

HEART LESIONS: SOME POINTS IN THEIR DIAGNOSIS AND TREATMENT.¹

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FROM the time of my promotion to the senior staff of the Royal Infirmary 81 cases of heart trouble have come under my observation. Two cases were admitted moribund and no post-mortem examination was obtained in either case. There were four examples of congenital disease; no points of novelty were noted. Mitral lesions were present in 39 cases; 13 presented the signs of stenosis in addition to those of regurgitation, and five, moreover, had definite evidence of adherent pericardium. In 31 out of the 39 cases there was a definite history of rheumatism. In this group three deaths occurred, two cases of regurgitation, aged 50 and 56 years, and one of stenosis, aged 21 years. Aortic regurgitation was found in seven cases; three of these were under 30 years of age and had a definite history of rheumatism; the remaining four were over 30 years of age, two had suffered from syphilis, and two showed marked vascular degeneration. There was one death in this group, a young woman, aged 29 years, who died suddenly; a post-mortem examination was refused. Combined aortic and mitral lesions were found in eight cases; four gave a history of rheumatism and four evidence of general vascular disease. The vascular cases were all in males over 40 years of age. There were two deaths in this group. The renal cardiac cases embraced 16 admitted with cardiac symptoms due to chronic renal disease. Six of these had on admission and discharge a mitral regurgitant murmur; three cases had a mitral systolic murmur on admission which disappeared and two had an aortic diastolic murmur on admission which disappeared. Four deaths took place in this group all over 45 years of age. Five cases of infective endocarditis came under notice; all died and were confirmed by post-mortem examination. In one of these the rise in temperature occurred irregularly and was only detected by taking the temperature every two hours, an important point. In another stenosis of the pulmonary valve was found, the infective lesion being a patch of acute vegetative disease of the pulmonary artery proximal to the bifurcation. This last case apparently followed an attack of pneumonia, but the infective lesion was streptococcal in nature (organisms were cultivated from the blood during life). In this group all the cases succumbed. In this rapid *résumé* the preponderance of mitral cases, nearly half the number, and the importance of rheumatism in their causation stand out prominently. These cases offer a wide field for discussion and if I must content myself with noting only a few points the limitation is made with good reason.

The possessor of a heart lesion may be absolutely unconscious thereof; on the other hand, he may present many symptoms which can usefully be classed as direct and indirect. In the former group we find various subjective sensations—palpitation, dyspnoea, and alterations in the cardiac rhythm; in the latter symptoms which for the most part are attributable to passive congestion of the various organs—lungs, liver, kidney, or brain. Yet another source, embolism, may give rise to symptoms far removed apparently from the primary lesion. It is unnecessary for me to detail the various symptoms but I shall touch upon a few to emphasise the difficulty which we shall often meet in our work.

Let us consider for a moment the subjective sensations that may be present. As you all know, they vary from the merest præcordial discomfort to the agony of angina pectoris. I should say that given a heart lesion the severity of the subjective trouble bears often absolutely no relation to the gravity of the lesion. The fatal case of aortic disease was attended with practically no pain at all. But a still more important point is this, even severe anginal pain causing collapse may occur without there being any actual heart lesion at all. These cases are not seen in my experience in hospital practice, they occur in better-class subjects, often neurotics, who are overworked, either mentally or physically. Close inquiry often elicits a history of irregular

feeding and possibly excess in alcohol and tobacco, especially the latter. Rest, careful dieting, and a holiday work wonders. We are all familiar with the abnormal sensations in the chest due to gastro-intestinal disturbance and I have a vivid recollection of a patient in the ward of the late Dr. A. Davidson whose "mediastinal tumour" was removed by a few doses of Gregory's powder. A much more serious difficulty I can illustrate with a case.

CASE 1.—A man, aged 48 years, was sent to me for severe anginal attacks. His medical adviser failed to find any condition to explain them. I in my turn found the heart and vascular system quite healthy. The man was kept under observation and eventually definite evidence of nerve-root implication was obtained and finally the case was cleared up by the appearance of œdema of the left arm. A necropsy showed a malignant growth starting from the upper dorsal vertebrae.

