only 3% of 332 persons with negative tests, while 'false positives' (a positive test in a person free from coronary heart disease) were recorded in no more than 3·8% of 158 positive reactors (Master & Rosenfeld 1964). However, doubt is thrown on its value by a much higher incidence of false negatives (12%) and false positives (39%) in a double-blind trial (Friedberg *et al.* 1962). The second use of the exercise test as a predictor of symptomatic coronary heart disease does not concern us here apart from this comment. When a positive test is obtained in a symptom-free patient with a normal resting ECG, the message is often passed on to the patient or his relations that he is 'due for a coronary some day', as it is often put. This statement is both true and misleading at the same time. True in that the incidence of myocardial infarction in positive reactors (15%) is higher than in negative reactors (1·5%) over a ten-year observation period (Franco *et al.* 1961); false in that the individual chances of escaping the disease are much greater than the chances of developing it.

Coronary arteriography is a very good method of demonstrating that angina pectoris and myocardial infarction are associated with coronary arteriosclerosis (Proudfit *et al.* 1966). As a method of identifying chest pain when the diagnosis is in doubt it is particularly inefficient according to the writings of experts in the technique (Hale *et al.* 1966). If an equivocal pain could be confidently labelled non-coronary or coronary according to the normality or abnormality of the coronary arteriogram, it would be irreplaceable as a diagnostic tool. Unfortunately this is not so, for the arteriogram may show little or no coronary disease with classical, even fatal, angina.

The chief use of coronary arteriography is as a dynamic method of following up the development of coronary arteriosclerosis, and the effect of diet, drugs and other treatment on coronary abnormality already present. It will also be a mandatory part of the pre-operative investigation if a successful surgical treatment is ever invented. Its main abuse is when it is regarded as the deciding factor as to whether a chest pain is or is not myocardial ischæmic. If the technique is sensitive enough it should delineate the macroscopic coronary arteriosclerotic lesions present in 78% of normal men aged 21–32 (Enos *et al.* 1955). So, given the fact that the majority of men have coronary arteriosclerotic lesions, how can their arteriographic demonstration have a diagnostic bearing on the identity of a chest pain? Or for that matter, how can a normal coronary arteriogram exclude myocardial ischæmic pain since small artery occlusive disease beyond the scope of current arteriography can almost certainly cause anginal pain?

It would seem that coronary arteriography is most likely to give an abnormal pattern when the clinical diagnosis of a chest pain is clearly ischæmia, and a normal pattern with non-cardiac pains. Its success rate (in identifying the correct cause of a chest pain) *vis-à-vis* clinical interrogation along well-trained lines would be difficult to establish. Allowing for the anxiety to the patient, time consumed by physicians and radiologists and the morbidity of the test, one feels that its application for the routine identification of chest pain represents a retrograde use of a test potentially valuable in other applications.

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Arthritic Causes of Chest Pain

The source of chest pain arising from lesions of the thoracic joints is usually readily established, leaving as the major problem the identification of the pathology. However, there are in addition a number of syndromes associated with arthritis which cause chest pain, and, as is well known, pain from lesions of the cervical spine may be referred to the chest. Rather than catalogue all the types of arthritis which can thus cause chest pain, which is hardly possible in this brief communication, I propose to refer only to those arthritides that particularly involve the thoracic and cervical regions or exhibit noteworthy features when they do.

Pain arising in the acromioclavicular joint radiates, if at all, into the upper arm and not into the chest, but it is instructive to contrast its degenerative lesions with those of the functionally more adequate sternoclavicular joint. Both clavicular joints are synovial and may incorporate an articular disc which when present in the former joint is flimsy. The acromioclavicular joint acts as a plane joint but the latter, in spite of its plane surface, acts as a ball and socket joint, and

exhibits a greater range of movement. Both joints are liable to trauma and, while forward subluxation of the medial end of the clavicle supposedly in response to repeated minor trauma is well recognized, occupational wear and tear particularly affects the acromioclavicular joint. As a result of such factors the acromioclavicular joint contrasts with the sternoclavicular in showing earlier, more rapidly developing and more marked regressive changes of the articular surfaces (DePalma 1957). Clinically, also, osteoarthritis is more common in the acromioclavicular joint but it occurs in both, not least in primary generalized osteoarthrosis (Arlet & Ficat 1958).

