A Post-Graduate Address

ON

HEART AND AORTA STRAIN AND
"LYING-DOWN HEART."

BY

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I HAVE chosen the subject of heart strain because it is one which has to be considered by medical men at the present time under a variety of aspects, in our private work and in compensation claims. As I think strain of the aorta may occasionally enter into the question too I have included it, and will give my reasons for doing so at a later stage.

Definition.

By heart strain I mean the sudden development of subjective signs of cardiac muscle failure which can be attributed to some unusual muscular effort, even though the effort was one which ought not to have had such a result in a healthy person.

The chief subjective symptoms of heart strain are pain over the region of the heart or behind the sternum, exactly like, or simulating, true angina pectoris, and shortness of breath on slight exertion. The objective signs are some dilatation of the heart with extension of dullness to the left, signs of valvular disease, feeble sounds or gallop rhythm, and a quick pulse rate with manifest shortness of breath on slight effort.

Compensation Claims.

Heart strain is often made the basis of a claim under the Workmen's Compensation Act, and such a claim was also common at the pensions boards set up after the war. The danger of heart strain occurring in workers who are known to have something wrong with the heart has not infrequently to be considered at the worker's or employer's request. This leads me to refer at once to a most important point in connexion with the causation of disablement from accidents. An accident in the meaning of the law is "an unforeseen or unexpected occurrence arising out of or in the course of employment."

The point I wish to emphasize here is that compensation for incapacity or death must be paid even if the injured worker has had some chronic disease or weakness, such as heart disease, arterio-sclerosis, aneurysm, or high blood pressure, which rendered him liable to suffer serious disablement or death from an "accident" which would have had no serious result in a healthy sound worker. This interpretation of the meaning of an accident caused a revolution in the decisions of the courts administering the Workmen's Compensation and Employer's Liability Acts, and consequently in the whole matter of claims for accidents.

The guiding case on accident claims is that of a man who, when tightening a nut with a spanner, finding it stiff, and making an extra hard effort to move the nut, burst an aneurysm and died. This was held by the House of Lords to be an accident, and this decision governs all analogous claims, the unexpected and unforeseen circumstance, I am told, being the bursting of the aneurysm, not the extra effort.

At present few employers or insurance companies require medical examination before engaging a workman, but the increased sums to be paid under the Compensation Act of 1923 for disablement—£600 lump sum instead of £300 and 30s. a week for life instead of 20s.—may lead to it, especially in elderly men and women. This, however, is a by-path and I will leave it.

Strain of Healthy Heart.

It is a much discussed question whether sudden muscular effort involving a heavy strain on the heart can produce muscle failure or valve injury in a healthy heart or valve of a normal person. I had the experience lately of some cases which at first led me to think that this could happen, if we were to believe the statement of apparently honest

steady men as to a history of accident or of venereal disease. But final investigation and clinical tests put a different complexion on the cases, and I formed the conclusion that I had seen no case of strain in a healthy person. This conclusion falls in with that of the physiologists, as stated by Bainbridge in his *Physiology of Muscular Exercise* (p. 200): "There is no evidence that in a perfectly healthy man, even the most intense exertion produces any harmful effect on the heart."

PREDISPOSING CAUSES OF HEART STRAIN.

All the cases of heart strain in my own experience have arisen through some latent weakness of the cardio-vascular system which has predisposed it to give way under strain which would not have affected a normal system.

I propose to consider the commoner predisposing causes of heart strain, and will take them in their relative order of frequency in my own practice. These are high blood pressure, valvular disease, syphilitic aortitis, and the senile heart. I will refer also to auricular fibrillation in its relation to heart strain.

High Blood Pressure and Angina.

The commonest form of heart strain I meet with is that caused by prolonged high blood pressure, the presence of which has probably not been known or suspected until shortness of breath or pain in the chest forces a visit to the doctor. This is also the commonest cause of angina pectoris, both in men and women, in my experience.

