspect and the clinical aspect. The physiological aspect is based on experimental data which are doubtless correct so far as they go, and so far as they go they guide us, but they do not cover the whole ground, although this is what sometimes seems to be claimed for them. In the average normal or healthy animal the arterial tension is mainly determined by two factors—the power of the left ventricle and the measure of contraction, that is the tonus, of the arterial walls. We have postulated an average arterial tonus, and we may postulate an average ventricle power, both tonus and power possessing a considerable margin. Experiment shows that if the tonus is artificially increased the blood pressure or arterial tension at once rises. The explanation is that any diminution in calibre of arteries acts as an obstruction, and requires more power on the part of the heart to carry on the circulation. This increase of power is available in the average heart in virtue of its margin of reserve power in the first instance. If continued long enough it leads to hypertrophy of the ventricle.

This broad physiological law, that if the arteries contract the blood pressure rises, has quite properly been accepted by the clinician, but it has been applied with much too little reserve in practical medicine. One of the curious outcomes of this has been that there is often a want of clearness in the conception of and distinction between the terms "tonus and tension" and "tonus and blood pressure." I certainly frequently find the terms "high pressure" and "high tension" applied to a radial artery which is only unduly thick in its wall either from excessive contraction or from permanent structural change, and the use of the term is justified by the argument that such changes in arteries lead to the raising of blood pressure.

This brings me to the practical application of my argument and it is this: That, while the broad physiological law is correct, so long as you are dealing with average people in early life, it is not true, or only partially true, of people below average vigour, and of many persons after middle life.

There is, however, an important number of persons in whom the physiological law is strictly and clearly illustrated through middle life, and on into later life, if they escape for so long a time the vascular dangers to which they are continuously exposed.

If we seek, as we all do, to interpret aright, and thereby to utilize aright the indications afforded by the phenomena of the circulation in individual cases, we must keep rigidly apart the two factors, (1) the arterial contraction or tonus, and (2) the blood pressure, and yet both must be recognized and estimated. A third point is in some respects the most difficult; it is the determining whether a thickened artery is thickened from hypertonus or from arterio-sclerosis. The age of the patient and the character of the symptoms help in the differentiation. For practical medical work it is indeed of the utmost importance to be able to recognize hypertonus, and it is especially, often urgently, important when we are dealing with people in and beyond middle life; not that it is confined to that period of life, for it occurs frequently in early life, but adaptability, elasticity, and endurance are greater then, and there may be no symptoms complained of.

HYPERTONUS: VISCOSITY.

If I have made my argument so far clear, it will be understood that hypertonus in vigorous people leads at once to heightened blood pressure and raised arterial tension. This active response on the part of the heart to increase of peripheral resistance to the blood flow may be continued into and beyond middle life. Degenerative changes in cerebral arteries are often present—indeed, are prone to be associated with this ready production of increased tension. This combination occurs in the persons who supply the examples of early cerebral haemorrhage. But hypertonus also occurs in people whose hearts are not vigorous, and such people may or may not have degenerated arteries. In them thickened arteries are associated with a low blood pressure, for they have no sustainable margin of reserve in their hearts. A temporary excitement gives increased cardiac action and raised blood pressure, but as soon as the one subsides the other disappears.

This point of the existence of hypertonus without increased blood pressure is dealt with more fully later.

These thickened vessels present another difficulty; the thickened artery is looked upon as arterio-sclerotic, and this is not uncommonly regarded as a condition requiring no further serious consideration—as a structural change for which we can do nothing.

I need not dwell upon the prevalence of thickened arteries during and after middle life; but I desire very strongly to impress upon you the fact that those thickened vessels may owe much of what may appear to be structural thickening to what I have called hypertonus. Whatever of true structural thickening may be present cannot be removed, but whatever thickening is due to hypertonus can be removed.

The acceptance of the occurrence of hypertonus as I have defined it, and the recognition of it, either with or without a raised blood pressure, enables us to do more for our patients than perhaps the recognition of any other single phenomenon in practical medicine.

