The distinction of gall-bladder lesions from cardiac damage may be exceedingly difficult, particularly in the first attack. On account of the comfort and reassurance which come from the knowledge that irreversible cardiac damage has not been sustained, if the acute phase of a massive pulmonary embolism is survived it is always worth while to endeavour clearly to distinguish this condition from an acute coronary occlusion, which it can on occasions mimic closely.

Difficulties also arise through the misinterpretation of certain physical signs. Apart from those arising locally in the heart, such as systolic murmurs and extraneous noises, hypotension and oedema can give rise to confusion, particularly when the exact nature of the illness is obscure.

Errors in diagnosis can be lessened by assessing the symptoms with care, checking the body weight against standard tables, estimating the haemoglobin level, considering the personality of the patient, and accepting as evidence of organic cardiac damage only well-recognized and indisputable physical signs.

There is, however, a cardiovascular borderland where physiological adaptations and pathological change overlap. The heart in pregnancy is an example. Without close scrutiny of the heart and careful assessment of the degree of exertional dyspnoea in relation to the degree of peripheral oedema, both of which are common in the later months of pregnancy, the ranks of those women known to suffer from rheumatic heart disease may commonly be increased by at least a fictitious 10% in whom the heart is fundamentally sound. On occasions it may be necessary to wait until after the confinement before a final opinion can be expressed regarding the state of the heart, so closely can the temporary adaptations of pregnancy mimic rheumatic heart disease.

It is a good rule never to condemn a heart without ample and conclusive proof.

I am indebted to my colleague, Dr. I. G. W. Hill, for the set of electrocardiograms recorded after a massive pulmonary infarct.

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A scheme for smallpox treatment is suggested by Johnstone and Fluker in the *Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service* (1949, 8, 188). They suggest that in confluent and haemorrhagic cases penicillin, in doses of not less than 0.5 mega unit daily for four days, should be given within 24 to 48 hours of the appearance of the focal eruption. When the vesicles become umbilicated, intramuscular streptomycin should be given in a daily dose of 1 g., and the daily amount of penicillin should, if necessary, be increased to 2–5 mega units. If the case is severe streptomycin should be continued for about a fortnight, or until the lesions have become well dried. The penicillin should be administered until most of the scabs have separated, though there can be no precise rule on this point. This scheme was put into operation by the authors for three cases. The effect on the skin lesions was remarkable, and far less scarring resulted than would have been expected.

PAIN IN THE CHEST WALL SIMULATING HEART DISEASE

BY

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Pain in the chest offers a wide field in differential diagnosis, and is particularly interesting to the clinician because in the majority of cases simple clinical methods are sufficient to establish its origin and nature. In spite of this, mistakes are frequent, and 10% of patients referred to hospital with a diagnosis of angina are found in fact to be suffering from pain arising in the chest wall only, with no evidence whatever of underlying cardiac disease. If the patients sent for electrocardiography on the suspicion of cardiac disease are included this percentage is very much higher. Many of these have been induced to give up their employment or been relegated to a life of invalidism, and in all of them the diagnosis has caused unnecessary anxiety.

The frequency with which such patients are seen in routine out-patient work emphasizes the need for a reorientation towards pain in the chest, and suggests that in clinical teaching pride of place is too often given to angina pectoris in explanation of the pain and too little regard is paid to local structural causes.

When the pain is specifically related to exertion it too frequently breeds in the mind of the patient or his doctor the thought of cardiac disease, particularly in over-anxious individuals and in middle-aged men and women who have some other symptom such as breathlessness to act as a pointer in this direction. In a proportion of such cases organic disease of the cardiovascular system can be excluded by a sufficiently detailed history and a careful examination designed not only to establish the normality of the heart but also to discover some local cause to account for the symptoms.