Palpitation is another symptom which may be considered for a moment or two. Perhaps of all heart symptoms it is most often unaccompanied by heart lesions. Anyone conscious of his heart's action may be said to have palpitation; the heart's action may be unaltered or may be irregular. In either case careful attention must be paid to the apparent exciting cause. When due to severe exertion it is a physiological phenomenon, but when it appears with slight exertion some underlying state can usually be detected, such as neurosis, chlorosis, or heart trouble. It may also appear, apart from exertion, after the intake of food or emotional disturbance, and its association with gastric disturbance is well known. By far the most frequent variety is the nervous one, with a weak nervous system, whether inherited or acquired. Palpitation appears under the strain of the body changes at puberty and the menopause, or under physical or mental overwork. Similarly many cases of tea and tobacco palpitation have a nervous basis.

Is nervous palpitation due to sympathetic stimulation or vagus inhibition? In many cases it is impossible to say. In some, however, the associated vascular changes justify the implication of the former. Two cases will illustrate this.

CASE 2.—A young male neurasthenic came to the infirmary suffering from palpitation which I found to be associated with marked pulsation in the vessels, notably the abdominal aorta. This condition has been termed by Dana pulsating neurasthenia.

CASE 3.—In private I saw a young male adult who with severe attacks of palpitation had marked tachycardia and wasting. Three months' rest produced a complete recovery which has been maintained for over three years.

I always teach that the symptoms of chest mischief may be due to lesions of practically any of the thoracic organs, but it is also important to note that chest lesions may give rise to symptoms pointing elsewhere. I may be allowed to give some examples not limited to heart cases. Some years ago I collected a series of cases of pneumonia and found that one-third of the number began with vomiting. I should add that my cases were all adults. A striking case was in my ward last year.

CASE 4.—A man, aged 44 years, was admitted for cough and pain in the chest. He had suffered from a septic finger. The temperature was a septic one, a well-marked leucocytosis was present, and he had signs pointing to effusion in the left chest, deficient movement, absent vocal fremitus, dulness, and feeble bronchial breathing. A needle drew pus but operation found the pleural cavity obliterated. A necropsy a week later revealed a suppurating bronchopneumonia secondary to an aneurysm pressing upon the left bronchus.

An important case with a direct bearing upon the point I wish to make is the following.

CASE 5.—A woman, aged 46 years, was sent to me with a history of hæmoptysis and some wasting. Her medical adviser had found evidence of consolidation at the apices. I corroborated his finding but discovered further that she had very definite mitral stenosis. No tubercle bacilli were found in the sputum and the further history has shown the lung lesion to be secondary to the heart state.

CASE 6.—Another case I well remember was sent for examination purposes some years ago as one of aortic aneurysm. I am happy to say that our Liverpool students who saw it recognised it as a case of fibroid contracting lung exposing a normal vessel.

A final case in this connexion:

CASE 7.—A woman, aged 27 years, was sent to my

¹ A paper read before the St. Helens Medical Society.

colleague, Mr. Rushton Parker, suffering from hæmaturia of recent appearance. No local pelvic cause was found and I was asked to see her. The onset had been attended by sharp pain in the loin and her heart showed definite evidence of mitral stenosis. The hæmaturia was then of embolic origin and disappeared after a week's rest in bed. I might say the patient was not aware of her heart trouble.

These are a few striking cases and I have no doubt you could multiply examples from your own experience. The point which I wish to make is then that nothing but a careful physical examination will enable us to disentangle the various chest lesions.

I have often been struck with the method of heart examination practised before inducing anæsthesia. An ear applied directly or with the aid of a stethoscope settles the question of heart soundness in an instant. The matter, however, is not nearly so simple. I suppose the diagnosis is based on the absence of a murmur with a regular rhythm. Now when valvular trouble is present good compensation makes it no bar to anæsthesia and in my earlier days I have given even chloroform in such cases without mishap. On the other hand, a murmur is not sufficient evidence of valvular trouble. It would lead me too far to discuss the whole question but I would remind you that a murmur depends largely upon the speed of the blood current. I am sure that some murmurs heard when the heart is beating very rapidly in excitable subjects are due to this factor alone. The opposite pole will be accepted, I think, by all—viz., that when the heart's action becomes weak murmurs often disappear; this I take it is the probable explanation of the murmur being absent in some cases of mitral stenosis. Again, when a murmur is present the question will often arise, Is it functional or organic? I am accustomed to teach that with no enlargement of the heart downwards or to the left, no accentuation of the pulmonary second sound, with the left ventricle first sound sharply defined, and a systolic murmur loudest at the pulmonary area, then in the presence of anæmia a diagnosis of hæmic murmur is justifiable. However, in some cases a mitral murmur is heard with an accentuated pulmonary second sound and yet we are in doubt. I have just had an interesting case in my ward which will show the difficulty that at times may arise.

