EXTRATHORACIC PAIN IN CARDIOVASCULAR DISEASE*

BY

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The pain which a crippled heart can inflict is one of the more frightening experiences of clinical medicine. To most of us its essential distribution and typical radiations have an accepted and well-recognized pattern. But occasionally cardiac pain is encountered whose qualities are different, or a pain from other causes in a disordered cardiovascular system occurs, for instance, in the head, in the limbs, or in the abdomen, and presents a less familiar problem of diagnosis.

The purpose of this paper is to present some conditions where extrathoracic pain is the dominant symptom of structural or functional cardiovascular disease and to explore its clinical significance. In reviewing a large subject the exclusion of chest pain will serve to emphasize other clearly defined syndromes of pain, some of which are perhaps not so widely recognized.

Pain is a prevalent symptom which is notoriously difficult to assess, being entirely subjective and dependent on the acuity of the individual's sensory perception and also on his ability to describe it in words. An attack of coronary thrombosis in a labourer may cause less constitutional disturbance than a chilblain in a typist. To judge the relative gravity of a pain it is therefore desirable, whenever possible, to witness its occurrence or at least, by interrogation, to record accurately the patient's personal statements; the description may be graphic and, as we shall see, sometimes distinctive. On the other hand, a painful episode becomes exaggerated in the telling of it, and this is proportional to the number of times the subject has been interrogated; due allowance must therefore be given to possible distortion of symptoms.

Headache

Headache more often than not is a symptom which recurs without signs. We can, however, distinguish several varieties which are peculiar to specific circulatory disorders, though it is not easy to find a single common cause. However, largely as the result of Pickering's (1933) studies on histamine headache, we recognize that pain perception arises in the tissues of the dura round the meningeal arteries, where the sensitive trigeminal nerve-endings are affected by stretching or traction. Pressure changes within the minute vessels of the brain are probably of importance in the pathogenesis of hypertensive headache.

The interpretation of headache calls for a very careful interrogation and clinical understanding of the patient; we must not forget that he or she may have two kinds of headache—the one perhaps of little consequence, while the other may be the symptom of a fatal illness.

Temporal arteritis is a malady of older age, and appears to be commoner in countrymen and countrywomen. Its special feature is a constant, dull, and throbbing pain in one or both temples, at times sharp, stabbing, and severe, aggravated by cold winds and by movements of the head. The headache tends to wax and wane, often enough preventing sleep, and persists for several months before gradually dying away. But it is also accompanied by a vague sense of malaise, weakness and fatigue, bodily aches, loss of weight, and low fever. Indeed, the patients seem ill out of all proportion to their physical signs, and may at the height of the disease become very ill indeed. The blood pressure is normal and there is no significant arteriosclerosis, but blindness may follow, due presumably to retinal ischaemia from involvement of the vessels behind the globe (Harrison, 1948). The temporal arteries are conspicuously thickened, nodular, and very tender. The overlying skin may be reddened and oedematous. Pulsation in them is diminished or absent. Histologically we find an inflammatory reaction in the adventitial coat, invading the media, with focal necrosis and granulation tissue in which giant cells are numerous. It seems probable that the arterial tree throughout the body is similarly affected, and there is a good deal of evidence to indicate that we are here considering a generalized disease, somewhat resembling polyarteritis nodosa, though with a better prognosis possibly because the kidneys are spared. Excision of a piece of the temporal artery for biopsy is followed by relief of the pain; procaine injections along the artery or of the stellate ganglion are said to have the same therapeutic effect (Roberts and Ashley, 1948).

The headache of *subarachnoid haemorrhage* is characteristic. I recall a patient who collapsed while pumping her bicycle. On regaining consciousness she recollected stooping over the punctured tyre, but insisted that someone had hit her across the back of the head with a steel bar, so sudden and arresting had been the onset of her pain. The rupture of the internal carotid aneurysm usually occurs on physical straining. Sudden agonizing headache is followed by clouding of consciousness, with evidence of meningism, a raised intracranial pressure, and blood in the cerebrospinal fluid. Sometimes localizing signs may appear, such as pulsating exophthalmos with knife-like retro-orbital pain or a third-nerve lesion with contralateral hemiplegia.

