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ANGINA INNOCENS: A CLINICAL STUDY*

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For the last eight years I have fully investigated and personally followed up a large number of patients suffering from cardiac pain. The starting-point in this investigation has been an analysis of the exact nature of the pain complained of. By observing the position of the pain, its radiation or lack of radiation, its relation to exercise, rest, emotion, and posture, it has become clear that patients with heart disease suffer from types of pain which are as characteristic and distinct as are those complained of by patients with gastric disease. These varieties of cardiac pain differ from one another according to the underlying pathological or functional condition.

The pain of coronary thrombosis is characteristic. Its onset is sudden, its severity is greatest at the start, and it tends to subside over a period of days. It may be constant during that period or may recur and remit. The pain of true angina of effort presents an equally distinct clinical picture. An intelligent patient is able to describe its exact quantitative relation to exertion—for example, a speed of one mile per hour may produce no pain, a speed of two miles per hour may result in pain after a walk of two or three hundred yards, and a faster speed or a hill will accelerate its onset. Rest invariably allows the pain to subside.

There are two further types of cardiac pain, the nature of which has not hitherto been exhaustively analysed. Spasmodic angina is an attack of cardiac pain, the onset of which may result from exercise, exposure to cold, emotion, or a heavy meal: it is relieved by amyl nitrite, but it is not exactly proportional to exertion. When once such an attack has started it will have to run its course and may increase in severity, although the patient is at rest. The nature and symptomatology of the fourth type of pain have remained unexplored: it is associated with no definite cardiac disease, is apparently of functional origin, and has a favourable prognosis.

Nomenclature

This variety of pain is the subject of the present communication. It tends to start in the left chest, frequently radiates, has no quantitative relation to exertion, and is found generally in hypersensitive or nervous subjects. The term "pseudo-angina" has in the past been applied to this syndrome; but it is a contradiction in terms, for either a patient is suffering from a pain or he is free of it. The description "neurocirculatory asthenia" is too wide, for it embraces such other symptoms as tachycardia,

palpitation, and certain nervous manifestations. There appears to be no serious objection to labelling this pain "angina innocens," for this suggests the cardiac and at the same time the innocent nature of it. There is an analogy with diabetes.

It may be thought that the word angina, even though qualified by "innocens," is not desirable in view of the possible implication of severe cardiac disease. This objection is inadequate for two reasons. It often happens that a patient with angina innocens has been told that he has "angina," and the same statement is perhaps more often made to the patient's relatives. One of the patients described in this paper was told at a hospital that he was suffering from angina and would have to live a very retired life. Being introspective, the young man's mental state became such that he seriously contemplated suicide. This case shows that there may be sufficient similarity between angina innocens and angina of effort, or spasmodic angina, if a careful history is not taken, for confusion to arise. Such a label as left submammary pain is too vague. A second reason for facing the fact that the pain has many of the characteristics of an organic cardiac pain is that the patient, from hearing of acquaintances dying with substernal pain radiating to the arm, may confound the two conditions and feel that a non-committal label is an attempt at concealment or prevarication on the part of his physician. It would appear more practical and more logical, therefore, to term the condition "angina innocens," stressing the latter word.

The observations in this paper are based upon the detailed study of thirty-eight patients suffering from this type of pain who have been under investigation for varying periods since 1929. Simultaneously a study of a large number of patients complaining of the other forms of cardiac pain has served as a background, against which the present syndrome has stood out more clearly with the passage of time.

Survey of the Literature

That cardiac pain can occur in the absence of organic heart disease has long been recognized. The chief observation upon which this view is founded is that patients may not die with cardiac pain and that many recover completely. This variety of pain has been labelled in the past as pseudo-angina. Sir William Broadbent (1897), who used this term, referred to the tendency of the pain to be left-sided, localized near the apex, and associated with hyperaesthesia. But the credit for the first full account belongs to Da Costa (1871). He notes that the

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pain is in many cases constant, though it may be accentuated by exercise, and describes it as occurring in paroxysms or in the form of an intermittent lancinating stab. Stating that it is frequent after exertion, he also observes that in a few cases the pain was not accentuated during exercise but even relieved by it. Da Costa describes its localization near the cardiac apex and its radiation to the left arm, and lays stress upon the presence of hyperaesthesia, which often made the pressure of military accoutrements exceedingly unpleasant. Finally he notes that the heart rate may be fast in the erect position and slow when the patient is recumbent. Very little has since been added to this description.