CASE 8.—The patient was a girl, aged 19 years. She was anæmic but nutrition was fair. The temperature was 101° F., the respirations were 24, and the pulse was 110. There were some redness, tenderness, and swelling of the right foot. The blood count gave the following result: hæmoglobin, 60 per cent.; red cells, 4,800,000 per cubic millimetre. The apex beat of the heart was in the fifth space, three and a quarter inches from the median line; it was strong, regular, excited; the dulness was within normal limits. The left ventricle first sound was sharp and followed by a systolic murmur, which was not conducted to the axilla. The pulmonary second sound was accentuated.

It is generally accepted that some cases of functional murmur are heard best at the mitral area and if weakness of the muscle ring be accepted as the cause thereof the increase in the pulmonary second sound is readily explained. I decided in this case, although rheumatism was present, that the murmur was functional and I did so because the first sound was so well defined. Within a week the murmur had vanished, so that the diagnosis of relative incompetence was probably correct.

The variety of valvular disease in which murmurs are most often absent is mitral stenosis. The following considerations will guide us in such a case. 1. Evidence of an enlarged right heart: displacement of the apex beat to the left, systolic heaving of the sternum with epigastric pulsation, and extension of cardiac dulness to the right of the sternum. I assume that the lungs have been examined. 2. A diastolic shock in the third left space will give evidence of high tension in the pulmonic circulation. Good observers have stated that a presystolic shock can in some cases be made out in the upper and left part of the cardiac area. I cannot say that I have ever seen it and as it is ascribed to the pulsation of the auricle I do not expect to see it, for I have never made a post-mortem examination in which the left auricle has formed part of the anterior surface of the heart. 3. A presystolic thrill may be present even in the absence of a corresponding murmur. The shock of the closing mitral valve may be detected as an abrupt sensation synchronous with the first sound and by Bard and Cassan is considered to be an absolute sign of mitral stenosis. 4. The left ventricle first sound is often altered in characteristic fashion, becoming

hard and sudden—I call it “thudding.” At the base the pulmonary second sound becomes markedly accentuated and persistent reduplication of the second sound is a good sign of the lesion. 5. Preceding the first sound at the apex is sometimes heard a dull rumbling sound which may end in a murmur before the first sound becomes audible, the *roulement diastolique* of French writers. The pulse is usually small, regular, and of fair tension. I have a case in my female ward at the time of writing which shows practically all these points. Yet another case:—

CASE 9.—The patient, a female, aged 26 years, was in my ward in January, 1904, with mitral regurgitation. She was readmitted on March 20th with marked loss of compensation, as shown by œdema of the legs, some ascites, right hydrothorax, with orthopnoea and cyanosis. The mitral murmur was absent on admission but became audible in the course of 24 hours after a full dose of digitalis.

It is obvious, then, that the most careful physical examination is necessary to make an accurate diagnosis in chest cases. The scheme which I adopt for the heart is the following:—
1. Inspection of the præcordium. 2. Apex beat: position, extent, strength, character, rhythm, thrills, mobility, abnormal impulses. 3. Heart dulness: change of absolute dulness with full inspiration. 4. Heart sounds: strength, character, relative intensity, rhythm; added sounds. 5. Murmurs: site of greatest intensity, time, lines of conduction, and character. The examination is completed by the investigation of the arterial pulse, venous pulse when present, also liver and capillary pulses, and further assistance is obtained by considering the symptoms and physical signs in the organs secondarily affected. In this way we can usually determine the existence of a heart lesion, but, as Peter has well said, this is merely an artistic satisfaction; a musician could arrive thus far; it takes a sound medical practitioner to make a diagnosis on which prognosis and treatment may be based.