In contrast to osteoarthritis, the incidence of inflammatory arthritis in the sternoclavicular joint is higher than in the acromioclavicular but the recognition of the type of arthritis may be difficult, particularly in relation to venereal arthritis. Tradition has it that sternoclavicular involvement is an outstanding feature of gonococcal arthritis. In fact, reference to three large series pre-dating the specific treatment of gonococcal arthritis – Wehrbein (1929), Lees (1932) and the classic paper by Keefer & Spink (1937) – indicates sternoclavicular joint involvement in only 1·6% of 1,138 cases, assuming unilateral joint involvement. Lees (1932) also quotes two further series comprising 1,168 cases with only 0·43% sternoclavicular involvement. Further, some of these cases were clearly examples of Reiter's syndrome as evidenced by references to keratodermia blennorrhagica and other manifestations of that syndrome. The clearer delineation of Reiter's syndrome, and the development of effective treatment for gonorrhœa, has led to a classification of *definite* gonococcal arthritis, that is, cases in which *Neisseria gonorrhœæ* are obtained from the synovium or synovial fluid, and *probable* gonococcal arthritis, that is, cases in which neisseriæ are found in the genito-urinary tract or blood, and the arthritis which occurs shortly after infection clears promptly with antibiotic treatment. In a study of 21 cases fulfilling these criteria, Partain *et al.* (1968) make no reference to sternoclavicular joint involvement as evidenced by pain on movement, tenderness with soft tissue swelling or joint effusion. In contrast, Wright (1963), in a study of 101 cases and using essentially similar criteria, reported the sternoclavicular joint involved in 17% of the definite group and 4% of the probable group. However, this study was retrospective with, as the author states, 'the usual attendant limitations'. Clearly further investigation is required to establish the true incidence of sternoclavicular involvement in gonococcal arthritis, itself rare, but present evidence hardly justifies the emphasis given to it in some textbooks.

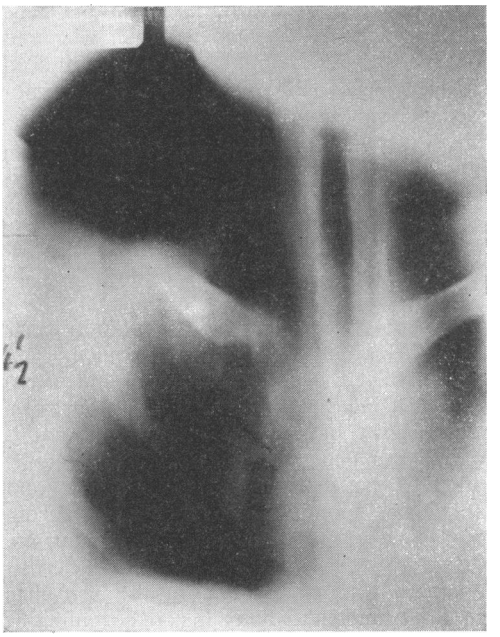

Fig 1 *Syphilitic lysis of the clavicle*

Wright (1963) also records sternoclavicular involvement in 14% of 51 cases of Reiter's syndrome, a figure higher than that reported by Paronen (1948), i.e. 4·4% of 344 cases (325 with arthritis) of the post-dysenteric form of the syndrome. Paronen also quotes another series of 136 cases with an incidence of 4·4%.

Inflammatory joint disease of the sternoclavicular joint in early acquired syphilis also seems to have been over-emphasized in the past although gummatous arthritis and osteitis in relation to the clavicle are well documented. A case of outstanding interest was presented to the Section of Dermatology by Sarkany (1965). This patient, a woman aged 24, attended the Royal Free Hospital with secondary syphilis complicated by effusions into the knees, tender swelling of the right sternoclavicular joint, and tenderness of both acromioclavicular joints. X-rays showed apparent lysis of the ends of the clavicle (Fig 1).