The heart in high blood pressure strain, with or without anginal pain, is considerably enlarged outwards towards or into the axilla, and the sounds are either feebly accentuated or there is gallop rhythm. There may be a murmur of mitral incompetence. The history is generally that of untireable activity in business or household work, then more or less sudden onset of bad shortness of breath or pain or both. When the breakdown has been apparently sudden it is quite likely that careful inquiry will show that there have been previous warnings, extending over a year or more, of slight pain and shortness of breath which the subject, unused to any shortcomings of health or power, thought could be worked off by extra effort.

Nature responds to the increased demands on the heart by laying down more heart muscle, and good compensation results, even if the heart has to be enlarged to twice or thrice its normal size. Life goes on until one of two things happens.

(1) The heart *gradually* becomes unable to do the work demanded from it and it shows signs of muscle failure, with breathlessness and perhaps pain. A common type of this event is the following case.

A lady, aged 73, sent for the doctor because she had a smothering feeling in the chest, shortness of breath, and pain behind the sternum, up the neck, and down the arms—typical angina pectoris. I was asked to see her eight weeks after she had called her doctor in. I found a systolic blood pressure of 180 mm., and the heart was about twice its normal size, with gallop rhythm. I was informed that she was a tireless worker at home and with manifold outside charitable interests. On pressing for the date of the onset of the first appearance of any discomfort I found that twelve months previously she had begun to have slight pain in the chest and shortness of breath on walking, but had said nothing about it to anyone. She had managed to get on with her work in a restricted way until the final breakdown.

(2) The heart *suddenly* fails under the stress of unusual muscular effort, often that of hurrying to catch a train, as happened in the following instance.

Mr. H. B., a business man, had worked hard for years at his own and public business, never getting tired. One day, when hurrying for three-quarters of a mile to catch a train, he became very distressed in his breathing, with oppression round the heart, and could hardly get along the last one hundred yards. The discomfort gradually eased off in the train, but he felt it again next day when walking to business. The pain was then shooting down both arms. When I saw him on February 20th, 1923, his systolic pressure was 200 mm. There was no arterio-sclerosis. The heart was enlarged, the apex extending to an inch further to the left than the normal and the sounds were accentuated. There was no albumin, sugar, or polyuria. At 48 he passed Grade 1 for the army. He was a lifelong abstainer from alcohol. He improved considerably under diathermy.

Here, I think, we had a heart that had been used to overwork for years, and had enlarged by good hypertrophy, but which was not able to stand the additional strain of a

rush to the station, and lost its compensation or failed suddenly. Rushing for trains, with the final dash upstairs—so many stations are above the level of the road—is responsible for many cases of heart strain, not only in subjects of high blood pressure, but in those with a senile heart.

Treatment.—I invariably treat these cases of heart strain in high pressure, especially those with the gallop rhythm, with digitalis, and with great benefit. Digitalis strengthens the failing heart muscle, and it does not increase the high pressure. Nitrites can be given as well, but the digitalis is essential. Less muscular effort, light and small meals, also are necessary.

Valvular Disease of the Heart.

Aortic valvular disease in the form of slight obstruction from roughened valve curtains without incompetence and with no dilatation of the heart or sign of muscle failure, is compatible with manual labour. Many elderly workpeople have it. I found it in one man, a mill hand of 79. He was then too old to work. Such a slight aortic valve change is not likely of itself to induce muscle failure, but its presence is an indication of atheroma and degenerative changes which may implicate the coronary vessels and so weaken the heart. Actual stenosis of the valve with thrill at the base may, in time, lead to heart failure. I remember a man about 40 years of age who fell dead from a telephone pole; his body was brought to the infirmary, and we found he had extensive stenosis of the aortic valve with much compensatory hypertrophy of the left ventricle; why he died we could not say.

Aortic Incompetence.

