CORONARY THROMBOSIS*

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Coronary thrombosis is the name which has come into use in recent years for the serious heart attack which results from thrombosis in a coronary artery or branch. The series of events following such complete obstruction is known as cardiac infarction, an alternative term with the same clinical meaning as coronary thrombosis. We do not propose to refer in detail to the pathological process, but in brief it is as follows. A large branch of a coronary artery, often but by no means exclusively the descending branch of the left coronary, happens to be the seat of a stenosing atheroma which narrows the lumen. At the point of greatest constriction, and perhaps favoured by atheromatous ulceration, thrombosis takes place and completely blocks the vessel. The resulting infarct occupies the territory hitherto supplied by that vessel. As the wall of the ventricle is thus involved, it may be that the necrotic reaction reaches the endocardium to precipitate an intracardiac thrombus. Less often the process reaches the pericardium to produce a patch of pericarditis over the infarct (infarct-pericarditis). Such is a usual post-mortem finding when sufficient time has elapsed before death for the infarct to form. On the other hand an infarct may result from extreme coronary narrowing without the complete occlusion of actual thrombosis, and yet produce all the symptoms of "coronary thrombosis." Syphilitic stenosis of both orifices may give the same picture. But I wish to leave quickly this aspect of the condition, for we are to deal with the clinical results of infarcts from which patients often recover, a fact only realized in the last decade.

THE CLINICAL PICTURE

An attack of coronary thrombosis is most commonly seen in a man over 40 who has apparently been in good health, or who has been inconvenienced for some months or years by anginal pain on undue exertion. While at his usual occupation, or while at rest or even asleep in bed, he suddenly begins to feel a dull heavy pain at the sternum or across the front of the chest. He will often think it is indigestion, but the pain persists and grows to such an intensity in a few minutes or an hour that he fears, and rightly fears, that he is in the clutches of something more serious, even at grips with death. He sweats in anguish; he may feel sick or actually vomit; maybe he faints. The pain becomes a relentless grind, and often extends through to the back and down both arms. A sense of constriction may be felt, though this is not essential to diagnosis. By this time he is so anxious and distressed that he may be unable to keep still because of his agony, and turns restlessly in bed or paces about the room. Unless the patient succumbs, after the lapse of two or three hours or longer the pain begins to abate, the next day little being left beyond a residual ache in the chest, though slighter attacks may recur for two or three days.

The doctor will find the patient in obvious distress and jangle, pale rather than cyanosed, restless rather than still. His one complaint is of pain, and his breathing is no more disturbed than would be expected with such pain. The pulse may not indicate the grave nature of

the attack, and no practitioner should be misled by its comparative normality into thinking this is no "heart attack": usually it is regular and only moderately increased in rate. Occasionally the rhythm is disturbed by auricular fibrillation or heart-block. The pain may be so abrupt and occupy such a site that a physician will often wish to exclude an acute abdominal condition requiring surgical intervention; but, having done so, he should speedily make the diagnosis of coronary thrombosis and give the patient relief by an injection of 1/4 grain of morphine, repeated as often as necessary to fulfil his obligation of relieving pain. The chief features of the further course include a rise of temperature as after an infarct elsewhere, though often it is less pronounced. In a certain number of cases a pericardial rub is heard, though perhaps only for a few hours. Shortly after the attack the blood pressure falls from shock, and a systolic reading of 100 mm. Hg is not unusual. It may happen, however, in a patient with a really high blood pressure that this fall does not bring the systolic pressure below normal limits, though it is lowered greatly for him.

The end of the terrible attack described is no more than the beginning of a long illness which is shadowed by death or complications. Sudden death may occur at any stage, though the risk of it diminishes with the passing of a few days and especially weeks. Heart failure of the congestive type may follow the attack within a day or two. Embolism is a complication which looms within the first three or four weeks. A fragment of the intracardiac clot is dislodged and launched into the systemic or pulmonary circulation. Despite these ominous risks, the average patient will get through his illness to comparative safety; indeed, if the illness takes a favourable course from the cessation of pain, the patient may feel so well that he has to be told why he should remain in bed.