I must, however, support this thesis by further fact and argument. Take a simple and commonly recognized type of case—the person with good appetite, the hearty and large feeder, who partakes freely of proteid food two or three times a day and drinks wine, spirit, or malt liquor regularly. When a person of this type gets a “bilious attack” it is well known that his arterial tension is raised; but this arterial tension is usually, probably always, associated with hypertonus. Mark the significance of this latter; it means that the heightened tension is not due to deleterious substances acting directly on the heart, stimulating it to more powerful contraction, with a consequent over-filling of the arterial system. The primary and initial influence is on the vessels. The arteries are irritated or stimulated, and become hypertonic; this calls out increased cardiac action, which is in reserve, and increased tension results. Does this point require to be argued? Is the truth of it not apparent as soon as it is expressed?

One argument appeals to us—the effect of mercury on the condition is universally recognized; it promptly lowers the arterial tension, not by acting on the heart and diminishing its vigour, but by relaxing the hypertonus. I need not at present discuss how the mercury has this effect. It is the power of producing this effect that makes the drug a dangerous one to give in many conditions; it is this which makes it specially dangerous in hypertonic vessels where the blood pressure is not raised, and where the heart has largely lost the power of easily and promptly adapting itself to vascular changes.

Pulse tracings of hypertonic arteries and of the same arteries after the hypertonus has been removed show that there is not only a marked difference in the swing of the needle of the sphygmograph, but that the hypertonic vessel has a smaller diameter than the same vessel when its hypertonus is relaxed. The two tracings given below illustrate this. In my opinion this repeated or continued hypertonus leads to the hypertrophy of the middle coat, which is one of the changes present in arterio-sclerosis, as that term is used in this communication.

1. Tracing from hypertonic artery; diameter of vessel 2.2 mm.

Let us look at another explanation which has been suggested of the phenomena in such cases as I have just referred to. This suggestion is, that the deleterious substances in the blood heighten its viscosity, and correspondingly impede its flow through the capillaries; that this leads to a measure of damming back of the blood in arterioles and arteries; and that this again stimulates the heart to greater effort, the result being increased arterial tension. Whether the presence of such deleterious substances in the blood so modifies its physical characters as to impede its flow is, I venture to think, doubtful; that they act so as to produce hypertonus of arterioles and arteries I have no doubt. That they also lead to capillary contraction is, I think, most probable. What the substances are which so act is a question of great interest; but, whether they are to be expressed in

terms of uric acid, of purins, or of something else, must be left for the future to determine. There is, however, a sidelight from which we can take some assistance. I refer to our knowledge of the influence of the secretion of the thyroid and adrenal glands respectively on arterial tonus and tension.

THE INFLUENCE OF BLOOD CONDITIONS ON TONUS.

The first point I have sought to establish is the active response of the arterial wall to impressions and influences, which places it in a totally different position to that which it occupies if it is regarded solely or mainly as an elastic tube.