Sternoclavicular joint involvement in rheumatoid type arthritis is of interest because of the occurrence of a monarticular form of the disease. Monarticular subacute non-infective arthritis of the sternoclavicular joint was first reported by Bremner (1959) in 12 middle-aged women. It was characterized by the gradual onset of joint swelling accompanied by slight aching, usually on the right side but without any history of trauma. Investigations included normal ESRs and Rose's tests, but in 60% of cases erosions of the articular surface of the clavicle occurred. Synovial his-

tology showed lymphocytic aggregations. The condition generally resolved within a year and involvement of other joints did not occur. Indeed clinical evidence of involvement of the sternoclavicular joint in classical rheumatoid polyarthritis is reported to be as low as 0·5–2%, although histological studies suggest a higher incidence of involvement (*see* Sokoloff & Gleason 1954). Sternoclavicular joint involvement in ankylosing spondylitis also occurs sometimes with the development of erosions.

A coracoclavicular synovial articulation exists in only 1·2% of otherwise normal shoulders, but may be involved in both rheumatoid and osteoarthritis (De Haas *et al.* 1965). The existence of a synovial cavity in the manubriosternal joint is less rare and occurs in some 30% of people. Savill (1951) has defined the changes in this joint in ankylosing spondylitis as a progression from loss of bone outline involving the manubrium, the sternum or both, to erosion and sclerosis comparable with that found in the sacroiliac joints and, finally, to bony fusion beginning at the periphery. He found radiological evidence of these changes in 72% of 61 spondylitics. Interestingly, he found no erosions in the manubriosternal joint in 22 cases of rheumatoid arthritis, but they certainly occur (Fig 2).

Of the syndromes associated with arthritis which can cause chest pain, polymyalgia rheumatica has excited considerable recent interest. The characteristic picture of pain and stiffness of the shoulder and hip girdle muscles and sometimes of the spine, more severe in the morning, generally occurring in elderly women who on examination show loss of shoulder movements, a high ESR but little else, is now generally recognized. Although most recent reports, particularly those from Scandinavia, have been concerned with the demonstration of giant cell arteritis in such patients, in a proportion of them synovitis can be demonstrated in the central joints, particularly those of the shoulder girdle. For instance, Bruk (1967), in a series of 80 patients, reported tenderness of the sternoclavicular joint in 69% and of the acromioclavicular joint in 72%. Biopsies of the sternoclavicular joints of 5 patients demonstrated a chronic non-specific synovitis in 4. Radiological evidence of erosions of sternoclavicular joints was also found. Neuralgic amyotrophy, clinically characterized by pain around the shoulder and upper chest and followed within hours or days by muscle paresis in the shoulder region, has been described in Reiter's syndrome (Catterall *et al.* 1965). Joint disease in hyperparathyroidism (Bywaters *et al.* 1963) has similarly been reported only rarely, but is of particular importance as a cause of chest pain. This is because, in common with other metabolic bone diseases, it may present as ill-localized back, chest and thigh pain of a 'rheumatic' type and, second, it is a cause of erosion of joints including the acromioclavicular and sternoclavicular.