For the purposes of this introduction I have had to limit my description to that of an average case, though clearly varying sizes of cardiac infarct grade the severity of an attack. Further, it may occur in a patient comparatively young whose coronary arteries are in fair condition apart from the flaw exposed by the infarction. It may, however, seize an older patient known to have been subject to undue dyspnoea on exertion or to angina of effort, showing that it is a faulty myocardium which has been infarcted.

AETIOLOGY

In considering certain points of especial interest, I would first refer to the aetiology of coronary thrombosis, which is a local product of arteriosclerosis. The most practical view of arteriosclerosis is to consider it as common to all of us as we age. In some it arrives early, in others late, but it is inevitable. It is unfortunate when premature, or when it is situated in the vital arteries of the heart or brain. As we do not know the fundamental cause of arteriosclerosis, we must likewise admit that we do not know that of a coronary thrombosis. Yet our ignorance of the primal cause need not deter us from seeking for secondary or accessory causes which may favour a local exacerbation of the arterial disease. Cardiac infarction is seen most commonly in patients over 45, though it is by no means unknown between 30 and 45, or even below 30. It is a disease of the late middle-aged as much as of the elderly. Women are not so liable to it as men, but there is a danger in practice, for this and other reasons, of its not receiving in women the prompt attention it deserves. Hypertension is present in a fair proportion of cases, though it is in no sense an essential part of the disease. Syphilis rarely enters into the aetiology, though its consideration is important in differential diagnosis.

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VARIATIONS IN SYMPTOMS AND SIGNS

The symptoms vary chiefly with the size of the infarct and the previous condition of the heart. Although the typical attack is now easy to recognize, much remains to be done in assessing the clinical features of minimal thromboses, or of those occurring in patients already the subject of other cardiac disease. Thus, an infarction may explain the sudden occurrence of auricular fibrillation, flutter, or heart-block. It may determine the onset or accelerate the progress of congestive heart failure in the course of hypertension. Another matter bearing on general medicine is that a cerebral or other embolus may once have been seated on a cardiac infarct. Even apart from these complicated cases, it is now quite certain that a cardiac infarct may for some unexplained reason lead to dyspnoea and failure without pain at all. It may also be one of the causes of certain syncopal attacks with shock and collapse which are seen in elderly patients.

Because there are no typical physical signs until long after the diagnosis should be made, I barely mentioned them in describing a typical case of coronary thrombosis. This lack of signs has proved a great hindrance to its wider recognition. We have been trained to rely more upon our own observation than upon the patient's statement. To make any progress in the diagnosis of coronary thrombosis this attitude must be reversed. The diagnosis can and should rest upon the symptoms and the history, physical examination seldom adding anything to clinch it. Let us remember that coronary thrombosis is primarily an arterial disease. Therefore the recognized marks of arteriosclerosis are the signs to be sought. Of these may be mentioned the accentuated aortic second sound and the thickening of the superficial arteries, of the finer visible arteries in the retina, and of the largest artery—the aorta—as revealed so clearly by radioscopy. Although it is true that the heart sounds are often distant, less distinct than usual, we dare not place much reliance on a sign so common in other conditions and so dependent on the personal factor. The heart is little or not at all enlarged unless there has been associated hypertension to cause it.

DIAGNOSIS AND PROGNOSIS

The diagnosis will be helped by requiring the patient to map with his index finger (not with his hand) the total area of the pain. This will almost invariably include part of the sternum, a strong diagnostic point being sternal pain. It is best to avoid the ambiguous phrase of "pain over the heart," with its loose associations in the mind of doctor and patient, "sternal" being a safer term than "precordial" for use in diagnosis, which, as already emphasized, rests upon the patient's description of his attack. Fortunately the electrocardiograph provides evidence which compensates for this absence of physical signs. In differential diagnosis we must be prepared to reject the patient's belief, often no more than a pathetic hope, that it is indigestion. More difficulty may arise in excluding acute abdominal conditions such as perforated gastric or duodenal ulcer and acute cholecystitis, and conditions like pulmonary infarction and spontaneous pneumothorax, which also cause sudden and severe pain in the chest.