Aortic incompetence is occasionally attended by a troublesome pulsating headache.

A hairdresser, aged 45, had contracted a chancre at the age of 17 and was pronounced cured after three months' treatment. When using a hair-massage vibrator on a customer he was suddenly overcome by a noisy beating in his chest. At the age of 42 his blood Wassermann reaction had been found

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positive on routine testing as a blood donor, and antisyphilitic treatment was begun. In this attack he had no pain or breathlessness, but when examined a loud "twanging" diastolic murmur could be heard all over the chest, front and back, with a diastolic thrill present at the aortic area. Blood pressure was 220/40 and the pulse regular. Radioscopy showed slight general enlargement with increased pulsation, but a normal aorta. The blood W.R. was then negative. Six months later he developed a continuous occipital headache with violent throbbing in his temples. The collapsing nature of the pulse in his temporal, carotid, and subclavian arteries was so pronounced that it could best be described as "detonating." This prostrating headache was relieved by sleeping with the head propped up by pillows.

Here the symptoms were evidently related to the unusually high pulse pressure resulting from probable partial rupture of an aortic valve cusp.

Headache develops in prolonged *anoxia* and in *polycythaemia*, but in the cyanotic plethora of *superior caval obstruction* it is perhaps uncommon. Intense headache may be a symptom of *dissection of the aorta* involving the carotid artery.

Many patients with *hypertension* wake every morning with an occipital or frontal headache, which varies in degree from a dull ache to a savage pounding and then gradually wears off as the morning progresses, only to recur day after day for many weeks at a time. It seldom awakens the patient, unlike the pain of increased intracranial pressure, and is not affected by straining but only by posture. Possibly this particular headache is more frequent in the unstable case of early or mounting blood pressure, being a less common finding in long-established benign hypertension. On the other hand, it may disappear without any fall in pressure; moreover, some patients with high-grade hypertension (Group IV), papilloedema, and a raised C.S.F. pressure have no headache.

A number of remedies have been tried for this distressing symptom, but in my experience bed-rest with the head propped high on pillows, barbiturate sedation, and aspirin are as useful as any of the more active lines of treatment and have at least the advantage of simplicity. Venesection often relieves the patient for a few days, but lumbar puncture, intravenous sucrose, dehydration, thiocyanate therapy, and even thoraco-lumbar sympathectomy may all prove disappointing.

Fishberg (1948) points out that the relief of headaches, which lumbar sympathectomy undoubtedly does achieve, may be due to a resultant redistribution of cardiac output, with a reduction of the fraction going to the head and arms. As evidence for this post-operative redistribution of blood flow he cites the clinical finding of cooler hands and warmer feet and the phenomenon of postural hypotension. That the vascular territory is not in fact denervated is supported by the persistence after operation of retinal arterial narrowing in the absence of headache. One of the clinical circumstances where diminished blood flow to the head is usually attended by relief of hypertensive headache is with the appearance of congestive failure, when, though the systolic blood pressure falls, the diastolic remains high.

Angina

The conventional reference of cardiac pain to the throat, arms, or lower jaw is familiar. But problems of diagnosis arise when a referred pain occurs alone or with insignificant pain in the chest. I have known a patient consult his dentist because of severe aching pain in the teeth when walking in the cold air. This could be relieved by closing his mouth and resting for a few seconds, but was in fact a true anginal pain. Cardiac pain may arise predominantly in the arms.

A retired Army colonel of 65, three months after a coronary thrombosis from which he had made a good functional recovery, was having to get up several times a night with severe pain in both wrists, eased more by papaverine than by trinitrin. Aching pain in the left forearm and left-sided toothache had been a prominent accompaniment of the heavy sternal pain which woke him at 3 a.m. in his original attack. Serial cardiograms in this case were typical of anterior infarction of the left ventricle.

A departmental manager, aged 63, complained of bad neuralgic pains on exertion, eased at once by rest, which always started in his left arm below the elbow and radiated up the arm to the jaw, the suprasternal notch, and right chest. The pain was also brought on in bed by cold linen sheets. His cardiogram showed changes of posterior infarction, returning later to normal. This pain syndrome is an instance of so-called "reversed angina."