This paper is based on a follow-up of 50 consecutive cases to which a diagnosis of coronary disease had been wrongfully applied, and in each case the cause of the pain was found to be in the structures of the chest wall. It takes no account of the many cases in which coronary disease is simulated by visceral disease elsewhere or is an expression of an underlying anxiety neurosis.

In almost all the cases the mistake in diagnosis had been due to the readiness with which precordial pain on effort had been accepted as indicating heart disease without realizing the need for the closest questioning on the time relationship between the effort and the pain and the exact nature of the effort involved.

The cases under review were classified as follows: (1) muscle strain affecting the chest wall and attachments of the diaphragm, 16 cases; (2) fibrositis of the chest wall, 20 cases; (3) kyphoscoliosis and osteoarthritis of spine, 14 cases.

Cases of disease of the lung and p'euira invading the chest wall were not included, though they are well recognized as an occasional cause of difficulty in diagnosis. No instance of a diaphragmatic hernia causing pain simulating coronary disease occurred in the present series, but such a condition might be borne in mind in assessing a difficult case. The salient points of a few selected cases are given for illustrative purposes.

Muscle Strain

Muscle strain can be of two types, acute and chronic. The acute ones simulate a coronary thrombosis and the more chronic ones angina of effort. Of the cases under review, five were acute and eleven chronic.

The acute ones are related to some specific exertion, usually some heavy lifting or muscle strain in an abnormal position. The pain comes on at the time of the exertion and is sudden in onset. It interferes with breathing and movement and may persist for some days, though the acute stage quickly passes off. It may be associated with palpitation and sweating, or even faintness in a person of nervous temperament, though it is not associated with signs of shock.

On examination there may or may not be local tenderness in painful areas, but the pain can always be reproduced by muscle-stretching against resistance. The simplest test is to get the patient to raise the arms and then press forward against resistance applied to the hands. In some cases it is only turning the trunk, or some sudden and unexpected movement, that will reproduce the pain. Following an acute phase the pain may mimic angina by its relation to effort. These cases respond satisfactorily to rest and support with strapping.

The points of differentiation from coronary thrombosis are the sudden onset at the height of exertion, the absence of shock, the close relation to posture and movement, and the relief obtained from the application of support. Specialized investigation is necessary only in a few doubtful cases in which the general disturbance is out of proportion to the local trauma.

Case 1

A man aged 51 was seen by his doctor in August, 1945, for a severe attack of pain in the praecordium which had come on while cleaning a car. It was associated with difficulty in breathing, was sharp and stabbing in character, and was made worse by movement. It was associated with a dull background of pain which lasted two days. A diagnosis of coronary disease had been made. A detailed history showed that the pain had first come on while bending down in a twisted position to polish the wheel of his car. It had been of postural origin and recurred on further bending. During his time in bed it had been made worse on turning in bed or on some movements of the arm, such as reaching for his tea-tray, etc. Clinical examination two weeks after the accident showed no evidence of cardiovascular disease; there was no local tenderness or anything to suggest intrathoracic disease. The pain could, however, be reproduced if the patient adopted the pose of the Discobolus—the attitude in which it had first occurred. A diagnosis of chest-wall pain was therefore made and he was allowed to return to work. Inquiry in 1949 showed that he had had no further trouble. The important points in the differential diagnosis were that the pain was of a sharp stabbing quality at first, that it came on while the patient was bending, and that it could be reproduced by specific movements, particularly by the movement which originated the pain.

Such cases do not require further specialized forms of investigation unless the shadow of heart disease has already been cast on the patient, and indeed they may do harm in the nervous subject.

Case 2

A man aged 40 was seen in 1945. While lifting a motor-car bonnet he felt an excruciating pain in the lower part of the chest. He was unable to breathe except in short gasps, and as he was alone he sat on the running-board of his car for ten minutes before being able to move. He then shuffled his way for 50 yards (46 metres) to his house and was subsequently sent to hospital as a case of coronary thrombosis. When he was seen at hospital the pain had abated somewhat, but he still preferred a crouching position. The pain was localized to

the neighbourhood of the xiphisternum and was made worse if he sat upright or attempted to take a deep breath. Clinical examination showed no evidence of an intrathoracic or cardiac lesion and a confident diagnosis of muscle strain was made. The pain was quickly relieved by strapping. In reply to a questionnaire in 1949 he stated that he had had no recurrence.