Referring to prognosis—as sudden death may occur at any time from the onset of pain, it must be admitted that coronary thrombosis implies a dangerous condition. Our attention, however, should be focused on the patient's chance of survival, for experience begins to show (as in so many other new diseases) that the outlook is by no means so gloomy as was at first thought. When the patient has weathered for a few days even an intense attack, the natural expectation should be that he will not

only reach convalescence, but will recover a fair degree of health, even good health, which may continue for years to come. A second attack is relatively infrequent—a fact which should be conveyed to the patient. Although the scope of physical signs has been decried in the immediate diagnosis, we need the strictest assembling of all that can be learnt about the cardiovascular system before we are in a position to attempt the further prognosis in a given patient.

PRINCIPLES OF TREATMENT

As students we found it difficult to understand why our teachers were so insistent upon teaching us diagnosis and were so apparently neglectful of treatment. If this paradox required an explanation, either to the student or to the layman, we could scarcely give a better example in justification than that of coronary thrombosis. Once the diagnosis is made, the treatment is indeed obvious. There must be quick relief from the pain, and ample time for rest to favour healing of the infarct. Morphine is indispensable, and the use of this veritable godsend should be the first care of the physician when he has made the diagnosis. Morphine also provides the best means of limiting the resultant shock by elimination of the pain factor. Digitalis is indicated directly signs of congestive heart failure appear. From the first it has been confirmed that amyl nitrite and trinitrin have little or no effect in allaying the pain, and taking into account the fall in blood pressure they are contraindicated. On the other hand trinitrin occasionally finds a place in the residual attacks of anginal pain which sometimes occur in the later weeks following the initial attack, and even while the patient is still confined to bed.

PROLONGED REST IN BED ESSENTIAL

It has always been a pleasure to me to find how deeply ingrained in the medical mind is the idea of six weeks as a minimum period for rest in bed for rheumatic fever. If medical wisdom insists on this rest as proper for an inflammation of the heart, it will surely not seem too much for an infarct of the heart. Therefore, comparable with the rest assigned in the standard treatment of rheumatic fever, we shall not err in fixing a minimum six weeks' rest in bed for a case of coronary thrombosis. Difficulties often arise in practice respecting the period in bed. The patient cannot visualize as we can the serious condition of his heart. When the pain subsides he may feel well again, and find it hard to accept our stringent advice to remain so long in bed. For this reason, and in keeping with the spirit of the times which advocates candour, I think we should explain to the patient, according to the measure of his understanding, a little about the nature of his "heart attack." I say heart attack because it is unjustifiable, where such issues are involved, to be a party to any policy of hoodwinking, or of allowing the attack to be regarded as anything else. If this frankness does not suffice, reference to a tiny clot in the heart muscle would not be out of place, though any detailed description is to be deprecated, for it presumes a knowledge the patient does not possess and would probably be misunderstood. The pleasantest reason to offer for this long confinement to bed is the need for rest to enable the heart to recover from the attack. To the friends, and to an obdurate patient, the other reason can be given, which is the fact of there being a real danger to life if the prescribed rest in bed with the requisite nursing is refused.

LENGTH OF CONVALESCENCE

When the patient does get up, it will be our further duty to advise as long a convalescence as he can afford, and this will often be longer than he thinks his circumstances

permit. A period of three months from the initial attack should elapse before he returns to his previous daily activities, and then only if they can be undertaken without any reminder of the pain or return of the pain on exertion. For those who are able, it is better still for them to arrange to abstain from work for six months. After the standard treatment of the attack itself there is ample opportunity for a medical man to prove his worth in individualizing treatment. The treatment of a disease may be impersonal, but the care of a patient must be personal. There is perhaps no occasion like the time following a coronary thrombosis for a patient to be advised to take stock of his position and to plan his future. Ambitions will often have to be relinquished and activities curtailed. In selected cases retirement will be an excellent solution of the difficulties so often inseparable from an active life.