No one with aortic regurgitation should undertake manual labour, because this is the most dangerous of all valvular lesions, and for two reasons. The first is that when the aortic valves do not close completely there is not the normal amount of force to drive blood through the coronary arteries which arise from the aorta wall just above the cusps of the valve. The heart muscle, therefore, is inadequately supplied with blood and proportionately weakened. The second reason is that the pressure in the aorta, whatever it is, which drives blood through the incompetent valves, acts at its full strength on every part of the interior of the left ventricle of the same area as that of the leakage in the valve, and is not diffused all over the wall of the ventricle. In other words, if there is a force of 100 mm. of mercury acting on a valve leak of 1/4 inch square, there will be a force of 100 mm. acting on every square 1/4 inch of the ventricle, and not 100 mm. diffused all over the ventricle. This can be a very great degree of pressure, and, acting as it does on anaemic heart muscle, a sudden extra strain is very liable to cause paralysis of the heart and death from syncope, or to break down any existing compensation and lead to death from progressive heart muscle failure. I will mention one case in point.

I was asked to see a young man who had a sudden and unexpected strain arising in the course of his work—namely, hastily throwing a skip or basket containing cotton yarn, weighing 50 lb. or more, twenty feet along a floor, to save the time of a worker who was late in getting to the weighing machine where the skips were handed out. He felt a sudden pain in the chest and great breathlessness, and had to sit down. After a time he managed to get to the doctor, who thought he was dying, and kept him in his surgery for a couple of hours. Eventually he improved enough to get out of bed, and five months after the accident he came to see me to ask if I could attribute the condition of his heart to the accident. I found him very short of breath with marked aortic incompetence, and a heart about twice the normal size, with dilatation and hypertrophy and cardiac muscle failure—that is, broken compensation. I said that I could not attribute the whole condition of the heart to the accident described, but that the broken compensation had, in my opinion, been brought about by a sudden effort acting on a heart which had had aortic disease, especially incompetence, with consequent dilatation of the ventricle and some compensatory hypertrophy, for some time previously—a heart with no muscle reserve. The man eventually died in a few months, and the county court judge held that an accident had occurred which had hastened death.

I had one man with incompetence who was a motor driver, and I advised him to give this up. He dropped down dead when walking; fortunately not when driving a motor car.

It is very important to recognize aortic incompetence early, and this must be suspected whenever there is enlargement of the heart with moderate or normal rate of beat and when there is no increase of blood pressure. The diastolic murmur must be sought for carefully down the left margin of the sternum, even in the fourth intercostal space. There may be no obstruction with the incompetence and no murmur heard at the aortic cartilage.

Injury to the Aortic Valve.

I will now mention two instances of aortic valve affection which apparently were caused by unusual strain acting on an already diseased aorta.

A joiner, aged 42, joined the army in June, 1915, and was demobilized in April, 1919, without any medical examination, although he was short of breath on moderate exertion. He served in the Royal Engineers, and in 1918 felt a pain over the heart after lifting an unusually heavy baulk of timber, and after this when the day's work was heavy. He also felt short of breath on marching from this time, but had no pain from walking. When I examined him in 1923 for a pension claim I found the apex to be in the fifth space and probably behind the sixth rib, and five and a half inches from the middle line—that is, two inches too far out. There was a musical squeak of diastolic rhythm to be heard all over the precordia and two inches away from the chest wall, without the aid of any stethoscope. A definite thrill was associated with the murmur. The nature, time, and area of audibility of the murmur were those of an aortic diastolic murmur, and the enlarged heart supported this diagnosis. He was passed as fit for service when he was examined by a recruiting medical board, and he had also been passed for insurance after medical examination before the war. His own doctor was sure that he had had no aortic disease before the war. The man said he had never had venereal disease, and I believed him. Here, I thought, might be a case of rupture or tearing of one of the cusps of a normal aortic valve causing incompetence of the valve, dilatation of the left ventricle, and compensatory hypertrophy, which was only partial because he was still breathless on exertion. A Wassermann test was carried out after a time at my request, and it turned out to be positive. There was therefore almost certainly some predisposing aortitis to weaken the valve.