Friedlander and Freyhof (1918) mention the frequency of sore throats as an aetiological factor, refer to the left-sided pain and hyperalgesia, and bring out the fact that palpitation, nervousness, and giddiness are common in these patients. Danielopolu (1927) considers that although there are cases of non-organic angina they are very rare. He states that the condition is commoner in women, and stresses the precordial hyperaesthesia, marked tachycardia, violent palpitation, and other evidence of an abnormal function of the vegetative nervous system. He also remarks that the disease is not fatal, but that it is very difficult to distinguish it from organic angina. Paul White (1931) writes: "The precordial pain is a dull or heavy ache as a rule, lasting for hours and not radiating. But occasionally it is interspersed with sharp stabbing sensations. A substernal oppression is unusual, though circulatory asthenia may, and in fact frequently does, complicate angina pectoris." An interesting analysis of patients belonging to this group was made by Doris Baker (1930). Her series was collected from a hospital out-patient department; it comprised thirty-two cases in which there was a history of up to twenty-nine years, and twenty others. In these cases there was a history of rheumatism in only six; there is no mention of the frequency of sore throats or of other specific infections. The patients apparently had normal cardiovascular systems, except that in ten there was high blood pressure and in four mitral stenosis. They were generally of the thin, anxious type. The pain was worse after exertion, having no quantitative relation to it; in most cases it was a dull ache, though a sharp stab was occasionally complained of. It was accentuated when the patient was tired. Exhaustion, as in the present series, was a prominent symptom; sighing and palpitation were frequent. Fainting was said to be unusual. The chief physical change was hyperaesthesia, which was present in 68 per cent.

Diagnostic Criteria

The usual criterion for differentiating harmless from harmful anginal pain has in the past generally been the physical condition of the heart. If there is evidence of cardiac disease it has been assumed that the cardiac pain is related to it; in the absence of abnormality it has been assumed that the cardiac pain has no organic origin. This method of approach appears to me to be in some ways illogical. It is well known that anginal pain may be caused by obstruction to the coronary flow by a syphilitic aortitis, that in these cases the heart may show no abnormality when examined by all available means, and that the pain frequently subsides as a result of vigorous anti-syphilitic treatment. Here there is no doubt that the pain is due to an organic lesion. Moreover, it is illogical to assume that pain has no organic basis until a definite physical change can be found, for the sensitivity of the nervous system varies so much that a lesion which in one

individual produces no symptoms can, in a more sensitive person, give rise to severe pain. In the latter case the pain may be prominent when the underlying organic basis is so insignificant as to escape detection by any method of examination.

Three criteria have been used here for the diagnosis of angina innocens: first, that the pain is not proportional to exercise; secondly, that there is no evidence of cardiovascular disease; and, thirdly, that the course of the disease in patients observed carefully proves its innocent nature. The absence of cardiovascular disease was demonstrated clinically, electrocardiographically, and radiologically. The absence of any deleterious lesion is shown by the following facts with regard to twenty-one of the thirty-eight cases. Six of the patients have recovered, four of these having been seen five years after the observed presence of the pain. Eight patients improved or were no worse after five years, and one after three years. Seven were so young as to make it hardly possible that they could be suffering from coronary disease, their ages being 12½, 15, 15, 16, 17, 17, and 20 years. This last patient of 20, in spite of attacks of pain, had been for months in the habit of cycling distances of sixty to eighty miles each week-end.