Relation of Angina Pectoris to Coronary Thrombosis

Having outlined the clinical features of coronary thrombosis, I think this is an occasion to consider its relation to angina pectoris, for its recognition as a common occurrence may clarify our conception of angina pectoris, the parent disease.

The first great event in the history of painful heart disease was the description of angina pectoris in 1768 by Heberden, who then separated it from heart attacks characterized by dyspnoea. He drew attention to the suddenness and severity of the pain, and its dependence upon exertion or excitement, at least during the earlier stages. He knew well the special dangers to life angina presents, but he also asserted (what many forget) that often it continues many years without any injury to health apart from the liability to pain on exertion. Although he said it was of brief duration, he added that "in one or two persons the pain lasted some hours or even days," and "once only, the very first attack continued the whole night." Almost every writer on the subject since Heberden has also mentioned or described patients whose prolonged attacks can now be interpreted as coronary thrombosis. Clear examples are found in Parry's book on syncope anginosa in 1799, and in Mackenzie's book on angina pectoris in 1923. So it is evident that for more than a century the clinical picture handed down to us as angina pectoris has been a composite picture of angina pectoris of effort and coronary thrombosis. The latter was regarded as angina in a severe degree if it was recognized as angina at all.

The second discovery of importance was the relation of the disease to the coronary arteries, as claimed a little later by Jenner and by Parry. Later still the remedial value of vaso-dilators was discovered. Lauder Brunton in 1867 introduced amyl nitrite, and Murrell in 1879 introduced nitroglycerin (trinitrin). Apart from these advances there seems to have been little progress in our understanding of the disease until suspicions arose that a prolonged attack of anginal pain might signify a particular pathological event in the heart muscle—namely, a cardiac infarction. Isolated records show that a physician here and there knew that a coronary thrombosis might cause a prolonged attack of angina, even with recovery. It was Herrick who, in 1912, began to insist upon the ease of diagnosis, the frequency of the condition, and the frequent recovery. His contributions were reinforced by those of other physicians and by the use of the electrocardiograph. We owe a debt to America for elucidating what proves to be a considerable sector of the whole field of angina pectoris. The isolation of coronary thrombosis shows that direct clinical observa-

tions, coupled with post-mortem work, can still contribute effectually to medical progress when physicians are not subject to the domination of authority and tradition.

If we inquire into what had been done between Heberden's discovery of angina and the recognition of coronary thrombosis as a common clinical entity, we are struck by the bulky disquisitions upon the nature of angina and the rivalry of various hypotheses respecting its causation. It would be a relief to the profession if this protracted discussion on the precise meaning of angina pectoris (disease) and its root cause could be ended in agreement. Clinical progress has been delayed by a preoccupation with the unknown cause, and our terminology has been tangled by confusing angina (disease) with anginal pain (specific symptom). Moreover, the mystery has created an atmosphere dismal to the doctor and terrifying to the patient. The advent of coronary thrombosis seems to many of us to have settled the main issue, and to have established the coronary theory of angina and the machinery of anginal pain. During its introduction the features which distinguish it from ordinary angina pectoris have had to be emphasized. What now needs emphasis is the essential *unity* of angina pectoris and coronary thrombosis. We have seen that the traditional description of angina by our predecessors was based on a combination of both. There is no need to labour their close relation, for it is patent in every communication on coronary thrombosis. The same individuals may suffer from one, but they commonly suffer from both. A large proportion—some think one-half—of all victims of coronary thrombosis can furnish a history of anginal pain on exertion. Even more important to my mind is the fact that angina of effort is often conceived in a first attack of coronary thrombosis, and that in this way an ordinary angina of effort may originate in a coronary thrombosis which escaped notice. Realization of this fact may have a gratifying effect in treatment, for by earlier recognition of coronary thrombosis and meticulous care in its treatment, the supervention of angina of effort may sometimes be prevented or postponed.