Time will not allow me to enter into the causation of heart disease but the nature of our problem may be illustrated by a case.

CASE 10.—A male, aged 50 years, was admitted into Ward X. for pain in the chest, dyspnoea, and cough. Physical examination detected an enlarged heart; the apex beat which was displaced downwards and to the left, was slightly diffuse, strong, heaving, and regular. There was some dulness in the inner part of the second right intercostal space. The left ventricle first sound was prolonged and muffled; the aortic second sound was musical and accompanied by a short “whiffy” diastolic murmur. The pulse was rather short (collapsing) and the radial artery was greatly thickened and tortuous. The diagnosis of hypertrophy of the left ventricle with a little dilatation, dilatation and atheroma of the aorta, and slight aortic regurgitation was readily made. It was further noted that the urine was of low specific gravity (1007), although deficient in amount, and contained a trace of albumin. The history revealed some renal dropsy, puffiness of the eyelids in the morning and of the ankles at night, of some duration, and, further, that for some years the patient had been obliged to get up at night to micturate. It was evident that the man had chronic renal disease and we had to decide upon the nature of the aortic valvular trouble. Murmurs in renal disease may be hæmic, due to relative incompetence, or the result of actual valvular changes. A diastolic murmur is practically never hæmic in origin, so that we were left with the two latter. It was quite possibly a relative murmur inasmuch as there was evidence of dilatation of the aorta but, so far as I know, there are no physical signs to help us in our decision. The course of the case will often decide and in this case it did so. The patient was kept in bed, his fluid intake was lessened, and in the course of a few days the murmur disappeared; it was therefore a murmur of relative incompetence and not one due to valvulitis.

It would be impossible to notice all forms of heart lesion in which difficulty arises but I should like to mention one other—viz., adherent pericardium. I do so for two reasons—one being its importance in prognosis and the other its bearing upon the diagnosis of mitral stenosis. This latter point was impressed upon my mind a good many years ago by the following case:—

CASE 11.—A young woman, whose age I have forgotten, was in the infirmary with evidence of heart failure, with presystolic thrill and murmur. Ascites was out of proportion to the œdema of the legs. She died and the post-mortem examination revealed general adhesion of the pericardium,

with calcification and absolutely no mitral stenosis. The specimen is in the Liverpool University museum. Since that time I have learned to pay less attention to murmurs and more to the other signs of valvular trouble and it has repaid me.

The point which I want to emphasise is this. In a valvular case, especially one of apparent mitral stenosis, always look for the evidence of adherent pericardium and be cautious in affirming the presence of mitral stenosis when such evidence is present. Inflammation of the myocardium and pericardium, pancarditis, is much more frequent than is usually believed. In my opinion the diagnosis of adherent pericardium can only be made when there is thickening in the mediastinal tissue, mere adhesion of the pericardial layers giving rise to no symptoms of any diagnostic value. It will be readily understood, therefore, what an important bearing mediastino-pericarditis will have on prognosis. It is, however, often a difficult matter to speak positively about. The points are as follows: systolic retraction of the intercostal spaces in the region of the apex of the heart; systolic retraction of the left lateral and posterior part of the thoracic wall; diastolic rebound in the lower præcordium; non-alteration of absolute cardiac dulness with respiration; fixity of apex position with alteration in decubitus; diastolic pulsation in the veins of the neck; and pulsus paradoxus. The fixity of the apex site is perhaps the only absolutely unequivocal sign of the condition. The disproportionate amount of ascites is a sign of some importance especially in young subjects, and often perihepatitis is found in association with it, the Zuckerguss Leber of the Germans. My old teacher, Dr. F. T. Roberts, taught me that the apex beat often had a curious character, which he aptly termed embarrassed, and I have found this sign of service.