The following case illustrates a difficulty which may arise when pain in the chest radiates to the shoulder.

Case 3

A woman aged 38 complained of pain in the region of the xiphisternum radiating to the left shoulder and associated with breathlessness. The pain came on while carrying a carpet up some stairs. It was of sudden onset and was so severe that she had to sit on the stairs until she could be helped to bed by her husband. After the first attack the pain recurred over a period of an hour whenever she tried to breathe normally, and she was forced to take shallow respirations, with a consequent feeling of dyspnoea. No abnormality could be found when she was examined a fortnight later, and it was felt that the history was sufficiently indicative of a strain of the diaphragmatic attachments to the chest wall to prevent any serious doubts about the diagnosis. If, however, a similar symptom occurred in a middle-aged man, difficulty might arise unless a detailed history were taken. The patient might well have had an attack of angina while carrying the carpet upstairs, but the differentiating points were the sudden stabbing nature of the pain and its relation to the act of breathing.

The more chronic cases may follow immediately on some acute strain or injury, or there may be a considerable interval between the trauma and the onset of angina-like pain, so that it is only on specific questioning that the patient may recall the injury.

The effect on the heart of non-penetrating injuries in the region of the praecordium has recently received much study, and there is no doubt that serious cardiac damage may occasionally result from trauma in this area. It is much more common, however, for a cardiac neurosis to be engrafted on to a local chest-wall pain or for the pain to be a local traumatic one which simulates angina by its relation to exertion. It is possible that some of the cases described in the literature as angina following trauma are in reality of this type. In perusing the case records of Warburg (1938), who described 197 cases of heart disease following trauma, one is impressed by the fact that chest-wall pain is not mentioned as a differential diagnosis even in those who showed no electrocardiographic changes, though from the facts given in many of the histories it would be quite adequate to explain the patients' symptoms.

Many cases follow on some unaccustomed activity at work or in the pursuit of a hobby, and the pain may very closely mimic angina of effort. It may arise in the pectoral or intercostal muscles and may radiate down the arm. It is never referred up into the neck or to the jaw, such a radiation being almost pathognomonic of true angina. It is often associated with tenderness and with increased pain on movement against resistance. Breathing usually makes the pain worse, and it is noteworthy that expiration is often more painful than inspiration. Sudden unexpected movements are very likely to increase the pain, and postural change may make it worse. The length of history in these cases is often enough, taken in conjunction with the patient's general condition, to exclude coronary disease, as in the following case in which the pain had been present for ten years.

Case 4

A man aged 58 was seen in 1949. He gave a history of injury to his chest and back when he fell between two joists ten years ago. The ribs were not broken, but he had some left-sided chest pain for many months after this. The pain returned

five years later and, because of its radiation down the left arm and its relation to exertion, was diagnosed as angina. Trinitrin was said to have given relief. He had been advised to retire from business and had since led a life of semi-invalidism.

On further questioning it was disclosed that the pain was made worse when the head was bent backwards, and that it was affected by movement of the left arm. It was worse on lying on the right side and eased by lying on the left. It was also made worse by breathing and by putting on his coat. Standing caused more pain than walking, and shaving was particularly troublesome. His easiest position was when he was bending forward. When questioned about the alleged response to trinitrin, it transpired that he felt some relief ten minutes to a quarter of an hour after taking a tablet, so that it is probable that some other factor was really responsible for the improvement.

There was no local tenderness, but pain could be induced by torsion of the trunk or on pressing with the left arm against resistance. A radiograph showed some osteoarthritic changes in the mid-dorsal spine but no disease in the chest. The cardiac outline was normal, and the electrocardiogram was normal in the limb and chest leads.