POINTS OF SIMILARITY

In considering the clinical unity of angina of effort and coronary thrombosis we note their similar quality, site, and extension of pain, their frequent concurrence, and their common danger. More correctly the danger to an ordinary angina patient is chiefly his liability to coronary thrombosis. Surely such a close relation points to a similar pathological cause. In the derivation of each from a disease of the coronary arteries, angina of effort and coronary thrombosis have acquired a vascular identity. The term "angina pectoris" should still cover the whole disease compounded of angina of effort and coronary thrombosis, and any grade of anginal pain expressing any grade of coronary disease between them. Thus a general acceptance of the coronary theory will rationalize our conception of painful cardiac disease; though, if this common factor is established, there is still scope for research into the pathological physiology and into the finer tributaries of cardiac pain.

Admittedly there are still difficulties in this acceptance, but they are dwindling. In the majority of cases of angina of effort, coronary disease is found at necropsy. Of course, coronary disease is commonly found where there have been no symptoms, and coronary thrombosis may even proceed to cardiac failure without pain. It is admitted that in a few reported cases of angina of effort the coronary arteries have been found intact, but I think that even fewer will be reported when a more searching technique is employed, such as the radiography of hearts in which the coronary circulation has been shown in all

its ramifications by previous injection with an opaque emulsion. Changes in the quality of the blood supplied (as in anaemia) may occasion pain similar to that due to an inadequate quantity. Yet we need not now despair if difficulties baffle us, and the coronary theory will serve us well to comprehend the great majority of cases, even if we cannot explain every case and fit it into our category.

THE CAUSE OF ANGINAL PAIN

There is the further problem to which I have alluded—how anginal pain is evoked in the heart. Opinion is tending to the conclusion that it arises from localized spasm in the finer coronary branches. The idea of coronary spasm would quickly be suggested by the spasmodic nature of the pain (fleeting cause—fleeting pain), and its relation not only to exertion but to emotion and to cold. The amazing effect of vaso-dilators, an almost unique phenomenon in therapeutics, carries its significance. Bearing on this problem several communications have been made in the last two or three years which show that in ordinary brief attacks of angina, spontaneous or induced, temporary changes often occur in the electrocardiogram, and the changes, though slight, are similar in nature to those which are known to be characteristic of coronary thrombosis. If these observations are further confirmed, no one will venture to deny at any rate the cardiac origin of anginal pain. It will be hard to imagine what can produce so distinctive and temporary a change in the heart muscle so well as spasm in the finer branches of a localized arterial territory, a spasm resulting in an ischaemia which possibly rehearses the bigger drama of a coronary thrombosis with infarct.

TERMINOLOGICAL CONFUSION

In the course of a century certain misconceptions and faulty classifications have become attached to that clear picture seen by Heberden, and many terms can now be regarded as relics of a classically educated profession. To begin with, the terms "pseudo-angina" and "false angina" have been a nuisance. Must one repeat the valid criticism that there are no false anginas, only false diagnoses? There seems no justification for the terms "angina major" and "angina minor," or for primary and secondary angina. The patient with angina decubitus is one already subject to angina of effort who has become worse, so that he is now liable to attacks at rest and when lying down—nocturnal angina, if the attacks are at night when the patient is in bed. The status anginosus is usually no other than coronary thrombosis. Angina vasomotoria is too rare and complex to detain us now. It is not quite so easy to dismiss angina sine dolore, because a coronary thrombosis sometimes produces an attack of acute and even fatal heart failure without pain. Having accepted the fact, however, the phrase is redundant. In the original and different sense Gairdner introduced it in an attempt to express a "specially indefinable and indescribable sensation," "a painless angina found with every kind of cardiac lesion which ends in death (whether sudden or not) in varying proportions." This definition is, you will agree, sufficient to stamp it as a doubtful and unnecessary addition to medical terminology. Since the term "angor animi" (the sense of dying) was introduced, its prevalence and significance have been so much exaggerated that it figures even in the definitions of angina pectoris. Worse still, some older writers have elevated it into something which equals or even eclipses the pain, and which has diagnostic value accordingly. It is held to be a weird sense of impending dissolution, not merely a fear of it. If such psychic experiences do occur, they have also been related by

patients suffering from diseases other than angina pectoris. Their origin is to be sought in the nature of the patient, and not in the nature of the disease. If it means no more than a sense of impending death, what could be more natural than for sensitive patients to feel it? The stronghold of life is directly attacked, and the patient may realize it—that is all. Surely there is nothing morbid or pathognomonic about that.