That the pain of angina may be felt in a phantom limb suggests that the pain reference is not a simple segmental sensory reflex but possibly depends on cortical connexions (Harman, 1948). A patient whose left arm was amputated 4 in. (10 cm.) below the elbow-joint developed angina pectoris and began to be troubled by pain on effort in the phantom hand of such severity that he was obliged to obtain a new artificial limb, thinking that the old bucket was chafing his stump.

In considering differential diagnosis it is wise to remember that in the older age groups where coronary disease is more prevalent many patients are also subject to spinal-root pains, fibrositis, and lumbago of varying degrees, due to spondylarthritis and other degenerative skeletal processes. Effort pain may then arise in the back and, resembling cardiac pain in its distribution, be referred to the front of the chest or abdomen, and sometimes to the arms. However, these pains typically come on, not on ordinary physical exertion, but with specific movements such as bending, turning, lifting weights, or straining. After prolonged sitting—e.g., in the cinema or at the wheel of their car—these individuals develop a restless discomfort, eased by standing up or stretching their back, not unlike the relief obtained in sufferers from hiatus hernia. Such symptoms can be reproduced by pressure over the dorsal vertebrae, by hypertonic saline injection of the interspinous ligament, or by forced flexion of the head, and alleviated by rehabilitation and orthopaedic correction of faulty posture (Davis, 1948).

Upper abdominal pain on exertion, eased by rest, but due to the hepatic congestion of early right heart failure, must also be distinguished symptomatically from angina pectoris. Patients may describe their distress as more of a feeling of epigastric fullness or discomfort on exertion than a pain; or they may state that the abdomen is "tender to the bedclothes." Occasionally an acute surgical abdominal incident, such as a perforated gastric ulcer, closely simulates coronary thrombosis, particularly during the initial stages. After the first twenty-four hours, however, the distinction more often than not becomes apparent.

Pain in the Hands

We have seen that both angina and fibrositis can cause pain in the arms, but now and then a cramp-like pain arises in the arms of manual workers with repeated muscular use of those limbs which is due to *obliterative arterial disease*, an ischaemic manifestation identical with claudication in the legs.

An upholsterer, aged 53, whose work involved the pulling and stretching of heavy materials to cover sofas, complained for four years of an aching, stiff pain in the forearms and backs of the hands, mainly right-sided. He habitually experienced this pain at his work, particularly in the cold and

towards the end of a heavy week, yet it always subsided at once on resting. He had no other symptoms, and physical examination disclosed moderate arteriosclerosis only.

The disagreeable, sometimes very severe, aching pain in the fingers of sufferers from *Raynaud's disease* when exposed to cold is due partly to nutritional changes and areas of recent necrosis in the fingers and partly to the sudden release of warm blood into chilled fingers which follows the release of arterial spasm (Lewis, 1936). Exquisite local tenderness is often associated, and can be so intolerable that it will reduce a man to a state of quivering sobs. Lewis disproved the earlier conception that arterial spasm was the cause of the pain in this condition, for, although digital arterial spasm accompanies every attack, pain is not complained of in the more benign cases of *Raynaud's disease*.

The *glomus tumour* is a more uncommon lesion but is clinically important because of the paroxysms of pain which it produces. It consists of a benign hypertrophy of the microscopic arteriovenous capillary anastomoses which exist in the skin, chiefly of the pulp and nail-bed of normal fingers (Masson, 1924), and in health probably subserves the function of controlling skin temperature. The *glomus tumour* appears as a small slate-blue elevation on the skin, though if subungual in position it may not even be visible, and its most discriminative feature is the agonizing pain which the patient feels on the slightest pressure, friction, or exposure to cold. Now and then localized erosion of the terminal phalanx can be demonstrated by radiographs. The rich supply of nerve fibres which terminates about the epithelioid cells that comprise the vascular spaces of the glomus body would seem to be unduly sensitive to changes in vascular tension and so account for the inordinate pain which simple manipulation produces. These tumours may be single or multiple, and vary in size from a pin's head to a cherry stone. Surgical excision is usually curative.