A weaver, aged 36, in October, 1923, slipped whilst carrying a beam of warp yarn weighing 186 lb. on his shoulder, and was jammed between a pillar and a loom with the beam pressing on his chest. To get loose he had to lift the beam vigorously off his chest, and whilst doing so he felt something "go" inside his chest—like a finger cracking. He had no pain then, and continued at his work for five weeks, getting worse gradually, and then he had to give in because of pain and shortness of breath. I saw him in July, seven months after the occurrence. He then had shortness of breath and the pain at the base of the heart even on dressing himself. I found that he had a pulse of 120. The apex of the heart was four to four and a half inches from the middle line. When he lay down flat I heard a slight soft aortic diastolic murmur in the third intercostal space against the fourth rib. I could not be sure of the murmur when he was sitting or standing. There was no systolic aortic murmur. The second sound at the aortic area was ringing like that met with in aneurysm of the first part of the aorta, but there was no other sign of aneurysm. There was no change in the radial arteries, no increased blood pressure, and no albumin. It seemed to me that he had most likely strained the aorta and the ring of the valve, producing some dilatation of the first part of the aorta and incompetence of the valve. He said that he had had no venereal disease, and I again thought he was speaking the truth, but subsequent investigation showed a definitely positive Wassermann test. The leakage of the aortic valve was not sufficient, in my opinion, to cause as much dilatation of the heart as I thought was present, and so I came to the conclusion that he had probably strained the heart muscle as well as the aorta and brought on acute cardiac dilatation. He probably had some aortitis, and that predisposed the aorta and valve to strain. I was unable to get an x-ray photograph of the chest or an electro-cardiograph record of the heart's action.

These two cases of strain of the aortic valve under severe exertion are striking lessons on the importance of taking no one's word about venereal disease. I must say that I believed the men, and thought in both cases that the strain had come on in men who had not had syphilis.

Aneurysm.—Any person with aneurysm of the first part of the aorta would be very liable to injure the aortic valve and strain the heart by manual labour. The chances of an aneurysm bursting under such conditions are obvious.

Mitral Valve Disease.

Incompetence of the mitral valve—that is, with a systolic murmur—is the commonest valvular lesion in adult life. When met with after 50 years of age it is almost always slight in degree and unaccompanied by any heart muscle change of moment and has been present in stationary form for many years. I have found it in working men and women.

over 68 years of age who did not know that there was anything amiss with the heart. Slight lesions occur in subjects with high blood pressure even at the same age. I have had carters, aged 53 and 45, referred to me by railway companies whose doctors had found loud systolic mitral murmurs and had certified incapacity. The men could not afford to give up work, and maintained ability to go on. There was a little cardiac enlargement, meaning good compensation, and no evidence of arterial disease or of muscle failure. The danger is the possibility of some extra exertion bringing on muscle failure and incapacity. A sudden event like this is not likely to occur with uncomplicated moderate mitral stenosis and incompetence. When the heart rate is normal and regular I generally let the men continue at their work and see them again in a month. If there is no change for the worse I recommend continuance of work which does not involve lifting of heavy weights. Men with such hearts would not get the disablement benefit of the National Insurance Act because they are able to do some work. One man I remember, in recruiting days, with a loud mitral systolic murmur and practically normal-sized heart, lifted 25 tons of pig-iron a day as part of his carting duties and felt no inconvenience.

Mitral and Aortic Disease.

All I could say in the following case was that army service had aggravated the heart condition.

A youth, aged 24, claimed disablement from war service. He enlisted at 15, was discharged on account of his age, and recruited again at 17. He was riding in the training camp when his horse bolted. He remembered nothing further until he found himself in hospital eighteen days afterwards. He had no rheumatism or its allied ailments. As a boy he had played games and managed physical drill all right. When in hospital his heart was found to be affected. He was in the army fifteen months only and never got beyond the training camp. I found that he had an enlarged heart extending 5 inches to the left of the middle line, and behind the sixth rib. There was a loud crescendo murmur with accentuated first sound typical of chronic mitral stenosis and a definite aortic diastolic murmur squeaking in character. Muscular compensation was good. I did not see how the accident could have brought on the valvular disease, especially the mitral stenosis, and there was no history in his army papers of any illness that could possibly have been rheumatic in nature. The valvular disease, especially that of the mitral valve, was almost certainly there on enlistment, although I could get no history of rheumatism in childhood.