The character of the cardiac enlargement is often of service in deciding how far any single lesion will suffice in satisfying the probabilities of causation. One or two points are of service in considering this. When the myocardium is weak dilatation rather than hypertrophy is the result of any increased work thrown on the heart and may if the strain be sudden and the heart is already diseased lead to fatal cardiac failure. Some years ago I saw a case with Mr. W. T. Clegg of Liverpool and within a few days was summoned to a second by him. Both were cases of gall-stone; the first had a sound heart and recovered and the second, who was the subject of mitral disease, died before I reached the patient's house. Potain taught that the tendency to syncope attending gall-stone colic was associated with dilatation of the right heart, and François Franck has shown experimentally that strong stimulation of the bile-duct causes a reflex rise in the pulmonary blood pressure. In this way the difference in the issue of the two cases becomes clear. Another point of value is the general rule that when a cavity of the heart is subjected to strain during its diastole dilatation is the primary result. On the other hand, when the strain occurs during systole then hypertrophy follows. In the first case, if time be allowed hypertrophy follows, and in the second sooner or later dilatation. For example, the high tension of chronic renal disease opposes the systole of the left ventricle and we find typical hypertrophy; in aortic regurgitation the strain meets the ventricle in its diastole and dilatation follows. This, with the secondary hypertrophy, gives us the well-known *cor bovinum*.

Before considering the question of treatment in heart disease which I shall limit to valvular lesions I must say a word or two on prognosis. There is little to be gained by tabular statements as to the gravity of the various valvular lesions, each case must be gauged on its own merits. Aortic regurgitation is usually regarded with great dread as a cause of sudden death. In my cases I have only met this accident twice, and as one patient had septic endocarditis I should say, perhaps, only once. In this case, that of a young woman, aged 29 years, no necropsy was allowed and I cannot say whether any other lesion was present. I mention this because in another case of sudden death in aortic regurgitation in which as pathologist to the infirmary I made the necropsy I found that the cause of death was the rupture of an aneurysm of the intra-pericardiac aorta into the pericardial sac. Myocardial lesions, too, may cause sudden death.

The nature of the lesion must be determined in order to make the diagnosis, the history considered with care, and the examination of the patient may give the clue. I have a patient in my ward as I write with aortic regurgitation in whom an attack of pneumonia seems to have been the exciting cause. Post-pneumonic

valvulitis is said to be usually aortic in site. I might mention that, according to French writers—Troisier, Teissier, and others—cases of mitral stenosis occur in which the tuberculous toxin is the exciting cause. In only one of my 13 cases does this seem the probable sequence of affairs.

The degree of the lesion is the second point. In regurgitant lesions the presence or absence of the corresponding sound will help but the main guide is the alteration in size of the heart. It cannot be too carefully borne in mind that loudness of murmur is no guide to degree of lesion.

The compensatory changes come next. When we find them adequate to the new condition the bearing upon prognosis is obvious, but when they have failed the problem is a little more difficult. We have now to consider whether compensation has failed on account of some special strain or because the reserve force of the myocardium is finally exhausted. In some cases only the result of treatment enables us to decide this question.

Finally, the general history and condition of the patient and his hereditary tendencies will give assistance in settling, so far as human skill can do so, the outlook so important to the patient. I need hardly say what importance the social status bears in regard to this matter of prognosis.

To turn to the second division of my subject I find the following subdivision of cases of service in outlining the treatment: (1) compensation practically equal to the lesion; (2) compensation only equal to the lesion when the patient is at rest; and (3) compensation unequal to the lesion.

(1) Should the patient in the first subdivision be informed of his trouble? I believe myself that this is always a wise procedure, otherwise it is difficult to get the necessary mode of life carried on. In general terms we must warn him against excess of all kinds, mental as well as physical. It is not necessary to forbid exercise in moderation; cycling and so on are permissible as exercise but not in competition. If cycling be indulged in care must be taken not to struggle against head winds or up-hill. Moderation in the use of alcohol and tobacco must be enjoined, abstinence even being advisable. General dietetic and hygienic measures should be borne in mind. I might add that even in this stage in mitral stenosis there is the risk of hæmoptysis or embolism. The treatment I have found most useful in hæmoptysis is a free saline purge, with a dose of opium. Are such lives insurable? I suppose most offices would reject aortic cases but would accept mitral cases with an addition to the premium. For my own part I should not advise acceptance, but when such a case comes for report make it a rule to give a full report on the lines I have sketched and leave it to the office to decide. My reason for refusal is the impossibility of foreseeing accidents, such as infection.