Abdominal Pain

Aneurysm of the abdominal aorta was stated by Osler (1905) to be seven times less common than thoracic aortic aneurysm. Improvements in antisiphilic therapy and an increasing average age of the population are modern factors which have probably diminished this ratio, so that the present picture has somewhat altered. Aneurysm of the abdominal aorta seldom arises before the age of 50, and is generally arteriosclerotic in origin. It is said to provide some of the most varied and dramatic pictures seen in clinical medicine (Jennings, 1941). The "dull, boring, steady pain with awful paroxysms," wrote Stokes (1854), "enables us to diagnosticate the disease with probable accuracy." The pain, he observed, could be relieved by lying on the face or turning on the side. The following personal case would appear to be an example of this condition.

A housewife, aged 72, complained of a throbbing, aching sacral pain of six months' duration, eased by walking about and made worse when she sat on a chair or lay down. She also stated that her feet had always felt cold. The only significant clinical finding was weak pulsation in the arteries of the left leg, but x-ray examination showed a calcified abdominal aorta of irregular calibre. Physiotherapy has not affected her symptoms.

In these instances the pain may be due in part to bony erosion of the vertebral column, yet it is interesting to note how, owing to their resilience, the intervertebral disks often resist. Palpation of the abdomen perhaps discloses a fixed, expansile, tender, pulsating tumour, maybe with a murmur heard or a thrill felt over it. The epigastric pulsation of an enlarged right ventricle must not be confused with an aortic

aneurysm—the systolic thrust of the latter comes appreciably after the ventricular pulse—or with the symptomless dynamic pulsation of a normal aorta which is so easily demonstrated in the patient with a scaphoid abdomen and exaggerated lumbar lordosis, particularly if there is nervous overaction of her heart. Although an aortic aneurysm may rupture several months after the pain first appears, the prognosis is very often better, depending on the location and size of the aneurysm. Rupture is not always fatal.

Dissecting Aneurysm

During the last 25 years at Westminster Hospital there were 26 deaths from this cause, giving an incidence of 1 in 152 necropsies (Pulvertaft, 1949, personal communication). Elsewhere the necropsy incidence has been 1 in 430 (Glendy *et al.*, 1937) and 1 in 454 (Warren and McQuown, 1948). Although clearly a rare and often fatal condition, the number of cases diagnosed during life is steadily increasing, and we now realize that survival for some years may be possible, especially when re-entry of the dissection takes place into the original aortic lumen.

The important clinical feature to recognize is the dramatic suddenness of the onset of the pain and its initial overwhelming severity. Tearing, stabbing, choking pain strikes the patient usually across the chest and arms, but may spread to the back and down the abdomen to the labia or testicles, later to the legs. It may be intermittent and disappear gradually, to recur in subsequent attacks at lower levels as the dissection extends. Unlike the pain of cardiac infarction it is singularly maximal at its onset; a sense of constriction is unusual, hypertension is invariably present, and the dissection commonly begins during an act of physical exertion or sudden emotional strain. Severe pain in the back or spine indicates splitting up of the walls of the descending aorta, with rupture of the intercostal and lumbar arteries (Shennan, 1934). The dissection may obliterate the coeliac axis to cause infarction of the bowel, or the renal arteries may be involved.

From a detailed study of 28 cases Shennan decided that the essential aetiological lesions in this condition were multiple faults in the aortic media, which were filled with homogeneous connective tissue. Contrary to the common belief, the development of aortic dissection by penetration of the blood into the wall of the vessel through the base of an atheromatous plaque is extremely rare. The fact that the intima was sometimes intact suggested that haemorrhage might originate from the vasa vasorum.

In the symptomatology of *mass thrombus of the left auricle*, which occurs in certain cases of mitral stenosis, Evans and Benson (1948) point out that attacks of anginal pain in the chest are usual.

A woman, aged 57, with mitral stenosis and fibrillation, whom I have at present under observation, was seized with sternal pain for 12 hours; next day she developed an equally severe pain in the back and right loin. Her blood pressure was normal. The whole sequence of symptoms, from which she eventually made a good recovery, was at the time striking enough to invite possible confusion with dissection of the aorta, yet the aetiology was probably mass thrombus causing acute coronary insufficiency, followed by a small right renal infarct.