Auricular Fibrillation.

Auricular fibrillation is a most important cause of serious heart-muscle failure, and its presence, either with or without valvular disease, contraindicates manual labour because effort makes it worse. At the same time a certain number of people work with it in its early stages before breathlessness comes on or before there is any indication of an abnormal heart. I have seen three people over 60 years of age claiming pensions with it, who were relieved of work at once. Auricular fibrillation is caused by failure of the auricular muscle, and it is conceivable that overexertion might bring this on in a weak auricle, but I have never met with a case in which fibrillation could be attributed to sudden strain, and at present I should be very unwilling to believe that such could happen. Auricular fibrillation is not uncommonly present, with or without organic valvular disease, without being suspected, and this fact would discount the value of its presence when recognized for the first time after a supposed accident.

Strain of Senile Heart.

By senile heart I mean a heart which is badly nourished because of atheroma of the coronary arteries or which has some fibroid degeneration—changes which occur in the later years of life.

A gardener, aged 62, used to mowing lawns, was delayed one day in trying to get a motor attachment to work. He failed to do so and had to push the machine, and unusually fast, to get the work done. He had an attack of palpitation and breathlessness with heart pain at night and all the next day, and had to give up heavy work, because of shortness of breath which came on when he made an effort. I saw him a month later, and found that the heart was a little enlarged and there was slight arterial sclerosis. His blood pressure was only 130. The pulse at the wrist was 64 and irregular, whilst the heart rate was 76. This meant

extra-systoles or auricular fibrillation, probably the former, for they are common at his time of life. He had ailed nothing before, but on being asked the question, said that for some years he had noticed his heart beating irregularly when he lay down in bed on his left side. Here, in my opinion, the man strained a senile heart muscle by unusually heavy exertion and had brought on some dilatation.

As I mentioned under high blood pressure a rush for a train at a railway station is not uncommonly the cause of the breakdown of a heart in older persons.

LYING-DOWN HEART.

Here is a case of pain of doubtful nature, but I wish to mention it because of a peculiar condition of the heart not uncommonly met with in people with intrathoracic pain.

E.W., wood machinist, aged 46, had had palpitation, shortness of breath on the least exertion, with precordial pains aching in character and occasionally very severe, on and off since 1917, when he was serving as a farrier in the army. He stuck to his work in spite of the pain. He had diphtheria in January, 1918, but went back to his farrier work, though the pains were worse than ever. He reported sick in April, 1918, because of debility persisting from the diphtheria. The doctors also put some letters on his sick sheet (? D.A.H.), and discharged him from the army in July, 1919. After a period of light duty as B2, with "something wrong with the heart" (again three letters, he thinks—probably D.A.H.), he took up his trade of cabinet-maker again, and was practically well until twelve months before he came to me, though he had occasional attacks of pain. He then began to find that the pains recurred regularly and that he felt done up by the Wednesday, but he kept at work until Saturday midday, when he went to bed for the week-end. Extra bad attacks would come on every two or three months and he had to go to bed; he then got ease by lying propped up on the right side with his knees up and by hot flannels about the left breast and some brandy. Walking a short distance against the wind would bring on an attack with palpitation and dyspnoea. Although the case was very suggestive of heart strain, there was practically nothing abnormal to be made out in the chest, and the man looked healthy and robust. The Wassermann test was negative. The x-ray photograph showed a heart of the "lying-down" type with the aortic shadow quite well defined, but no dilatation or aneurysm. There was some general peribronchial thickening, especially in the right lung, but otherwise the lungs showed no marked abnormality. The movements of the diaphragm were normal. There was an old-standing adhesion from the left root to the left diaphragm. Pleuritic adhesions do cause pain sometimes when stretched by deep breathing.

The electro-cardiograph record showed impaired conduction from the auricle to the ventricle, but this does not account for the pain.