In order to give a wider view of the syndrome sixteen more cases with pain of an identical character have been added to the above twenty-one. They consist of eight cases in which the observed duration of the pain has been less than three years and eight of organic heart disease. The latter group is of particular interest: three patients with mitral disease have suffered from an angina innocens type of pain for five years; three with hyperpiesis have remained otherwise well for four years, and one with hyperpiesis for three years. In the last case the pain was also associated with a microcytic anaemia, and disappeared when this was treated. In the remaining patient the only sign of a cardiac lesion was a P-R interval of 0.22 of a second. The fact that these latter patients have remained reasonably well, and with no signs of failure in spite of organic heart disease, is additional proof that this particular type of pain is "innocent."

Methods of Examination

In all cases a full history was taken. In the past history the following points were specifically investigated: rheumatic fever, growing pains, chorea, scarlet fever, tonsillitis, diphtheria, syphilis, and the history of the puerperium. Note was also taken of other diseases or surgical operations. A history of previous mental stress or trouble was not specially inquired into. The following symptoms were investigated in each patient: pain, dyspnoea, sighing, orthopnoea, feeling of constriction, palpitation, exhaustion, cough, giddiness, dyspepsia, state of the bowels, catamenia, frequency of micturition. With regard to the pain the points elicited were: its character, position, radiation, duration, relation to exercise and emotion. The patients were asked whether the pain occurred during exercise, in a quantitative manner, or whether it would be worse when the exercise had been finished; also whether it was ever present when they were resting or in bed. Inquiry was made about their present and past work, their ability to play games, to walk (with special reference to hills), to climb stairs, and to run; and also the quality of their sleep and their habits as regards smoking and alcohol.

Physical examination included special notice of: the presence of dyspnoea, orthopnoea, cyanosis, pallor, pulsation, clubbing of the fingers, and tremor; the pulse rate (in twenty-three cases the pulse rate and blood pressure were measured, both standing and lying); the state of the peripheral arteries and veins. The heart was examined by inspection, palpation of the apex beat, palpation of the praecordium for abnormal impulse or thrills; the position of the apex beat; the area

of cardiac dullness; and the sounds were noted. Precordial hyperaesthesia was tested for in every case, and the lungs and abdomen examined. An electrocardiograph was taken, and in some cases repeated. The Wassermann test was done. A six-foot film of the heart was taken, the contour of the heart noted, and the heart-chest ratio measured.

Age and Sex Incidence

Of the thirty-eight patients who were examined twenty-two were females and sixteen males. The ages at which the syndrome manifested itself were as follows: of the twenty-two females eleven were between the ages of 36 and 46; ten of the sixteen males were between 41 and 55. Four males and five females were between 12 and 25 years of age. The youngest female was a girl of 15. In one case the onset was between the ages of 56 and 65.

Past History

In the past history two facts stand out. The first is the high incidence of rheumatic infections and tonsillitis, and the second the frequency of psychological disorders. In twenty of the thirty-eight patients there was a rheumatic history; rheumatic fever occurred in seven, growing pains in twelve, chorea in two, and scarlet fever in six. Twenty-one of the thirty-eight patients had had definite tonsillitis. In twelve of these the sore throat had been recent, and in nine there was a history of sore throat in the past. In the latter group one patient had had sore throats for twenty years, two for twelve years, and one until tonsillectomy at the age of 21. No quinsies were noted, the tonsillar inflammation being apparently more likely to run the course of the recurrent hyperaemic type of inflammation found in acute rheumatism. No definite relation was found between an attack of tonsillitis and the onset of cardiac pain. Nor was there evidence that tonsillectomy relieved the pain. In one case angina innocens appeared immediately after an attack of rheumatic fever and persisted for eighteen months.

Sixteen of the thirty-eight cases had had definite psychological difficulties. Two had attended the Maudsley Hospital. Six had suffered from severe domestic worry and depression. Two complained of severe night terrors and nightmares. One had been a sleep-walker and had been in the habit of diving off the end of his bed. One had had a functional hemiplegia, and another functional dysphagia and dyspnoea. One had seen Sir Henry Head for psychasthenia, and three had suffered from various phobias—syphilophobia, cardiophobia, and agoraphobia. A history was obtained of the following diseases: syphilis, adequately treated in three cases; influenza in two; and diphtheria, typhoid, paratyphoid, malaria, jaundice, diabetes, nephritis, asthma, epilepsy, mucous colitis, and gonorrhoea—each in one case.