The second point—that blood conditions are amongst the influences which so act upon the arterial wall—I wish to examine more fully. We may, I think, assume it to be generally accepted that generous feeding, and the free use of alcoholic and malt liquors, lead to high arterial tension, especially if there is not full, free, and daily evacuation of intestinal contents. I include under the phenomena of arterial tension the important part taken by arterial contraction or hypertonus. This is the gross type, and I think it is more common in some parts of this island than in others; and it occurs without doubt more commonly in some classes than in others. This type is so prominent that it seems to me as if other types were so overshadowed by it as to be overlooked; and yet there are other types. One of these is found in persons who are moderate or even spare eaters and very moderate drinkers, but who in later life present sclerosed arteries, and from time to time an unsafe measure of arterial tension. This increase of arterial tension is in my experience always due to hypertonus. I have had the opportunity of watching for years a case of this kind in a gentleman, in easy circumstances, who ultimately died well on in the seventies. My main work for several years for this old gentleman was the restraining of a marked tendency to hypertonus, and along with that a corresponding raising of arterial tension. The case illustrated several points of great practical interest: the first I may mention was the marked effect the hypertonus produced on the brain. It is, indeed, the symptoms referable to the cerebro-spinal system which often lead to medical opinion and counsel being sought for in such cases. The patient to whom I am referring became unusually somnolent after meals; his speech became a little thick and blurred; but what annoyed him most was that he began to spell words incorrectly in his letters, a thing he had never done before; his gait also became slightly unsteady. In fact his condition at the time to which I refer, and some years before he died, was such as to suggest rapid cerebral degeneration. I was, however, satisfied that the symptoms were due to hypertonus of cerebral vessels curtailing local blood supply. I knew that in eating and drinking he was very moderate, as judged by ordinary standards, yet after careful consideration I strongly advised him to give up the glass of claret he took at lunch and dinner. Fortunately my patient and his wife, when they saw I was in earnest, at once fell in with my advice; and we were rewarded by the disappearance in a very short time of the symptoms which had given us so much concern; and along with this there was a corresponding reduction of hypertonus and arterial tension. I mention this case because it is illustrative of a type, and because it was my privilege to watch this patient very closely, and to be allowed to regulate his living as I deemed expedient.

2. Tracing from same artery after relief of hypertonus; diameter of vessel 2.4 mm.

There was in this case a very marked readiness on the part of the arteries to tighten up a little, and for long periods a small quantity of potassium iodide was sufficient to control it. After the continuance of such a period there would be some deviation from the ordinary austerity, some proteid added to the dietary, or some small measure of alcoholic fluid taken at lunch or dinner, and as sure as this occurred there was a threatening of the former symptoms. The total quantity of proteid—and, indeed, of every kind of food taken—was

relatively small; and the same was true of the amount of wine or of spirit. A more regular and temperate man could hardly be found, and yet he was constantly threatened with dangers more associated with the opposite habit. How are we to look at a patient of this kind? Are we to be content to regard him, as is not unusual, merely as an old man whose "strength" has to be kept up? This view, when acted upon, ends speedily in disaster. I submit that this man showed in later life in a concrete form what had been his physiological standard during life. There was in him a physiological barrier to his being a large feeder or a big drinker. We all know such persons—persons who cannot eat big dinners daily, or take alcoholic liquor of any kind regularly save in small quantity. This may have no more virtue in it than the declining to do what experience soon teaches brings about unpleasant results. The barrier is really a physiological one; the organs concerned are not capable of dealing with the amount or kind of work thrown upon them, and auto-intoxication or toxæmia of one kind or another results. Fortunately it is not a question of first-rate importance what the precise composition of the auto-intoxicant is, but I believe it is of great importance that we should recognize that, whatever its name, it has the power of acting upon the blood vessels—tightening up arteries and arterioles, modifying the capillary circulation, and affecting renal function. Neither is it necessary to dwell upon the varied symptoms which can be manifested during the intoxication. Recent observations suggest that we may have to alter what has hitherto been our standpoint as to what the primary and initial defect may be, in some at least of our patients. The possibility that thyroid secretion, on the one hand, or, on the other hand adrenal secretion, may be materially influenced by the quantity of proteid in our food puts the whole matter a step further back; for were this fully established it only gives a fuller explanation, a more detailed account of the physiological steps by which the auto-intoxication is brought about. In the patient to whom I have particularly referred it may be that the small doses of potassium iodide given to him acted by helping the thyroid secretion to counteract the adrenal secretion, a too active condition of the latter being, perhaps, the constitutional basis for the prolonged continuance of phenomena which ended in arterial thickening, in kidney changes, and in a final upsetting of equilibrium that we endeavoured in vain to restore. Be this as it may, it is to my mind clear that it is primarily to the feeding and to the gastro-intestinal tract that we have to look for the explanation of the vascular phenomena and the conditions associated with them, with which I am dealing. I think it probable that the kind and quantity of food taken may produce evil effects owing to deficient thyroid secretion; but also that under corresponding conditions the adrenal secretion may be over-stimulated. I can, however, hardly believe that all auto-intoxications following upon dietetic errors act through those special glands, although the cases where vessel changes are pronounced may always have a close relationship to a constitutional, congenital, or inherited tendency on their part. We fortunately can wait for the final solution of those most interesting problems; and meanwhile, we can move forward.