Physiotherapy failed to give him complete relief, and when reviewed six months later he still complained of some pain on standing, probably due to the arthritic spine; but he was back at work and much happier.

Case 5

A man aged 52 was seen in June, 1947. Two weeks previously he had been gardening and had clipped grass with shears in a confined space between two frames. On waking next morning he had pain at the lower end of the sternum. This pain lasted three days. It was a steady ache made worse by deep breathing and by postural change in bed, and though he was not shocked his doctor had diagnosed coronary infarction and had given him some morphine.

When seen on the third day there was no evidence of cardiovascular disease and the electrocardiogram in the three standard leads showed no abnormality. There was no local tenderness, but the pain could be elicited by adduction of the arms as in the movement of clipping with shears. A diagnosis of chest-wall pain was made, and he was advised to return to work. He was reviewed a week later, when he stated that he had been weeding in his garden for a quarter of an hour and while stooping the discomfort in the chest had reappeared. He described it as a slight ache or tightness which lasted some hours until he went to bed.

In February, 1948, a follow-up showed that he had had no further trouble in spite of gardening and golf, and the electrocardiogram was still normal. In April, 1949, in reply to a questionnaire he stated that he was entirely free from symptoms in spite of full activity.

It is difficult to locate the structures involved in the production of his pain, but it is probable that the site of origin was in the diaphragmatic attachments of the chest wall.

Fibrositis

Fibrositis is a term used loosely for a condition characterized by integumental pain without any known gross pathology. It is probable that we are now getting nearer to an understanding of its pathology on the lines suggested by Copeman (1949). The majority of cases seen in this group have a tendency to obesity. The pain is of a constant aching type, with exacerbation on movement, particularly where the movement is sudden and unexpected. Even though it is widespread it is associated with focal tenderness, and sometimes actual nodules may be felt, particularly over subjacent bone. The tenderness is best elicited with the finger-tips, and it is often so localized that it may be missed if the fingers are not carefully run along the intercostal spaces as well as over the ribs and sternum. Pressure on the tender spots is so painful as to make the

patient wince. This is quite different from the generalized tenderness of the chest wall found in cases of coronary infarction, in which a wide area of tenderness is commonly associated with some skin hyperaesthesia.

Kellgren (1947) points out that pressure on the deep tender spot causes a steep increase in pain together with referred tenderness, and states that the deep tender spot often lies altogether outside the distribution of the pain. It is therefore very likely to be missed if search is confined to the area which the patient describes as painful. The pain may mimic angina of effort very closely when the intercostal muscles in the region of the sternum are affected, particularly as the pain may radiate down the arms.

Infiltration of the tender area with a local analgesic is usually sufficient to cure a local fibrositis, but heat with massage is required for the more widespread form.

Five cases in the group under review were in obese women who complained of breathlessness on exertion and pain in the region of the sternum. The tenderness was limited to an area about 3 in. (7.6 cm.) in diameter where the breasts join the sternum, and appeared to be localized to the fatty tissues; these cases would therefore be more accurately described as panniculitis. It is these cases which tend to acquire the label of fatty heart because of the breathlessness and pain, and the diagnosis is still further clouded if thyroid medication has been instituted for the obesity, as this often adds palpitation to the patient's list of symptoms. Treatment with diet for the obesity is the keynote of treatment, though physiotherapy may be of some value.

Case 6

A woman aged 40 complained in 1948 of tightening across the chest over a period of three or four years. The pain felt like a tight squeezing in the upper part of the chest with some radiation down the left arm. The first attack had come on when walking and carrying some groceries. It had been diagnosed as angina, and her activities had been curtailed.