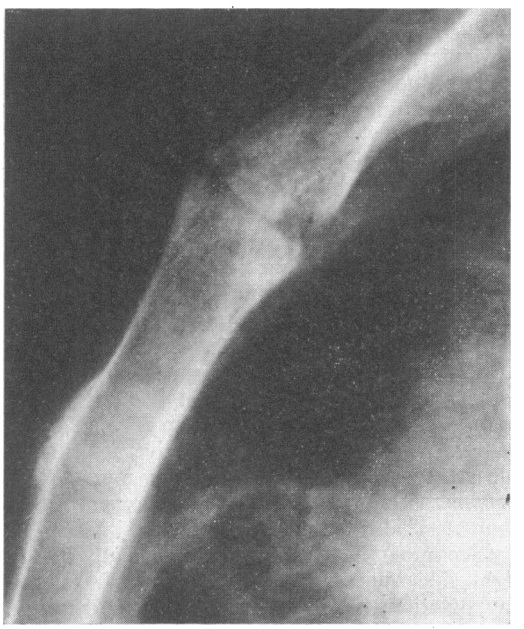

Fig 2 *Manubriosternal erosions in rheumatoid arthritis*

Spinal lesions are, of course, a major cause of chest pain either because of referred pain, most commonly from spinal degenerative changes in the cervical or dorsal regions, or from involvement of the dorsal nerve roots in spinal disease such as tuberculosis. A similar destructive lesion can occur in ankylosing spondylitis (Hicklin 1968). Another interesting spinal lesion associated with arthritis is paravertebral calcification in psoriasis which Bywaters & Dixon (1965) described in the thoracic and lumbar regions. Fig 3 illustrates such calcification in the cervical spine in a 62-year-old male psoriatic who had no clinical evidence of a peripheral arthropathy.

Arthritis of the costovertebral joints in spondylitis with its accompanying loss of chest movements is readily diagnosed. Not so well recognized is the existence of degenerative arthritis of the costovertebral joints. Nathan *et al.* (1964) found costovertebral arthritis in 48% of 346 skeletons and 17 out of 100 random X-rays of the dorsal spine. It was found earliest in the third decade, rapidly increasing to maximal incidence

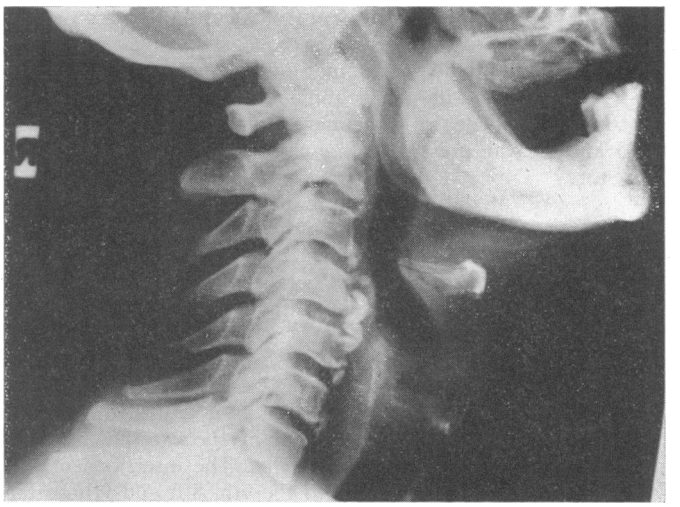

Fig 3 *Psoriatic paravertebral calcification*

during the fourth decade. It had a characteristic distribution in that the 1st, 11th and 12th costovertebral joints, which are formed by full facets, showed a higher incidence than the 2nd to 10th joints, which are formed by hemi-facets with the intervertebral disc between them. The incidence also seems related to the length of the ribs and the inferior costovertebral facets were more often involved than the superior.

One syndrome that I would particularly like to discuss in relation to the spine is Tietze's syndrome. This syndrome is described as consisting of a benign, tender, bulbous or fusiform swelling in relation to the costal cartilages, generally the 2nd and 3rd. There is a tendency towards remission but this is rarely complete. Following Tietze's first description of 4 cases in 1921, something in excess of 250 have been reported in the literature under various names, e.g. costal chondritis, costochondral syndrome, thoracochondralgia and chondropathia tuberosa. It has been blamed on strains of the sternocostal joints or the costochondral junctions, for example from coughing, and on premature calcification of the costal cartilage. It is clearly not a chondroma, there being no evidence of steady growth, lobulation or distribution outside the upper costal cartilages. No abnormal histology has been demonstrated and indeed the macroscopic appearances have suggested forward angulation of the costal cartilage rather than increase in mass (Beck & Berkheiser 1954). I believe a high proportion of these cases are related to spinal deformity with secondary prominence of one or more costal cartilages. For example, a woman aged 45 who complained of pain in the upper chest was clearly worried about swellings in the region of two of the costochondral junctions which she had made tender by repeated palpation fearing as she did the existence of a cancer. Examination of her spine showed a scoliosis and of her chest prominence but no enlargement of two adjoining costal cartilages. Tietze's syndrome could in theory be imitated by arthritis of the sternocostal synovial joints but this must be rare indeed. However, aching at the anterior chest wall in rheumatoid arthritis occurs and may well be due to involvement of the sternocostal and interchondral joints. Episodes of anterior chest pain due to gout have also been described (Frank *et al.* 1960), as has tophaceous gout involving the sternoclavicular joints (Sokoloff & Gleason 1954).