HYPERTONUS WITHOUT INCREASED BLOOD PRESSURE.

Now I wish to draw your attention to a class of person in later life who also has sclerosed arteries, whose arteries still retain a measure of adaptability, and are not mere tubes retaining a degree of elasticity, but in whom hypertonus does not lead to a rise in blood pressure. I deliberately distinguish this class of case, and it comprises a large body of persons, although to some of my clinical friends my position appears to be heterodox. I am physician to an institution where I see scores of old and feeble people of the respectable artisan class. It is half a paying home and half a hospital. The feeding is simple and spare. From my observations there, as well as in other directions, there can be no doubt that hypertonus occurs without increase of the blood pressure; that is, no increased sustaining of the column of blood in the hypertonic artery, but the reverse. This hypertonic condition of arteries leads to a number of interesting manifestations in those old people; it may give rise to insomnia and nocturnal restlessness; it may render an inmate who was moving with freedom about a ward yesterday unable to get out of bed to-day without being lifted or greatly helped by the nurse; and it may lead to hemiplegia or to aphasia. When these things occur the diet is carefully regulated, the gastro-intestinal condition is attended to; drugs are given in some instances to relieve arterial spasm; whisky

is given in some, in others strophanthus. These cases return to their ordinary routine in due time as a rule. The condition is most commonly the result of gastro-intestinal auto-intoxication due to error in diet. The quantity of food taken is not large as measured by ordinary or popular standards, but it is over the limit for the individual, and the boundary is easily passed. Practically it is not of much importance what particular share of responsibility can be attached to the various organs for the production of the symptoms; but it is of essential importance that the condition be recognized as an auto-intoxication, caused by over-feeding or wrong feeding, by the absorption of the products of intestinal decomposition, or by the slow eliminating power of deteriorated kidneys; these act and react on one another, and the two last can in a measure be influenced by the first, can even be determined by it. The recognition of the condition puts the remedy in our hands, although it is not always easy, or indeed possible, to apply it thoroughly, owing to popular ignorance and prejudice.

The interest of these cases is not, however, confined to this aspect of them. As has been already indicated, the arterial hypertonus does not lead to an increase of cardiac power; in fact, it is often associated with, or followed by, a definite threatening of heart failure. It becomes, indeed, in some of these cases, somewhat of a fine art to determine which is the best line of treatment, or, it would be more correct to say, the lines of treatment to adopt, for diet, gastro-intestinal condition, hypertonus, and threatening cardiac failure are collectively presented for consideration and decision; and that in persons who are at best living on a narrow debit and credit account from the physiological or metabolic standpoint. The ledge is sometimes so narrow that even the measures adopted may precipitate the end. If the art is a fine one, is it not art growing out of knowledge which makes our work so attractive? Sometimes it is asked, "Is the result worth the trouble?" To the physician it certainly is, for it gives him an understanding which is an invaluable possession, while the acquiring of the skill is an education which moulds every side of his professional work. Other aspects of the question I readily leave for others to determine.

GASTRO-INTESTINAL DISORDERS CAUSING HYPERTONUS.