On questioning it had been found that though she had periodic attacks of pain there was a constant residuum of discomfort in the third intercostal space, and the attacks of pain that had come on were of a stabbing nature and left increased soreness behind them. It was impossible to take a deep breath during the attack. Posture had no effect. She was able to walk without pain, but some of her domestic duties were apt to bring it on, particularly scrubbing or using the vacuum cleaner. There was extreme local tenderness in the third intercostal space on the left and no evidence of cardiovascular disease. She was treated with physiotherapy and a reducing diet, and one year later in reply to a questionnaire she stated that she was better but that she had occasional recourse to heat and massage, a procedure which was always effective if her pain threatened to return.

Case 7

A woman aged 63 stated in 1948 that five months previously, when walking quickly, she had had sudden pain at the lower end of the sternum. It was quite localized, and was worse on breathing. She had rested on a wall for ten minutes and then went home to bed. The pain had persisted for a few days and a diagnosis of coronary thrombosis had been made. On questioning she stated that during her stay in bed the pain had been worse on taking a deep breath and on turning or raising herself from the pillow. Since then she had had a return of pain after activity such as cleaning out a cupboard. Ordinary exertion such as walking did not affect it. When seen she still had local tenderness in the region of the xiphisternum and on both sides of the lower end of the sternum. She was rather over weight. No disease was found in the cardiovascular system. She was reassured about her heart and given a reducing diet, and in reply to a questionnaire one year later she stated that she was quite well.

Kyphoscoliosis

The cases in this group are of particular interest, as they may very closely mimic cardiac pain and may be associated with breathlessness due to the deformity and fixation of the chest. It is a common thing for symptoms to appear for the first time in middle life, owing presumably to some further alteration in structure or to osteoarthritic changes in the spine.

The pain may be due to root pressure, to local pressure between the ribs, or to intercostal cramp. Posture plays a large part in its production, and it may come on in bed when sleeping in some unsatisfactory position. It commonly occurs, however, on standing, or on sitting for a long time in one position, as in driving a car. Many cases with a moderate degree of occupational stoop come into this category, and their pain is usually due to muscle fatigue, with resulting ligamentous strain.

These cases differ from other types of chest-wall pain in that there is no extreme local tenderness, and it may be impossible to reproduce the pain on movement during the examination because fatigue or relaxation of muscle tone plays a part in its causation.

One must be on guard, however, in these cases, as pain may be too readily ascribed to deformity when in fact there is some other cause. This was so in two cases, formerly included in this group, which during a follow-up were found to have had posterior gastric ulcers, one of them malignant. One patient had died and the other had lost his pain after operation. In both cases the pain had been of typical anginal distribution but had also been closely related to posture, and both had a severe kyphoscoliosis, which had seemed adequate to explain the symptoms.

Patients in this group may give a history of increasing pain going back many years with clear intervals of months. The chief reliance in diagnosis should be placed on the relation of pain to posture and to the nature of the pain itself. This is of two types—a dull aching pain and occasional sudden stabs followed by an increasing ache suggesting periodic pressure on nerves.

Case 8

A mining engineer aged 51 was seen in 1947. He gave a history of pain in the left side of the chest of one month's duration. It came on originally during a long motor journey and caused him to go to bed. It was of a dull aching type with exacerbations like a knife-thrust, particularly on taking a deep breath. The pain was felt over most of the praecordium and to some extent in the left shoulder-blade and on twisting movements of the trunk. It was associated with breathlessness on exertion. It was relieved by rest in bed, and he was referred as a case of coronary disease.

On examination there was no evidence of disease in the cardiovascular system. There was pronounced kyphoscoliosis with crowding of the ribs on the left side, but no local tenderness in the chest wall. The pain could be increased by firm lateral pressure on the left side of the chest. A radiograph showed severe osteoarthritic changes in the spine, and a diagnosis of spondylitis with referred pain was made. He was treated for some weeks on fracture boards and then referred for remedial exercises. When reviewed in February, 1948, he was looking and feeling much better and had changed his work to employment requiring no stooping. There was still some pain on deep breathing and occasional aching precordial pain when tired, relieved by pressing firmly back into a hard chair.