Symptoms

In addition to the precordial pain the other symptoms were as follows. Marked lassitude was complained of in thirty cases; this was the outstanding symptom, apart from pain; it was severe, and almost invariably more complained of than shortness of breath. If the question, "Which causes you the most trouble, tiredness or shortness of breath?" was put the answer would almost invariably be "tiredness." Shortness of breath of a not very pronounced degree occurred in twenty-nine cases. Orthopnoea was found in seven cases, one of them a case of mitral stenosis and one of hyperpiesis: in the other five the symptom was not prominent. Palpitation was complained of by twenty-nine patients. It was precordial in position, being generally felt over the apex beat. It

was not, as a rule, noticed at the base of the throat, as is often the case in paroxysmal tachycardia or in patients with hyperpiesis. Giddiness was a symptom in twenty-seven cases, and faintness, without loss of consciousness, in seven. Thirteen patients gave a history of fainting; in eleven of these the syncope immediately succeeded a sudden severe attack of left-sided precordial pain, generally radiating to the left arm. The details of this syndrome will be discussed later. It is thus apparent that twenty of the thirty-eight patients suffered from vasomotor attacks. Sudden painful stabs, possibly due to premature beats, were complained of by ten patients: a dull, left-sided precordial ache often followed these.

Twenty-nine patients complained of a sense of constriction. This was generally left-sided and was not of the typical substernal central variety found in organic disease of the coronary vessels. A feeling of heaviness over the left praecordium was a commoner symptom than a sense of internal pressure or suffocation. Sighing, in spite of its frequency in patients with neurocirculatory asthenia, had not been consciously noticed except by four patients, although it is probable that had careful respiratory tracings been taken the respiratory irregularity demonstrated in cases of neurocirculatory asthenia by Paul White would probably have been found more often. Dyspepsia was present in fourteen cases, and in several appeared to be a predisposing or trigger symptom, for successful treatment of the dyspepsia generally led to considerable diminution in the severity of the cardiac pain. In no case, however, could it be said that the dyspepsia was the cause of the cardiac pain.

Character and Position of the Pain

In thirty-three out of thirty-eight cases the pain was felt on the left side of the chest, either over the apex of the heart or higher over the praecordium. It was felt in the mid-line by five patients. Radiation to the left arm was present in twenty-six cases and to the left shoulder in five. In twelve the pain remained localized. The relation of the pain to exercise is one of the most characteristic and important points in the symptomatology. There was no quantitative relation to exercise in any one of these patients. Although it could be said that in twenty-four of them the pain was increased as a result of exertion, there was no definite quantitative increase with more, or decrease with less, exercise; moreover, in most of these patients the pain was chiefly noticed when the exercise had ceased, and generally persisted for some hours afterwards. In some cases, for example, a hard day's washing or other physical exercise could be performed, but this would subsequently be followed for a period of hours or even days by a left-sided dull precordial pain. The pain also tended to be produced or accentuated by excitement in sixteen cases: a mother complained that this was so when she was worried about her children. Standing produced the pain in seven cases; and in twenty it was present at night when the patient was in bed. In two cases it appeared to be related to food. The duration of the pain was variable. In four cases the attacks would only last for a period of less than ten minutes, but in the majority they would persist for some hours, and in one-third of the cases for days. Soreness of the chest, or hyperaesthesia, or intolerance of precordial pressure (such as might be produced by braces or by hard objects in a breast pocket) was definitely associated with the pain. This soreness was always accentuated after a severe attack. The dull ache and the associated soreness might follow the sudden momentary stabbing pains described above.

Syncopal Type of Angina Innocens

The following cases are examples of a special syncopal type of angina innocens in which the pain starts over the praecordium, or rarely in the mid-line, and radiates to the arm. The onset is almost always abrupt, though most patients are able to reach a chair or a couch in time to sit down. The period of pain, which is long enough to be remembered and to allow of the radiation to the arm to be noticed, is followed by an attack of fainting or faintness; the sharpness of the pain is believed by most of the patients to be the cause of the fainting attack. The loss of consciousness may not be complete. In several cases these syncopal attacks were the first manifestations of the commoner type of angina innocens described above. Generally the attacks are somewhat infrequent, but in one case amounted occasionally to ten a day.