Embolic or Thrombotic Occlusion of the Aorta

In a patient with long-standing rheumatic mitral stenosis and fibrillation, or with a past history of coronary thrombosis, the onset of an abrupt, sharp, intense, and agonizing pain in the legs, loins, abdomen, or back, followed by shock, loss of power, and gangrene, with absence of pulsation in the abdominal aorta, suggests embolism at the aortic bifurcation (Morest and Rubin, 1948). Multiple visceral

embolic incidents may have preceded or may accompany it. Thrombosis of the aorta, on the other hand, is characteristically of gradual onset and develops usually in an arteriosclerotic subject; it is often preceded by symptoms of claudication in the legs (Banowitch and Ira, 1928). These features are illustrated by the following case.

A stock foreman, aged 50, with 2½ years' history of bilateral saphenous thrombophlebitis, followed by a coronary thrombosis and increasing claudication in the right calf, was in hospital for treatment of gangrene of the right big toe. He had clinical signs of generalized arteriosclerosis with an impoverished arterial circulation in both feet. Electrocardiograms showed fibrillation and left bundle-branch block. One morning a month later a mild gripping umbilical pain developed, gradually increasing in intensity. By midday it was severe, and vomiting, bloody diarrhoea, and shock came on. By evening morphine eased the pain, but two days later a sudden haematemesis closed the scene. Necropsy revealed an old apical infarct of the left ventricle with a large mural thrombus. An extensive thrombus occluded the aorta from above the origin of the superior mesenteric to below the bifurcation of the iliac arteries. The whole of the small intestine was infarcted. Atheroma was present in the coronary arteries, aorta, and right popliteal.

From time to time, as in the following case, the pain of a *false aneurysm* presents difficult diagnostic features.

An ice-cream manufacturer, aged 26, with a history of previous rejection for military service because of a rheumatic heart, developed a septic rash and fever. Three weeks later, when admitted to hospital, the blood culture yielded haemolytic *Staphylococcus aureus*; penicillin therapy was started at once (500,000 units a day).

On examination there was a loud apical systolic murmur and a mitral diastolic murmur. Rhythm was regular. After three weeks the fever was still present, so the penicillin dosage was doubled. He now began to complain of a series of pains which baffled his physicians, though admittedly examination was always difficult because he was a grossly obese and excitable subject: first a severe suprapubic pain with vomiting; five days later acute cramping pain in the right leg, yet without any unusual physical signs. Three weeks later he had an intense pain in the right groin, with shock, and developed signs of psos irritation. Rectal examination was negative. A week later, with groin pain and thigh flexion continuing, thrombosis became evident in the right saphenous vein, and within a few days his leg was oedematous. Although he repeatedly complained of a great deal of pain throughout the next few weeks, his temperature fell to normal, and a non-tender cord-like thickening of the vein in the groin could then be made out. Three months after admission he became suddenly breathless with pain in the chest; anuria set in, and in forty-eight hours, with signs of a left lung infarct, he succumbed.

Necropsy showed that his attacks of pain had been due to a false aneurysm of the pelvic vessels, probably mycotic. A blood cyst the size of a large grapefruit lay against the right brim of the pelvis, containing laminated clot and compressing the right common iliac artery and vein. Venous thrombosis extended down the veins in the thigh and up the inferior cava to the right renal vein. Old infarcts were present in the right kidney. In the heart the left ventricle was considerably hypertrophied, and the aortic cusp of the mitral valve contained a perforation ¼ in. (1.9 cm.) in diameter surrounded by healed vegetations. The upper lobe branch of the left pulmonary artery was occluded by recent thrombotic embolus.

Pain in the Legs

The uncomfortable, continuous, aching pain that arises in the calf of the healthy leg when walking fast in tight puttees, or in the shin when bicycling hard or skating, is identical with the pain of *intermittent claudication*. It is produced by a relative ischaemia of the working muscle. There are, indeed, many pathological processes in which this disproportion between the blood requirement of the leg and its blood supply is enough to induce such a pain,

but the commonest is *obliterative arterial disease*. Claudication is an occasional symptom of *severe anaemia*, *aortic stenosis*, *arteriovenous fistula*, *coarctation*, after *aneurysm*, *thrombosis*, or *embolism* of the *femoral* or *popliteal artery*, in some cases of *mitral stenosis*, and, rarely, in a prolonged attack of *paroxysmal tachycardia*. The pain is described as cramp-like only because of its resemblance to the pain of muscle cramp, for tonic spasm of the ischaemic muscle does not in fact take place.