Discussion and Conclusion

A detailed history is of paramount importance in all these cases, and due regard should be paid to a family history of coronary thrombosis. The exact conditions in

which the pain occurs should be checked and counter-checked, and the time relations of the pain should be ascertained. The factors which bring relief must also be considered, and, in particular, relief from change of posture is important.

Relief from trinitrin should not be accepted without inquiry about the interval before relief is obtained, as this is sometimes half an hour or more, or in any case too long for a drug to be really responsible for the improvement.

Pain induced by excitement and anger is most unlikely to be of chest-wall origin, and this is therefore a most important differential point.

Pain arising in the chest wall has certain characteristic features, the most important of which is local tenderness, particularly in fibrositic cases. In these cases the tenderness is well localized, sometimes to small discrete areas which can best be felt with the finger-tips. These tender spots may be on the surface of the ribs or sternum or may be in the intercostal spaces, and the pain elicited is acute when the exact spot is found.

The second characteristic of pain arising in the chest wall is related to movement. This is especially pronounced in those pains associated with muscle strain and fibrositis. The pain is made worse on initiating movement before any real exertion is involved, and it may be made worse by turning in bed or on reaching towards a bedside table.

The question whether a patient's pain is made worse on turning in bed is probably the most important question to ask in these cases of chest-wall pain simulating coronary disease. A patient with acute coronary infarction may find that turning in bed is painful, but it is more the change of posture than the actual initiating movement of turning which causes the increased pain; many patients state that their pain comes on in bed when they really mean that it comes on when turning in bed.

Chest-wall pain, though usually related to specific movement, may be increased on general exertion if this involves increased respiratory effort. True anginal pain is usually felt long before there is sufficient dyspnoea to increase respiratory movements.

Pain associated with kyphoscoliosis is often made worse by certain postures in bed, and patients with such a condition may wake up with pain suggesting a coronary lesion as the result of lying in some awkward position. Clinical examination, however, is usually sufficient to differentiate these cases.

The third most important point about chest-wall pain is the possibility of reproducing the pain by particular movements of the trunk against resistance. This is true particularly of pain arising as the result of muscle strain, but a detailed history will usually relate the onset of pain to some specific exertion in the past.

In all cases emphasis must be laid on the detailed history, and in many the length of the history taken in conjunction with the clinical findings is enough to exclude coronary disease.

In spite of a detailed history and careful clinical examination there remain some cases of pain referred to the chest wall in which the diagnosis will remain in doubt, and only full electrocardiographic examination before and after exertion will serve to make the position clear.

The difficulty arises from two factors—local tissue changes in an area of referred pain and the phenomenon of summation of pain. Davis and Pollock (1932) have suggested that trophic disturbance in the area of referred pain may produce local tissue changes, with resulting areas

of tenderness, and Steinbrocker *et al.* (1948) have described the painful stiff-shoulder syndrome in coronary disease. There is no doubt that fibrositic lesions of the chest wall and painful shoulder-joints are very common accompaniments of coronary disease, and it would be interesting to know how many of these patients are to be found attending orthopaedic clinics, their cardiac condition undiagnosed.

The summation of pain from the heart with pain arising from some local condition in the chest wall introduces a further difficulty in diagnosis, and Cohen (1947) has postulated this explanation of some cardiac pain being referred to particular areas of the same segmental supply. An old gunshot wound or some area of fibrosis may become the focal point for the expression of an underlying cardiac pathology; it is therefore important to realize the danger of being too readily satisfied with a local chest-wall explanation of a patient's symptoms in any case where he is a potential sufferer from heart disease.

The reverse mistake of diagnosing heart disease when the trouble lies entirely in the chest wall is, however, more frequent and disastrous. It might well be said that it is better that one patient should suffer a sudden cardiac death within a few hours of a reassuring prognosis than that one should go through life with a mistaken label of cardiac disease.