I meet with this so-called "lying-down heart" occasionally in people who complain, as this man did, of intrathoracic pain or shortness of breath and in whom I can find no objective signs of heart abnormality. It is only revealed by an x-ray plate which I have taken in obscure cases. I cannot say why it should be associated with pain and cardiac debility, or how it can cause pain, if it does cause pain.

FIG. 1. FIG. 2.
Orthographic tracings: Fig. 1, normal heart;
Fig. 2, lying-down heart.

The term "lying-down heart" is used because, as the diagrams show, more of the heart is in contact with the diaphragm than is normal. It is a descriptive and convenient term. I wish to thank my radiological colleague, Dr. J. M. W. Morison, for the figures which he has drawn from orthographic tracings (Figs. 1 and 2).

THORACIC MUSCLE STRAIN.

I will mention briefly as a contrast to the foregoing cases a railway carter who was thought by the railway surgeon to have strained his heart by muscular effort. As a further contrast this carter did not want to claim compensation,

but to be allowed to resume his work. I have mentioned it before (*Clinical Journal*, July 23rd, 1924), but should like to do so briefly again.

G. H., a railway carter, aged 55, was wheeling a hand truck carrying 7 cwt. of goods in October, 1922, when he felt a sharp pain behind the sternum and over the heart but not down the arms. For half an hour he felt short of breath and had to sit down, and attempts to breathe "punished him like a knife." After this he felt practically normal, but reported the incident, according to instructions. He went to work next day, feeling sore before the heart and in the chest, and with a sharp pain now and again when he was lifting heavy weights. He was sent, against his wish, as he wanted to work, to see the company's surgeon, who found nothing wrong, but thinking of the possibility of heart strain advised that the man be sent on to see me. I found systolic blood pressure 150. There was no albumin or sugar, and no arterio-sclerosis; the heart was practically normal, rate 90 standing, no sign of aneurysm. An electro-cardiograph record was taken, but it showed nothing abnormal.

The seat of the pain and its nature and mode of onset certainly suggested the likelihood of strain of the heart from sudden overexertion, but I thought it might be due to strain of a skeletal muscle and of the triangularis sterni. As the man was very anxious to get back to work we accepted this view, but put him back on a lighter job, and he kept perfectly well until he had an attack of encephalitis in February, 1924; from this he recovered.

DIFFICULTY IN PROVING HEART STRAIN.

Heart strain is not easy to establish in a court of law because of the difficulty in proving accident. I will briefly relate an instance of this.

A railway porter claimed compensation for strained heart, caused when carrying full milk-cans weighing up to 300 lb. up the station steps with the assistance of another porter, each porter taking hold of one handle of the can. Whilst doing this one morning the projecting flange which strengthens the bottom of the can caught under the overhanging edge of the stairs and pulled the porters up suddenly; this caused a pain over the heart of the claimant and subsequent shortness of breath, which persisted for several months on slight exertion, and kept him in bed most of the time until I saw him on his own behalf. I found that he had some slight enlargement of the heart and symptoms which could have been caused by heart-muscle strain. He was a healthy youth of about 24, though not of robust type; there was no history of venereal disease, but there was some neurasthenic element in the case. I told his lawyer, from past experience, that it would be very difficult to prove "accident"; but I agreed to say what I could on the strain, neurasthenia, and debility theory. It then transpired that the man had been first treated for a feverish cold or influenza, and as his doctor found unexpected cardiac weakness four or five days after the commencement of the illness or influenza he asked the porter if he could have strained his heart in any way. The man then thought of the accident, but had never mentioned it to his fellow porter working with him, or to anyone in the station at the time it occurred, nor did he do so until a week after the doctor had asked if there had been a strain. The railway company's doctor put the condition down to influenza, and on the above evidence produced in court, and heard for the first time, the judge held that an accident had not been proved and that the heart condition was due to influenza, which I think was a proper decision on the evidence.

I really did think that the man, who was not robust, could have strained his heart in the way claimed.