In this patient the attacks had been spread over a period of six years, and at the end of that time the heart was apparently perfectly normal.

A second patient was seized with a sudden attack of precordial pain while washing, became dizzy and half unconscious, but managed to get back to bed. The pain in this case started in the left chest, radiated to the left arm, and lasted for about half a minute.

The third patient was seized with an attack soon after rising, and ascribed it to excitement at the prospect of seeing relations.

The fourth patient had four attacks in eight months: the attacks lasted for the unusually long period of ten to fifteen minutes before she lost consciousness; the onset was sudden, and the pain started in the praecordium and radiated through to the back. At this time the pain came on only with the syncopal attacks, although previously she had typical precordial pain after exercise. She had made a complete recovery when seen eleven months later.

The fifth patient had a severe sudden pain in the left chest, radiating to the left axilla, not to the arm, and then fainted.

The sixth patient had fainted two or three times, but only in association with precordial pain, which preceded the attack by a few seconds.

The seventh patient, while getting her husband's breakfast, had a sudden precordial pain, which passed down the left arm, and a sense of tightness in the throat. There was also a feeling of precordial constriction, and she then fainted.

The eighth patient, while stooping down to clean a stove, was seized with a sudden severe attack of pain in the chest, radiating to the left arm, and felt very faint. Subsequently she had several similar attacks, one in church, associated with fainting.

The syndrome was found in twelve other patients. It would appear that this type of attack often accompanies severe mental or psychological stress: two of the patients stated that their domestic life was unhappy, one suffered from syphilophobia, one had had epilepsy, and one had been treated for "psychasthenia." Standing and excitement appear to be immediately predisposing factors.

Physical Findings

Examination of the heart in thirty-eight cases of angina innocens gave the following results. The apex beat was internal to the mid-clavicular line in thirty-five. In three it was displaced to the left: two of these had hyperpiesis, and the third was suffering from a microcytic anaemia which cleared up under treatment. Precordial hyperaesthesia was present in twenty-four cases out of thirty-eight. This was frequently very marked, and might be localized to the apex or be more diffusely spread over the left precordial region: some patients winced when the praecordium was palpated. In eight cases the heart sounds were abnormal; in three of these there was a mitral

stenosis and in one mitral regurgitation; in one case a systolic murmur was heard at the apex, and in another a systolic murmur which was loudest at the apex and conducted to the aortic base. No abnormality was discovered in the lungs. In thirty-seven cases the abdomen was normal, but in one tenderness was present over the right renal area.

The blood-pressure findings in this series of cases of angina innocens are worth careful attention. In thirty-one cases the recumbent systolic and diastolic pressures were normal. In five the blood pressure was subnormal; in two there was some hyperpiesis, which responded to dietetic treatment in one instance. In twenty-eight cases the blood pressure was taken with the patient in two positions: after recumbency for five minutes, and again after assumption of the erect posture for two or three minutes. In twenty-five of these patients there was a marked fall in the systolic pressure in the erect posture, which was associated as a rule with an increase in the heart rate. The average fall for the twenty-five cases was 21 mm. Hg, and this was accompanied by an average increase in the pulse rate of ten beats a minute. The maximum fall was 50 mm. Hg, but this was in a patient with hyperpiesis. In three instances no change in the blood pressure or the pulse rate was observed in readings taken standing and lying. These findings are somewhat significant in view of the frequency of giddiness and fainting attacks in angina innocens.

Radiography disclosed abnormality in four cases, and some slight prominence of the pulmonary conus in the patients with mitral stenosis. In none of these three was there any gross deviation from the normal. Two of the patients with left ventricular enlargement had hyperpiesis. In twenty-nine cases the electrocardiographic tracing was normal; in eight there was some abnormality. These cases all showed left axis deviation, which in five was physiological for the age of the patient. In no instance was there any abnormality of the T waves. In one case the tracing is lacking. The Wassermann reaction was negative in twenty-nine cases; in eight the blood was not examined; and in one patient who had previously had a positive reaction the result was now negative.