In elderly patients who have a doubtful chest pain and some arteriopathy no serious harm is done by reasonable limitation of activity on general grounds, but cases in the younger age group demand a more accurate diagnosis, and if after complete investigation there remains some doubt it is much better that the heart should be passed as normal.

Summary

The differential diagnosis of chest-wall pain is discussed and stress is laid on the need for due consideration to be paid to local structural causes as well as to the possibility of an underlying cardiac pathology. Some illustrative cases are briefly described.

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In the U.S.A. the Communicable Disease Centre is the division of the Public Health Service responsible for providing assistance to State health departments in meeting public health emergencies. According to Surgeon-General L. A. Scheele the most extensive investigation carried out by the Centre in 1949 was in Muskegon, Michigan, in August and September. An epidemiological team composed of 12 persons conducted there one of the most thorough studies ever made of a community in connexion with a poliomyelitis epidemic. Some phases of this research are to continue for several years. "An interesting feature of the poliomyelitis investigation in Arkansas in August," Dr. Scheele said, "was the use of a 'circuit rider,' not unlike the preacher on horseback who toured rural areas of the South around the turn of the century. The 1949 circuit rider in this case was a public health representative. He made rounds three times a week, collecting from field workers certain important specimens for laboratory study." A physician-veterinarian team was sent from the Communicable Disease Centre to investigate a baffling epidemic which afflicted human beings and horses at the same time last August and September in Colorado. They found that two virus diseases existed simultaneously, creating a complicated diagnostic problem. Poliomyelitis was prevalent among human beings at a time when encephalitis was occurring in both man and horses.

CANICOLA FEVER IN BRISTOL

CLINICAL, BACTERIOLOGICAL, AND EPIDEMIOLOGICAL NOTES ON SIX HUMAN CASES

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Canicola fever is an acute infective disease caused by *Leptospira canicola*. This organism belongs to the same genus as *L. icterohaemorrhagiae*, which produces Weil's disease (leptospirosis or infective jaundice), and is normally a parasite of dogs. It occasionally infects man, giving rise to an illness which may simulate influenza or meningitis, and which may be accompanied or followed by eye symptoms. The prevalence of the condition in the Low Countries has been demonstrated by a number of workers since the species was first isolated there by Klarenbeek and Schüffner (1933), but its existence in this country has been appreciated only within recent years. We here record six cases to add to the six previously reported in Britain by Baber and Stuart (1946), Laurent *et al.* (1948), and Weetch, Colquhoun, and Broom (1949).

In two of our cases canicola fever was not diagnosed during the febrile attacks, and was suspected only when ocular complications supervened. The patients, a husband and wife, were referred to the Bristol Eye Hospital on August 10, 1949, both complaining of dimness of vision in one eye following an influenzal illness. They were found to be suffering from iridocyclitis, and were sent on August 22 to the medical clinic of the hospital for complete examination by one of us (A.M.G.C.). When the history of the febrile attacks was reviewed it was noted that both patients had been nursing a sick dog before the onset of the illness. The husband, an engineer, constantly had abrasions on his hands, and the wife had a cut finger at that time.

The possibility that the pyrexial attacks had been canicola fever was considered, and specimens of serum were tested for the presence of antileptospirosis antibodies. The sera of both patients agglutinated *L. canicola* to high titre, so confirming the retrospective diagnosis of the cause of the febrile illnesses and the iridocyclitis.

These cases occurred in a so-called "squatters' camp." The term is likely to convey a completely wrong impression of the general hygiene and living conditions—which in fact are very good—so it will not be used. The community, inhabiting former American Army huts, was relatively circumscribed, and through the co-operation of its members we were able to investigate the agglutination reactions and to make ophthalmoscopic examinations of most of the adults. Serum from all the dogs on the site was also tested. The results obtained, and certain conclusions we think may be drawn from them, seem of sufficient interest to be given in some detail.