The reason why the effects of arterial obstruction by intravascular thrombus are more profound than simple ligation is because the collateral vessels are usually already diseased and clotting is often extensive, obliterating the mouths of many collaterals.

An office worker, aged 49, for five months had a localized pain in the right calf on walking, which had gradually become so severe that slow walking for 100 yards brought it on and made him stop to obtain relief. Skin temperatures of the feet were normal, but pulsation in the right popliteal, tibial, and dorsalis pedis arteries was reduced. Arteriograms showed an obstruction in the middle of the femoral artery with collateral channels. He was operated upon and 4.5 cm. of the right femoral artery excised; this contained organized thrombus on an atheromatous plaque. The vessel was reconstituted with a portion of saphenous vein from the left leg and joined by vitallium tube. A month later, although he still had no palpable pulse in the right foot, he was able to walk a mile without pain.

Many patients such as he provide a clear enough description of their symptoms to make recognition easy, but it may be remembered that various orthopaedic conditions give rise, on using the limb, to pain which is also eased by rest. It will be noted, for example, that the pain of arthritis is not relieved by simple halting, but that the patient has to sit down to be relieved of it. Exercise tolerance in claudication relates to the extent of the arterial disease, frequently an asymmetrical process; so it happens that pain in the worse limb may restrict walking before the sounder limb is fully tested. Similarly, an advanced degree of arterial insufficiency in both legs can be masked by a limitation to the capacity for exercise which angina or dyspnoea may impose; the converse is also true. In arteriosclerosis obliterans the reduced blood supply to the limb sooner or later causes coldness in the foot, and the toes are often cyanosed. The skin may undergo further trophic or inflammatory changes as a result of damage by cold, and become glossy, telangiectatic, and unduly sensitive to warmth and friction. In these circumstances pain is felt *at rest*.

A clerk, aged 71, for two years complained of pain in his calves which pulled him up after five minutes' walking. Resting eased it at once. For the previous nine months he had also had constant pain in the toes of the right foot—"They burned in bed at night"—so that he was obliged to hang his foot out in the cool air or stand up by his bed to obtain relief. The toes were cold and plum-coloured, and arterial pulsation was absent from both feet.

In cases of rest pain the simple correction of a bed whose mattress sags in the middle may prevent some postural ischaemia of the feet.

The third variety of pain in arteriosclerotic disease of the lower limbs is that of *gangrene*. When the vitality and healing power of the tissues is so reduced that infection and necrosis may proceed unchecked, gangrene starts readily from a superficial abrasion, a corn, or a damaged toenail. Pain may be extreme. Lewis's advice to care for the feet as though they were pieces of porcelain cannot be given early enough to sufferers from this complaint.

With *arterial embolism* the dramatic onset of a sudden severe pain in the leg, becoming more intense, aching, and

diffuse, usually precedes the other signs. Lewis (1936) has pointed out that this pain does not arise in the artery at the site of the obstruction but much lower in the limb in the muscles deprived suddenly thereby of their blood supply, and that its onset even when abrupt is delayed. It is relative in severity to the size of the occluded artery only because a greater territory of tissue is rendered ischaemic, and it is relieved instantly when the blood supply is restored by removal of the clot, or it is reduced in degree if the plug passes on. Thus embolism of the brain or spleen is painless, because those organs contain no muscle. There is no evidence that arterial spasm alone directly produces pain.

The clinical associations of embolic catastrophes are well illustrated by the following case.