If the patient is a female, what should be our advice, if asked, as regards marriage? The less common form, aortic trouble, is a bar to matrimony. Well-compensated mitral regurgitation cases often pass through pregnancy without trouble; in stenosis cases, although the same favourable course is possible, yet at times grave symptoms arise and notably attacks of tachycardia or acute œdema of the lungs. If pregnancy occurs in a case of mitral stenosis the patient should be kept under observation and the slightest sign of distress should be actively treated. Limited diet, free action of the bowels, with possibly bleeding in the more threatening cases, will sometimes ward off the necessity for terminating the pregnancy. Our advice should certainly be against marriage.

(2) In the second subdivision we find cases in which palpitation, præcordial pain, and dyspnoea are manifest on exertion, and in a more advanced degree some passive congestion and its results in the kidney and liver. In aortic cases throbbing in the neck vessels, headache, and giddiness may be troublesome. Absolutely the best treatment is a week in bed with limitation of fluid and regulation of the diet and moderate purgation. A belladonna plaster often gives relief, whether it acts medicinally or psychically matters little. In aortic cases I have found bromide and iron give relief. General tonics, iron, strychnine, and the like, with perhaps small doses (from two to three minims) of tincture of digitalis where rest cannot be taken will help to put our case into subdivision (1). It is of great importance to determine the nature of the valvular lesion: Is it associated with vascular and renal disease or is it purely cardiac? The tension of the pulse and the state of the artery are valuable guides to treatment; when the former is high and the latter shows signs of disease a rigid milk diet coupled with diuretics

acting on the kidney, such as the xanthin derivatives and the use of some mineral water, will help to lower vascular tension by eliminating toxins.

(3) The case of valvular disease, whether aortic or mitral, which reaches hospital suffering from the signs and symptoms of failing heart needs little description. The heart chair to maintain the trunk upright is often necessary. I need not dilate on the necessity for care in feeding but would simply say that limitation of fluid is often of great service. The bowels may be gently acted upon. Sleeplessness is often a troublesome symptom in mitral cases attended with marked mental depression. In aortic cases excitement and even maniacal attacks may be present; I have seen one case of violent mania in aortic regurgitation. The modern hypnotics—trional or sulphonal and paraldehyde—sometimes suffice but opium is our sheet anchor. I have never forgotten a case of heart disease in a young subject which was admitted to the infirmary when I was a house physician. Apparently all our efforts had proved futile but opium before food was ordered in the form of liquor opii sedativus and liquor pepticus after food. The girl forthwith began to mend and eventually was able to leave the hospital in very fair condition. I have seen good service rendered by the old pill of squill, digitalis, and mercury when the engorgement of the stomach and liver has been marked, and blue pill is the well-spoken-of remedy of many sound practitioners. Should we use digitalis in aortic cases? The answer to this is not quite simple. When we find thickened arteries and possibly some increase of tension in the radial pulse, placing the case in what Huchard and his pupils would call the cardiovascular class, there is no doubt that the best line of treatment is to limit fluid intake, open the bowels, and give renal diuretics. If direct cardiac stimulants are used I prefer strophanthus. As common practice in these cases I give strophanthus through the day with a dose of agurin night and morning. Even in these cases, however, when the pulse tension is low, digitalis is demanded, and I have no hesitation in giving it to an aortic case when the mitral valve has yielded. Trousseau's diuretic wine is a good remedy in similar cases. When digitalis cannot be taken by the mouth the hypodermic use of digitalin will often start the case on the way to convalescence. I need hardly say that in some cases cedematous fluid will have to be removed by instrumental measures; this especially applies to the accumulations in the serous cavities. Dyspnoea is often improved by the use of atropine and strychnine hypodermically. The sinking sensation so often complained of by cardiac patients in the later stages can be alleviated by the exhibition of solid opium or neat brandy.

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THE RELATION OF INEBRIETY TO INSANITY AND ITS TREATMENT.