Differential Diagnosis

The pain of angina of effort is characteristic, and a careful history will in all cases differentiate the condition from angina innocens. If pain is only complained of as occurring during exertion, if it is related to exertion and never arises at rest or at night, it may be taken that the patient is not suffering from angina innocens but from angina of effort.

A group of patients suffering from pure angina of effort is at the present moment the subject of another investigation. In thirty-seven of these there is no history of gross mental abnormality or psychological instability, such as was found in some of the patients with angina innocens. In thirty-three there is definite organic cardiovascular disease; one patient was probably so affected, and the remaining three were diabetics, in which condition coronary atheroma is frequently found.

The presence or absence of radiation to the arms is of no help in differential diagnosis, and although the pain of angina of effort is in the majority of cases central this is not absolutely constant. In angina innocens, however, the pain is left-sided in nearly every case. Lassitude as a marked symptom, in contradistinction to shortness of breath, has already been stressed, and it can again be stated that the lassitude in angina of effort, though present

in some degree, is not severe. Palpitation is prominent in angina innocens and was complained of by twenty-nine out of the thirty-nine patients, whereas in only nine of those with angina of effort was this at all severe, though three admitted to having suffered from it in a slight degree. Fainting or syncopal attacks strongly support the diagnosis of angina innocens, for none of the thirty-seven patients with angina of effort suffered from these, their only analogous symptom being in the case of hyperpetics, some temporary dizziness after stooping, or on rising suddenly. Seventeen of the patients with angina innocens gave a history of fainting and none of those with angina of effort did so. Orthopnoea is a point in favour of angina of effort, for twelve of the thirty-seven patients complained of it in a marked degree and two slightly. Definite evidence of cardiovascular disease suggests angina of effort, and in the complete absence of all clinical, radiological, and electrocardiographical evidence of organic cardiovascular disease angina innocens is the more likely diagnosis; this is, of course, not conclusive, for a slight coronary lesion may obviously be present in a sensitive individual or in syphilitic aortitis and yet be physically undiagnosable. Mitral stenosis is an exception to the above rule, for patients with this lesion, when they suffer from cardiac pain, complain of pain of the angina innocens type. The blood pressure in angina innocens is generally normal, subnormal, or shows a definite tendency to vary with posture; that in angina of effort is frequently raised.

There should be no difficulty in distinguishing this condition from coronary thrombosis. The syncopal type of angina innocens, starting with a severe attack of precordial pain and followed by faintness or a syncopal attack, may possibly suggest this diagnosis in some cases. There is, however, very rarely any loss of consciousness in coronary thrombosis. The absence of fever, cardiac enlargement, leucocytosis, and any electrocardiographic abnormality will confirm the diagnosis.

Paroxysmal tachycardia or auricular flutter may at the onset of an attack be associated with cardiac pain radiating to the arm and with faintness or fainting. Here a very high heart rate, which shows no variation from minute to minute, is characteristic. The electrocardiograph will again clinch the diagnosis.

The syncopal type of angina innocens may suggest an epileptic attack with a visceral aura. The onset, however, is less abrupt than that of epilepsy. The patient generally has time to reach a chair or a couch before losing consciousness. There is a sufficient period to allow of the type of pain being recognized, and later described. The tongue is not bitten. Convulsions do not occur. There is no incontinence of urine. The course of the disease is unlike that of epilepsy, for there is no progressive increase in the severity of the symptoms. The patients, on the other hand, generally show steady, if slow, improvement. (Since completing the above study I have seen a patient who suffered both from epilepsy and from angina innocens. In no instance were her attacks of epileptic unconsciousness associated with the presence, or any exacerbation, of the cardiac pain.)