I seek in the next place to refer more directly, than I have so far done, to the influence of gastro-intestinal disorder, including of course food, in causing arterial changes. I have referred to the big feeder and free drinker, with his recurring attacks of "biliousness," and to the condition of his vessels. He often dies in comparatively early life from the result of vascular changes in his brain or kidneys. Now this gross type is readily accepted in all its bearings, but there is often a failure to recognize that there are minor degrees of the same class of phenomena as those presented by it. I have also referred to phenomena of this nature in persons with already sclerosed vessels. The point I would now specially submit to you is that in many persons in early and middle life suffering from one or other form of gastro-intestinal functional disorder—some form of dyspepsia or of constipation—there occurs an auto-intoxication of long duration, but relatively mild in its manifestations. It may manifest itself by a variety of subjective symptoms; it may be recognized by urine reaction. In a considerable proportion of cases of this class arterial hypertonus is to be noted. Very commonly the thickened arterial wall is regarded as a sclerosis occurring in early life, while as a matter of fact it is a hypertonic vessel and not a permanent thickening. That it is so is shown by the disappearance or diminution of the thickening under treatment, a result which could not follow were the thickening due to hyperplasia or hypertrophy of the tissues of the arterial coats. I am not prepared to say that all gastro-intestinal morbid conditions cause hypertonus, but when they do I believe we are face to face with the first step in the development of the sclerotic changes which are so common in middle and later life, and which materially determine the duration of individual life. I have seen vascular degeneration in persons who in early manhood had been martyrs to dyspepsia; such marked degeneration of the cerebral as well as of the general arterial system that by the age of 60 years repeated local brain softenings had rendered them physical and mental wrecks. This in persons who were moderate eaters, practical abstainers, and who neither in themselves nor in their children suggested any suspicion of syphilitic taint.

A measure of indigestion and constipation rank in the public mind as such commonplace conditions that the one is accepted as an inevitable accompaniment of life in the present time, with its buzz of mental activity and strain, and the other as only demanding alleviation after prolonged intervals of neglect by a dose of cascara or of some widely-advertised liver pill. It is my conviction that this combination of conditions, common though it be, leads slowly, insidiously, and surely to vascular changes in a great number of people.

The problems surrounding indigestion and constipation are too large to be entered upon fully at present; they are intimately bound up with the problems connected with food, and the general attitude of the medical and the lay mind to food problems. It may, however, be noted that the raising of the standard of comfort has, as has been shown by official returns, led to a great increase in the consumption of flesh amongst all classes. Along with this has unfortunately come the fixed popular idea that flesh is the only strength-giving food, that it is the essential part of the daily dietary. Now, there is no question that an excess of proteid food throws a strain upon excretory functions, it does not as a rule favour free bowel action; it becomes, in fact, a potent source of auto-intoxication. In some persons living on ordinary and very simple food, yet suffering from marked and even grave symptoms of auto-intoxication, you will find that by materially reducing the proteid element and increasing the carbohydrate you remove those symptoms, and in addition you will arrest arterial changes and remove much of what appeared to be established. You are often in such cases met with the cry that you are reducing strength. People seem to have forgotten that the stalwart men who were to be found on every farm in Scotland fifty years ago were reared and maintained on carbohydrate food and milk for breakfast, dinner, and supper. I fancy constipation was not then a common complaint, and judging from some of those who remain, vascular degeneration was not an early development.

Indigestion, constipation, and feeding are so interwoven that we hardly see their relations until we induce some of our patients to submit to a revolution in their diet habits. The reduction in quantity of the more pure proteid food with an increase in the more pure carbohydrate food is the change which has to be effected. The latter—that is, the carbohydrate food, is mainly provided by the cereals. The true place for cereals in a rational and healthy dietary begins to be recognized in some measure, if we can estimate it by the number of these introduced to the public within the last few years. It is, however, a matter for surprise that all the enterprise in supplying us with novelties in the way of ready or readily cooked, or of partially digested, cereals is supplied by our friends across the Atlantic.

REFERENCES.

¹ *Lancet*, June 1st, 1901. *Encyclopædia Medica*, vol. xiii, article Vessels.

ABSTRACT OF

Two Lectures

ON THE

ESSENTIAL SIMILARITY OF INNOCENT
AND MALIGNANT TUMOURS.