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THERE are several groups of alcohols in which the physiologist and the physician are interested and these vary from the monovalent alcohols, such as ethylic, propylic, and amylic alcohol through cholesterine, glycerine, and cellulose to the pentavalent phenols and other aromatic compounds. Speaking generally, from their action upon man only the monovalent group concern the subject of our consideration. The effect of alcohol upon living protoplasm has been carefully studied by competent workers and the literature of the subject is full and extensive. Alcohol stops the germination of spores and grains and when the proportion of alcohol exceeds 20 per cent. in the material undergoing alcoholic fermentation further action is stopped, so that this quantity of alcohol becomes lethal to its own development and all further fermentation is arrested. Alcohol is a dys-osmotic and when ingested it passes with difficulty through the living cellular membrane of the capillaries into the tissues. It causes a reaction on the part of these cells which in consequence proliferate and the capillary walls thicken, in this way not only depriving the special organs of their necessary nutriment but also retarding the excretion

of waste material which thus accumulates in the tissues and interferes with their healthy and normal functions. It is this accumulation of effete material which has given rise to the view that poisoning from alcohol is not a direct toxæmia but an indirect one from the production and the accumulation of leucomaines. It is further known that alcohol is a strong dehydrating agent, and as dehydration is as hurtful to protoplasm as superhydration, and as a fixed amount of water is therefore necessary to healthy protoplasm, indeed more necessary for protoplasm than is oxygen—for oxygen cannot reinvigorate dry protoplasm—this dehydrating action of alcohol upon protoplasm is highly injurious. The effect of alcohol upon protoplasm is to paralyse irritability, diminish sensibility and contractility, and when sufficiently concentrated to suspend the action of all ferments. The experiments of Féré upon incubating eggs also show the evil influence of alcohol upon the metabolic, the motile, and the reproductive functions of animal cells.

The causes of inebriety.—It has often been asked, "Why do men drink?" There are probably different reasons among different classes in the social scale. The rich drink for artistic and æsthetic reasons—a good meal is made a better meal. The poor drink in order to get a good meal. It is believed that the stimulation—mistaken for nutrition—is more easily obtained from drink than from a meal, which is more expensive, more difficult to select, to prepare, to cook, and to serve. The poor also get light, warmth, comfort, and society in the public-house. Poverty itself is a cause of drink and drink helps them to forget their human misery, but I believe drink is more often the cause than the effect of poverty. It calms physical and moral pain and the pleasure obtained becomes a customary want. The rapid increase in the volume of wealth and the profits of trade make it easier for the mass to obtain alcohol and there is in many instances a temptation (through facility of access to it) from the number of public-houses. Lastly, there is no doubt that the hurry, the artificial desires, and the quest of pleasure characteristic of civilisation favour drinking habits in both men and women.

Physiological effects.—Alcohol brings about marked degenerative changes in the nervous, muscular, and glandular tissues. Its action is a structural one, and change of structure implies altered function. We find these altered functions manifested in commencing intoxication, when the ideas flow with unaccustomed facility, words are uttered more freely, and language becomes more expansive and confiding, cares vanish, everything seems more full of attraction, and the world seems better. There is a sense of *bien-être*, the eye is kindled, and the visage and the physiognomy are illumined; but this picture soon falls into shadow, for the ideas soon become dissociated, the intellect pales, and words become a vertiginous whorl. The memory becomes affected, the gaiety and the optimism of the previous stage are replaced by sadness, tears, and a querulous or dour disposition; all the senses are overshadowed and the mind ere long becomes a listless chaos without order or purpose. The higher psychic faculties disappear in the order of their importance, there is loss of prevision and judgment, there is failure in the concentrating or focussing power of the mind, and the memory and the attention are both enfeebled. Alcohol attacks first the hierarchy of the organic functions, those which are in the front rank, and they are affected in the inverse order of their development, those last developed being the first to surrender; inhibition or self-restraint—the reserve characteristic of the self-respect of higher man—being first withdrawn. The initial flush of excitement referred to above is readily explained upon physiological grounds by the effect that alcohol exerts upon the capillaries which undergo initial dilatation and subsequent paralysis. The intellectual exaltation noticed has no bearing or relation to genius; it is merely a suspension of the highest faculties which permits the next highest to rise into prominence; it sets free the shackles and removes the veil from a less highly evolved mental plane, and free play is given to the uncontrolled feelings. The effect of alcohol is to stimulate the desires whilst diminishing the power to gratify them. It creates a false feeling of strength which when taxed is not sustained, and alcohol is dissipative rather than conservative of energy. The extension of the effect of alcohol to the motor areas is characteristic and the final stage of complete muscular paralysis is well known as occurring in the wake of unsteadiness, the gait of the inebriate being too familiar to need description. Coexisting with the