BETTER PROGNOSIS IN ANGINA PECTORIS

It is quite time for a reconsideration of our views on the prognosis of angina pectoris. It is seen how patients with its severest form, coronary thrombosis, may not only recover from the attack, but live in fair health, or even in good health, for months and years following it. If now we exclude coronary thrombosis, and also that other dangerous variety, syphilitic angina, we are left with the average patient, one subject to sternal pain on exertion—that is, angina of effort. This is just as certainly angina pectoris, and it connotes an uncertainty with regard to life; but the prognosis is far better than is commonly supposed. These patients—and they are not uncommon—usually live for years. Are these years to be darkened by dwelling on the possibility of sudden death? Coronary thrombosis has not taken the sting out of angina pectoris, but it has removed much of it.

Not only has the imminence of death in ordinary angina of effort been exaggerated, but I dare to say that even the severity of the pain in the average patient has been exaggerated. The horror of some cases of coronary thrombosis has coloured the brief attacks of angina to as dark a hue. Even those cases with as intense a pain as any in human experience, now that they are recognized for what they are, can be alleviated by the bold use of morphine. Let us take, however, an even commoner clinical case, no other than that chiefly seen by the eye of Heberden. A man begins to notice that on hurrying he has to slow down or stop because of pain at the sternum or across the chest. It is an unpleasant pain, but he may deny that it is severe. There is often a direct relation between its severity and the degree of effort, and the term "paroxysmal" is scarcely applicable. Such a man is to be pitied and treated, but nothing is gained by magnifying this into an awesome and terrible attack. The temperament and tastes of the patient will largely decide how far his life is rendered unhappy by this handicap. For years he often succeeds in dodging the pain or keeping it within bounds; yet there may be added sooner or later true paroxysms which are unavoidable and independent of exertion, and coronary thrombosis may eventually supervene. Another dogma about angina pectoris is its incurability, and it is incurable in so far as coronary sclerosis is the foundation of it. Yet in that the process may become stationary, or may be corrected in part by a collateral circulation, it is not unreasonable to expect occasionally long remissions in the symptoms; and they do occur. In fact, after a cardiac infarction it is sometimes found that with proper rest and restriction the patient may actually find himself able to do more without pain than he could before the attack.

Until our own house is set in order the public will continue to view angina with morbid apprehension. It has been inculcated by a century of medical fearfulness. The popular conception of angina pectoris is attached to the disease in its severer forms. The public do not know, and some of us have forgotten, that all degrees of severity are seen in this disease, as in many others. A brighter view is justified, though no view can be called exactly bright. My point is that patients with angina often find

life worth living, and that they will live longer than is commonly supposed.

Conclusions and Summary

By way of summary and to conclude: Angina pectoris is a serious disease of the heart which is manifested by pain across the chest or at the sternum, often extending into the arms, and which is due to deficient coronary circulation or imperfect blood supply. There are two main varieties, but their specific pain differs only in intensity, and their pathology has a common factor in myocardial ischaemia. When the pain is brief and depends on exertion, anginal pain is known as angina of effort, and arises from ischaemia due to localized arterial spasm. When an attack of anginal pain is unrelated to exertion and sufficiently prolonged, and complicated by the symptoms of infarction, it is known as coronary thrombosis, and arises from the resulting necrotic ischaemia. Between these two varieties all grades of angina pectoris are encountered, in the main depending on the severity and extent of the coronary disease and the effect on the myocardium—whether the ischaemia is transient (arterial spasm) or permanent (infarction). Cases of angina pectoris range from simple to severe, and present a similar pain

(anginal) of varying intensity, and reveal at their source a vascular identity.