Another case was that of a collier, aged 38, who said that one day in the pit he had lifted a crane weighing $4\frac{1}{2}$ cwt. which had become displaced. He felt short of breath but complained of no pain at the time. A couple of weeks afterwards he had some pains in the joints. He worked for four weeks after lifting the crane and then had to give up because of shortness of breath. I examined him nine weeks after the supposed strain. There was little to be found, only some slight dilatation of the heart with weak and doubled sounds. There was no albumin in the urine and no increase of blood pressure. The man was very short of breath. He was sent to bed for two weeks, and then got up again, feeling no better. He dropped down dead, presumably from heart failure, five weeks after I saw him. No *post-mortem* examination was held, unfortunately, but the predisposing cause of death was very likely atheromatous coronary arteries.

Here I had no doubt that the man strained his heart in some way lifting the heavy weight, but the court found against him, holding that strain was not proved, and that even if it had taken place could not so badly affect a sound heart, and that there must have been pre-existing heart disease. The case was tried in 1907, before the House of Lords decision on the aneurysm and the spanner claim, which influenced so greatly the meaning of an accident. I am sorry that I was unable to get a Wassermann test here, for very probably, in one so young, we should have found it positive.

An Address

ON

A NEW OUTLOOK ON CANCER:

IRRITATION AND INFECTION.*

BY

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MORE than three years ago I¹ published an account of an organism obtained from carcinoma, and since then I² have described in increasingly more complete detail the morphological characters of this parasite. I have claimed, on the basis of a study of more than sixty cases, to show that this microbe can be obtained almost constantly from cancer. My conclusions have in some quarters met with opposition on two grounds. In the first place, it has been urged that to account for the cancer phenomena it is unnecessary to predicate the existence of a parasite. In the second place, it has been stated that my views are opposed to the established science of bacterial morphology. Within the limits of this paper I propose to deal with these two objections.

Of all the diseases of man cancer has probably the most unique features; it has little direct contact at any point with other morbid processes, and, for that reason, analogy is of small positive help in a search for the cause. The essential fact which marks it off is that, beginning in the proliferation of a small group of cells, it ends, when running its ordinary course, as a myriad host of anarchic cells destroying by direct inroad the healthy surroundings and burrowing often into the springs of life itself. Any class of cells in the body may be seized with this tragic impetus, and, according to their site of origin, so will the cancer differ in structure and, it may be, in deadliness; but in all, no matter what the origin, the same unique features dominate the process, in all we have a plague of riotous cells spreading as a blight without control or cessation.

It is important to stress the essential unity of the cancerous process, especially at the present time, for some observers are so immersed in detail that they have allowed themselves to lose sight of this unity and have accordingly, I believe, fallen into serious error. The differences in detail are seen when malignancy involves structures of different kinds. For example, when originating in squamous epithelium, squamous epithelioma is produced; when in gland acini, glandular cancer; but that these structural differences, which may be considerable in detail, are non-essential in regard to the ultimate nature of cancer is shown by the fact that, in an organ possessing both types of epithelium—for example, the uterus—we may find both types of cancer side by side. So, in the same way, carcinoma and sarcoma may occur together, and the essential similarity between these two such divergent types of "cancer" is further proved by the experimental finding that transplantable cancer may end as transplantable sarcoma, the cancerogenic factor in this case having been transmitted from one class of cell to the other. Considerations of this sort indicate that, where the malignant change is occurring in widely different tissues, the diversity in structure, and it may be in other points as well, cannot be held to justify any diversity in the ultimate nature and cause. The differences are rather local and environmental, and therefore non-essential.

The unique phenomenon of malignancy is not limited to man; it is found throughout the lower animals, and a process of an analogous kind is found also in the vegetable kingdom.

CHRONIC IRRITATION.

One of the most important positive facts in regard to the etiology of cancer acquired within recent years concerns the role played by chronic irritation. The experimental production of cancer in animals by the exposure of the skin to chemical irritants, of which there are now several, has lifted a long-established clinical observation into the realm of proved laboratory fact. It can now be claimed with

* Delivered in opening a discussion on cancer at the Liverpool Medical Institution, December 11th, 1924.