The painful xiphoid syndrome or xiphoidalgia has been described as a variant of Tietze's syndrome. It is characterized by pain associated with tenderness of the xiphoid process (Wehrmacher 1958). I have little doubt that this syndrome is usually traumatic and recall that the xiphisternum is peculiarly tender as can be readily seen when an enthusiastic student palpates it or when it is displaced with abdominal swelling. Another possible articular syndrome is the so-called precordial catch, that is a sharp pain felt near the cardiac apex which is short lived and may be brought on by slouching in a chair, &c. Miller & Texidor (1959) reported this in 28 patients and suggested it was due to a lesion of the parietal pleura. However, the very suddenness of its onset suggests a mechanical cause and it may well arise in one of the rib joints.

One may conclude that the synovial joints of the thorax can be the site of one or more of the various arthritides known to involve this type of joint, but some regional peculiarities exist. The cartilaginous joints of the thorax seem particu-

larly liable to acute or chronic trauma and they are probably the source of a number of painful anterior chest-wall syndromes, the exact cause of which needs further investigation.

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Mechanical and Postural Causes of Chest Pain

Chest pain arising in skeletal structures may be local or referred. Local pain is either due to direct trauma or to self-induced soft tissue injuries.

Local Trauma

A blow on the chest wall causes a hæmatoma or contusion of the periosteum and the diagnosis presents no problem if the patient is seen soon after the injury. Pain due to periosteal contusion, however, persists for several weeks, or even months in some cases, and may be aggravated by occupational factors. Difficulty in diagnosis then arises when the patient is seen later and does not remember the circumstances attending the onset of symptoms.

In other cases there may be no relevant history and the only evidence to suggest a traumatic origin is the presence of local tenderness over a rib or intercostal space. Careful investigation of the patient's occupation usually reveals the cause as self-induced trauma: for example, leaning over a bench or machinery or the application of chest wall pressure in the use of a hand tool.

Establishing the exact cause of the injury in such cases is made more difficult by the fact that there may be no acute pain at the time and the patient does not therefore connect his activity with the subsequent discomfort.

Pain in the lower chest due to strain is normally caused by some physical effort involving torsion of the trunk. This may cause a 'tennis elbow' type of injury of the origin of the external oblique from the lower eight ribs. The insertions of the internal oblique into the 7th, 8th and 9th costal cartilages and of the rectus abdominis into the 5th, 6th and 7th costal cartilages are also possible sites of local trauma. Similar injuries occur as a result of pushing and pulling strains of the upper limbs involving the pectoral muscle origins from all the rib cartilages or the origins of serratus magnus from the borders of the first eight ribs and the intercostal aponeuroses.

Although such local lesions, whether caused by direct or indirect trauma, normally cause pain only on certain movements, which either stretch or compress the injured soft tissues, this is not invariably so. There is frequently increased œdema and swelling at rest leading to a dull ill-defined ache which causes the patient to seek medical advice. In elderly patients with chronic bronchitis it is possible for similar musculoskeletal pains to develop as a result of the strain imposed by chronic coughing. Accurate diagnosis depends upon a careful history, including in some cases detailed analysis of the individual's method of work.

Many patients are resistant to the suggestion that their symptoms could be due to work or 'do it yourself' activities and prefer their own diagnosis that they are suffering from rheumatism brought on by a draught in the office or factory. Further explanation may, however, be successful in obtaining the information leading to a correct solution of the problem.

In the absence of any evidence to the contrary, a significant response to infiltration of a local anæsthetic over the site of maximum tenderness is usually adequate confirmation of the source of the pain. A more difficult problem is presented when local trauma occurs in the ligaments of the dorsal spine. In such cases, the pain is almost