Angina of effort may be initiated, complicated, or terminated by coronary thrombosis. By this a simple angina (of effort) can be converted into a sort of compound or complicated angina (with shock, etc.). The correlation between anginal pain and vascular lesion is not exact, for the worst cases of angina of effort approximate to coronary thrombosis. Angina of effort is fairly inclusive of all cases of angina pectoris excluding coronary thrombosis, cases with spontaneous attacks of pain at rest being almost invariably subject to angina on exertion.

Too much stress has been laid upon the gravity of angina pectoris. It is inseparable from a risk of sudden death, but this may be deferred for years. In attacks of coronary thrombosis and in the severer grades of angina of effort the pain is extreme and the danger is great, but milder forms are common and often run a tolerable course. There is every gradation between mild angina of effort and severe coronary thrombosis, as there is every grade of coronary disease. Too much attention has been centred on the anginal death, and too little on the anginal life and its management.

IMPORTANCE OF VOCATIONAL GUIDANCE FOR PRESERVATION OF THE HEALTH OF THE INDUSTRIAL WORKER *

BY

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There are no statistics which afford any exact indication of the number of human beings engaged in work definitely unsuited to their capacities. In America it has been estimated that approximately 50 per cent. of the working population are vocationally maladjusted, but this calculation, based solely on questionnaire reports from satisfied and dissatisfied workers, is for various reasons unreliable. In any case, it is certain that the problem of the misfit is one of considerable magnitude, and that there is great need for improved methods of advising young people on the choice of occupation. It is a problem with important economic and sociological aspects, but the present paper is limited to a discussion of unsuitable employment as it affects the individual industrial worker, and, more especially, as it affects his health. In these days there is a growing realization of the importance of mental factors in the causation of sickness; it is clear that the whole man—body and mind—must be taken into account in any discussion on employment and health.

VOCATIONAL GUIDANCE AND THE PHYSICIAN

In this country the part played at present by the medical profession in vocational guidance falls short of the ideal. In school the child is examined at infrequent intervals by the school medical officer, who as a rule is concerned merely with the treatment of physical defects. In some areas the rule is that the child must be examined within two years of leaving school; hence, in certain cases, the medical information available to the child's vocational advisers is approximately two years out of date. Moreover, although there are exceptions to the rule, this information is commonly, for the purpose of vocational guidance, altogether too meagre.

So far as the writer knows, neither the certifying factory surgeon nor the doctor employed by the large

industrial firm can be said—with exceptions—to give guidance in any real sense of the word. These officials render a valuable service by rejecting candidates for work whose physical defects might make them a danger to themselves or to others. But to be rejected is not to be guided; and it is perhaps questionable whether all the rejections are necessary or desirable. It appears that in this country the industrial doctor often sets a uniform standard of physical efficiency for all entrants to the factory, no matter what variations exist in the physical requirements of the individual factory operations. Recent writers have protested against this practice, insisting that *adaptation* of the defective individual, not *elimination*, should be the aim.

"The object of the physical examination is not to make the plant a haven of physically perfect specimens . . . strict exclusion is a selfish policy, which deprives the doctor of his best field for improvement."¹

Further, the industrial doctor not only gives no positive guidance to those whom he rejects, but takes no part, as a rule, in the allocation of suitable work to those whom he accepts. This would seem to be a matter in which his services might be used to greater advantage.

PHYSICAL MISFITS

As to what proportion of the young people annually entering the ranks of industry succeed—despite the efforts of school and industrial medical services—in obtaining employment unsuited to their physical condition, one can only speculate. In an American investigation it was found that of 400 working boys and girls, not specially selected in any way, 49 per cent. had physical defects which were being generally aggravated by their occupation.² In this country a recent investigation of load-carrying among women in a number of different industries showed that there was a definite correspondence between the heaviness of the trade and the strength of the women employed, but the comment was made by the investigators that

"in view of the relatively casual method used in the selection of women, the only probable explanation is that it is a case of the survival of the fittest";

and

"no doubt the 'below par' young persons filter through to a level of work which is within their capacity, but at what cost to the individual one can only guess."³

* Paper read in opening a discussion in the Section of Public Health at the Centenary Meeting of the British Medical Association, London, 1932.