Other Types of Cardiac Pain

If it be conceded that the pictures of angina of effort and of angina innocens are sufficiently distinct, anyone with experience of patients with cardiac pain will agree that these two conditions do not account for all cases. Spasmodic angina is one condition in which a severe attack of cardiac pain arises. The exciting cause may be exercise, anger, or possibly some digestive upset. The

characteristic of the attack is that when it has once started it has, in the words of one patient, "to run its course." It would appear as though the exercise or emotion started some definite pathological mechanism and that the pain persisted till such mechanism had ceased. The therapeutic action of amyl nitrite in these attacks suggests that spasmodic angina may be due to coronary spasm. The present investigation has as yet thrown no definite light on the mechanism of spasmodic angina, but it has served to show clearly that there is such a symptom-complex, and that this can quite easily be recognized. More work is necessary before any deductions can be made as to why some patients with angina of effort also suffer from attacks of spasmodic angina, or why a patient with angina innocens very occasionally appears also to suffer from these attacks. It would seem to be sufficient for the time being to analyse and to separate out the two clinical entities of angina of effort and angina innocens.

Possible Mechanism of the Pain

It is difficult to understand how the pain of angina innocens is produced. There is quite obviously no coronary disease. The severe spasmodic type of attack may just possibly be associated with some temporary coronary spasm, but of this there is as yet no evidence. The known factors which may be concerned with the production of pain in these cases are worth enumerating. There is no doubt that the angina innocens type of pain is that found exclusively in those patients with mitral stenosis who suffer from cardiac pain. There may be some analogy between these cases of mitral stenosis and those with angina innocens and an apparently normal heart. In mitral stenosis the left ventricle is contracting in an abnormal manner. The diastolic filling of the chamber is probably somewhat reduced, so that the systole tends to be over-emphatic. The high-pitched, accentuated first sound, and the slight but persistent degree of tachycardia, bear witness to this continuous cardiac over-action. In these circumstances the patient's heart or praecordium may become gradually sensitive to the persistent knocking and the sensation may ultimately be magnified into pain.

A second factor is an over-active central nervous system, due to psychological or endocrine factors. The vasomotor system is also unduly labile: the sagging of the blood pressure in the erect position, the history of syncopal attacks, and, finally, the curious association between a severe attack of pain and an almost immediate fainting attack, all suggest this system may be at fault. A final factor, the mechanism of which still remains obscure, is the known association between chronic infection and "irritable heart." In Da Costa's experience the infection was largely of a dysenteric nature, but in the present series of cases it would appear to have been pharyngeal.

Prognosis

Of the thirty-eight cases twenty-one have already been considered from the prognostic point of view in determining the criteria for the establishment of the syndrome, angina innocens. If all thirty-eight cases are analysed with reference, not to the period during which they have been under personal observation, but to the length of time they have suffered from the pain, it is found that some have recovered and that most have definitely improved. Six patients have completely recovered; twenty have improved—in two of these there is a history of pain for fifteen years, in eight for seven to nine years, in four for six years, and in five for two years. In eight the pain

has persisted with remissions and relapses, and is on the whole unchanged. One of this final group has had symptoms for twelve, two for seven, three for four to five years, and two for two years. It is thus apparent that the prognosis is very good as regards life; but it is also shown that the symptoms on the whole tend to be chronic. This chronicity, when considered in conjunction with the absence of evidence of physical disease, suggests very strongly that, although there may be some slight physical basis, the nervous or functional element preponderates. It is also clear that pain of this type has no bad prognostic significance, even if the patient is suffering from an organic cardiac lesion.

Conclusions

1. A cardiac pain of an anginal nature is described, the characteristics of which enable it to be differentiated as "innocent": the term "angina innocens" is suggested.
2. The differentiation of angina innocens from other types of cardiac pain is described.
3. An account is given of a syncopal variety of the syndrome.
4. The prognosis as regards life is good, but the duration of the pain, with remissions, is often protracted.
5. Factors possibly concerned in the production of the pain are discussed.