*Delivered before the Royal College of Surgeons of Edinburgh
on March 4th and 11th, 1904.*

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AFTER some introductory remarks Mr. Cathcart pointed out that the subject of the lecture had an important bearing on many of the problems now under discussion with regard to malignant tumours. He meant the title to indicate that there was no hard-and-fast line of demarcation between innocent and malignant tumours. He held that the differences were those of degree and not of kind, and that the contrast was not even so great as might be drawn between the characters of a benevolent man and one who had given himself up to crime. While many pathologists had taken up the position which Mr. Cathcart proposed to defend, long before he did, there were still a large number who opposed the view either directly or indirectly, and there were possibly a larger number who were unable to make up their minds definitely on the subject. It seemed, therefore,

worth while to try to concentrate attention on this proposition and to bring all the arguments in favour of it together, so as if possible to establish it as a recognized principle in pathology. Instances were given of pathologists opposed to and of those uncertain as to this view, while those in favour of it would be mentioned in the course of the lectures. In order to have a clear understanding of what was meant by innocent and malignant tumours reference was made to Mr. Bland-Sutton's discussion of the subject in the recent edition of his book on *Tumours*. The key to the difference is, as he expressed it, that "the baneful effects of innocent tumours depend entirely upon their environment, but malignant tumours destroy life whatever their situation." Mr. Bland-Sutton describes the characters of malignant tumours under the following five heads: (1) Infiltration, (2) gland infection, (3) recurrence, (4) dissemination by the blood, (5) inevitable destruction of life. If these characters, which are accepted by all, are analysed, the lecturer thought they might be stated in tabular form as follows:

Essential Feature of Malignant Growths.			Type retained.
{	Growth...	...	(a) Indefinite size.
	Without restraint	...	(b) Local dissemination.
			(c) Local infiltration.
			(d) Gland dissemination.
			(e) Blood dissemination.

Recurrence, due either to b, c, or d.

An explanation of this table may be made shortly as follows: Malignant tumours follow the structure of the tissue or organ in which they originated, in the majority of cases so closely that the similarity is quite recognizable, both in the primary and secondary deposits. This may be taken as sufficient illustration of the view that they are modifications of growth. But that they are not under the restraining influence that the normal tissues are, is indicated by (1) their continuous growth, leading to an "indefinite size." This local overgrowth many call "local malignancy," and although some people object to the latter expression, it seems a perfectly satisfactory one to explain one of the ways in which these tumours "inevitably destroy life." (2) By their cells leaving the original mass so as to produce secondary growths in the neighbourhood, even when the original mass seems encapsuled. This is what is meant by "local dissemination," and the same breaking-free tendency is shown by (3) infiltration of the surrounding tissues where there is no capsule, and by (4) dissemination through the lymphatic system, or (5) through the vascular system in the epithelial and connective-tissue types of malignant tumour respectively. Recurrence could in some cases be traced to local dissemination where a capsule was present, in others to infiltration of the surrounding parts where the knife had passed through the actual extension of the tumour, or in others to involvement of glands.

The lines of argument which were to be followed out in support of the view taken by the lecturer were indicated as:

1. The presence of gradation in malignancy as shown in a series of specimens selected and arranged to illustrate different degrees of malignancy in the same kind of tumour, taken from different individuals.

2. Transformations in character, where a tumour of a certain kind had changed from innocent to malignant in the same individual.

3. Combinations of characters where tumours otherwise innocent have shown certain characters which are generally believed to belong only to the malignant forms.

These lines of argument were supported by appeals to specimens examined both by the naked eye and microscope, and illustrated by lantern slides thrown upon the screen; by reference to published cases, and by reference to published opinions of well-known pathologists. The greater number of the specimens illustrated were in the museum of the Royal College of Surgeons of Edinburgh, some had been selected from other museums in Edinburgh, and one from the Hunterian Museum in London. It had been impossible to cover all the ground that might have been covered, but it was hoped that the specimens were sufficiently illustrative for the purpose in view.

I. GRADATION IN MALIGNANCY.

The gradation series specially worked out were those of bone tumours. The museum of the Royal College of Surgeons of Edinburgh is relatively rich in this class of tumour, and the latter is also one which lends itself to lantern illustration.

(1) Cartilaginous tumours formed the first series; innocent forms were shown first and their microscopic characters illus-