A man aged 38, who eight years previously was found to have mitral stenosis, developed auricular fibrillation and at the same time a sudden (painless) right hemiplegia due to cerebral embolus. Two years later his digitalis was inadvertently stopped for a while and his heart began to race. Ten days after redigitalization he woke with a violent pain in the hemiplegic leg and the foot went cold. The agony of his pain lasted twenty-four hours and was relieved only by hanging the foot down. From 4 in. (10 cm.) below the knee his leg was found to be cyanosed, oedematous, and icy cold. The diagnosis of popliteal embolus was made and he was treated conservatively. Within ten months he could walk unaided for 200 yards without pain, but four months later he suffered another stroke and died.

Tragic examples of this nature are only too common where embolic incidents have accompanied the onset of fibrillation or have followed mistakes in treatment. Because the pulse rate is slow or the symptoms have abated, digitalis maintenance therapy is stopped; the insidious acceleration of the ventricular rate which ensues is fraught with dangerous consequences. Such accidents would happen less often if we always remembered to warn each patient with auricular fibrillation that digitalis was his life-line, to be depended upon perpetually for the rest of his days.

Conclusions

In those circulatory conditions where a prevailing symptom is extrathoracic pain (in the head, limbs, or body) the degenerative arterial disorders play an important part with their attendant thrombotic and embolic incidents. Hence the symptom of extrathoracic pain in cardiovascular disease for the most part is rare in childhood, unusual in youth, but commoner as age advances. The pain usually conforms to some distinctive clinical picture. Examples are considered in this paper with the object of promoting familiarity with their qualities.

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VIOFORM IN THE TREATMENT OF SKIN DISEASES

BY

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The quinoline derivative known as "vioform" (5-chloro-7-iodo-8-hydroxyquinoline) is a stable, almost odourless, greyish-yellow powder which is only slightly soluble in water. It contains 41% iodine and 12% chlorine, and its use in the treatment of amoebiasis is world-wide (Cameron, 1947). For several decades it has been used as an antiseptic dusting powder and as the active ingredient of Alexander's ointment on the Continent (Perutz *et al.*, 1933). Only within recent years has its value in dermatology been appreciated in America (Wise and Sulzberger, 1942; Sulzberger and Wolf, 1942; Pillsbury *et al.*, 1943), and the external therapeutic uses were recorded by the American Medical Association in 1944. In Great Britain its value in dermatology has not yet been estimated.

Early in 1948 I obtained a small quantity of vioform powder and, in a 3% strength in a water-miscible cream, found it to be most useful in the treatment of pyococcal skin infections. The importing chemists then kindly offered to manufacture two types of preparation for trial, and a clinical investigation was started in June, 1948.*

Preparations Used.—Guided by the experience of Sulzberger and Baer (1946), it was decided to use only the following two preparations: (1) vioform cream (V.C.), which consists of 3% vioform in a water-soluble base composed of sodium lauryl, stearyl alcohol, spermaceti, glycerin, yellow soft paraffin, and water; (2) vioform ointment (V.O.), consisting of 3% vioform in a simple petroleum-jelly base.

Selection of Cases.—The majority of the cases were treated as out-patients. The greater number and variety of such cases outweighed the uncertainty of proper application by the patient. Patients of any age or sex were treated, but in view of the limited supplies of vioform no cases of widespread dermatoses were included. With the knowledge that other hydroxyquinoline ointments were useful in the management of pyodermias, it is not surprising that many cases of this type were chosen.

Control of Results.—It was considered that the number of cases would be too small to compare this remedy against a standard topical treatment by the method of treating alternate cases suffering from the same skin disease. Likewise, the better method of comparing the results of two similarly affected and symmetrically situated lesions in the one person treated by two different methods was thought impracticable in dealing with out-patients. Where it is considered to be of interest, however, reference will be made to previous treatments which had failed.

Classification of Cases.—A total of 115 consecutive cases of various skin diseases were treated. These have been grouped thus:

Group A	..	Sycosis barbae	..	..	15 cases
" B	..	Folliculitis of other areas	..	..	17 "
" C	..	Impetigo contagiosa	..	..	14 "
" D	..	Acne vulgaris	..	..	10 "
" E	..	Impetiginized eczema	..	..	14 "
" F	..	Post-auricular dermatitis	..	..	10 "
" G	..	Miscellaneous group	..	..	35 "

Where more than one type of infection was present the case was listed under the more prominent condition.

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