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M. Sallick (*Ann. Surg.*, November, 1936, p. 853) has investigated the late results in seventy-four cases of acute perforated peptic ulcer treated by simple suture alone. In the majority of cases at least three years had elapsed since operation, and in no case was the post-operative period less than one year. All the patients were male, and during the last ten years it was noted that not a single case of acute perforation of peptic ulcer occurred in a female. The youngest patient was 19 and the oldest 67, the largest number of cases being in the 30-40 age period. The majority of acute perforations were found in gastric ulcers, forty-one being situated proximal to the pylorus, twenty-six lesions were in the duodenum, and seven were found at the pylorus. All the duodenal perforations were on the anterior surface or superior border of the intestine; of the stomach perforations four were through its posterior wall into the lesser cavity, and the remainder were equally divided between the lesser curvature and the anterior surface. In the series sixty-two patients (86 per cent.) had a history of typical ulcer symptoms, and diagnosis was easy in nearly every instance. Operation consisted of simple closure in three layers except for three cases, in which a single suture layer only was used. Only six cases were drained. There were eight deaths in the series, the highest mortality rate being noted in patients over 50. The mortality percentage was found to rise directly and sharply with delay in operation. Follow-up results were obtained in forty-five of the total sixty-six patients who survived operation, and it was seen that twenty-nine were found to have had further gastric trouble, a recurrence of 64 per cent. It is considered that the low mortality rate and excellent early results warrant the use of simple closure as a routine procedure in cases of acute perforation, but further medical or surgical care will be required in most instances.

PREVENTION OF DISEASE IN INDUSTRY*

BY

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Industrial medicine is the practice of medical supervision, preventive medicine, and public health within the confines of an industry. Its aim is to safeguard the health of the employee and to minimize time lost from work because of sickness or of accidents. Though one of its functions is treatment it is mainly an advisory and preventive service. The industrial medical officer has to co-operate with managers, workers, engineers, chemists, and architects. He has to discover faults in the working environment and try to find remedies. One of his most interesting duties is to bring into the effective service of industry the discoveries of the research worker.

The Factory Department of the Home Office is constantly engaged in effective and progressive work for the prevention of disease in industry. It numbers on its staff men and women who are among the greatest living authorities on different aspects of industrial hygiene and toxicology. Their profound store of knowledge, constant helpfulness, and unfailing courtesy industry sometimes fails to appreciate to the full. It is important that doctors should have the special knowledge and training which enables them to offer advice to the employer as to how his industry may be carried out in safety. At present, with a few exceptions, it is only the medical inspectors of factories who have this knowledge. No doubt with the growth of this branch of preventive medicine more doctors will make themselves competent in this interesting work.

The main principles underlying the prevention of disease in industry can be summarized under fifteen headings.

1.—Protection of the Workman by Law

In this country the number of diseases notifiable under the Factory and Workshops Acts and subject to compensation under the Workmen's Compensation Acts indicates the existence of an alert and enlightened legislature. From 1878 onwards provisions were made to control dangerous trades by so-called special rules of the Home Office. The early knowledge of industrial diseases was obtained by placing an obligation on the medical practitioner and employer to notify them to the Chief Inspector of Factories. Between 1896 and 1927 anthrax, toxic jaundice, epitheliomatous ulceration, chrome ulceration, chronic benzene poisoning, and poisoning by lead, phosphorus, arsenic, mercury, carbon bisulphide, and aniline were made notifiable. The scope of the Workmen's Compensation Acts is wider than that of the Factory Acts, as they are intended to cover all cases of sickness of strictly occupational origin, the individual being considered and not merely the conditions under which he works. There are now some thirty-eight scheduled diseases, and of these the diseases of miners and dermatitis alone constitute more than 90 per cent. of the cases compensated. New occupational risks constantly arise; asbestosis and dioxan poisoning can be quoted as dangers recently brought under control. Sometimes great expansion will occur in one trade, as was the case with the manufacture of accumulators from 1922 to 1924 owing to an increase in the use of motor cars and wireless apparatus. Such expansion of an industry makes great demands on those responsible for the prevention of disease in the factories concerned.

* Introductory remarks to a discussion on occupational diseases, Medical Society of London, March 22, 1937.