MERCER'S

# Orthopaedic Surgery


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EDWARD ARNOLD

# Preface to Seventh Edition

It is over 40 years since the first edition of this book appeared and over 8 years since a major change occurred when I joined Sir Walter Mercer for the last edition. This was an attempt to produce a more transatlantic approach fashioned after the classic textbook of Jones and Lovett. With the death of Sir Walter and my return to U.K. I have been fortunate in that Professor Albert B. Ferguson Jr. of Pittsburgh has joined me in this contribution to world orthopaedics. I know that the readers will welcome him in this our first combined edition.

Authors and publishers are quite rightly being criticized because there are so many textbooks which are so expensive and with delay in production do not adequately incorporate recent advances. However, because we have carefully selected material of tested value we believe this new edition is still relevant for its content of essential knowledge both for the postgraduate and the undergraduate. For example, basic sciences of the musculo-skeletal system have been expanded, for in them lies the basis for all orthopaedic thinking, practice and contributing to future advances. Since the last edition there have been remarkable advances in joint replacement and reconstructive procedures based upon an increasing knowledge of biomechanics and of properties of materials; in the use of phosphonates in certain metabolic disorders of bone; the increased understanding of genetics, e.g. of hip and of foot malformations, of Dupuytren's contracture etc.; in the understanding of coagulation defects and their substitution, allowing greater freedom of surgical attack; and in the ultrastructure of articular cartilage as the basis for understanding the common arthritides. All of these are in our everyday vocabulary and practice but now must be brought into the general literature of orthopaedics.

It is important for the postgraduate student of orthopaedics to read original articles and monographs and to study the textbooks of multiple authorship with their almost encyclopaedic sectional accounts. But we believe there is still a place for a combined authorship, covering and overlapping their knowledge in all sections. This unity of thought and expression provides a uniform style of presentation and emphasis, thereby giving a better opportunity to learn.

We have purposely avoided making this a manual of surgical practice. The clinical presentation of various diseases and disorders of the musculo-skeletal system have been emphasized and the basic sciences of anatomy, physiology and pathology stressed. This 'core' material is vital not only to the undergraduate to bridge the gap between the pre-clinical and clinical subjects but also to the surgeon in training if he is to rise above the mere technicalities of orthopaedic practice.

The references have been carefully selected from British, American and European literature with a preference for more recent authors—not in order to be fashionable but rather to provide a list of worthwhile articles which often contain detailed references to their predecessors. Some references are stated with their titles to indicate their completeness of topic as well as some for their more historic interest to ensure that they are accredited for their contribution.

This has been a major task of rewriting, reorganizing material and adding much new illustrative material. We hope that the balance and contribution have been maintained without too great an increase in length.

R.B.D.

Oxford, 1973

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It gives us great pleasure to have the opportunity of acknowledging the enormous amount of help and encouragement given by our many friends and colleagues. John Cockin, George Bentley, Roger Smith, Alastair Mowat, Campbell Semple, Martin Frances, Charles Galasko and Roland Parry—to name but a few—all have helped unstintingly. Professor Louis Goldstein has revised his section on scoliosis and Dr Philip Nichols adds a new and very important section on rehabilitation and physical therapy.

We would also like to thank Mr R. Emmanuel and his staff of the Medical Photography Department of the Nuffield Orthopaedic Centre and our secretary Mrs J. Johnson who has been so prompt and efficient in her typing. It is also a pleasure to thank Mr David Fuller who has been the proof reader, and Miss Barbara Koster of Edward Arnold who has been so patient, helpful and understanding of errant authorship.

Throughout the text we have attempted to acknowledge the source of all the reproductions and data. For these valuable contributions we are most grateful but do ask to be forgiven for any inadvertent omissions.

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# 1

# Introduction

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The term 'orthopaedy', adapted from the two Greek words, ὀρθός, meaning straight, upright or free from deformity, and παιδιον, a child, was originally used by Nicholas Andry, whose work, *L'Orthopédie óu l'Art de prévenir et de corriger dans les enfants les déformatés du corps*, first appeared in 1741. The elder Sayre considered that the word Orthopaedics was derived from ὀρθος and παιδευω, meaning 'to educate', and as such emphasizes the preventive and advisory nature of the specialty.

Modern orthopaedics is concerned with the study of the form and function of the musculo-skeletal system; its attack is directed against those affections that deform the architecture or arrest the balanced mechanisms of man's body, injuries and diseases of bones, muscles, nerves and soft structures which result in loss of form or function.

Andry originally taught orthopaedics as a branch of preventive medicine rather than as an offshoot of surgery, and the various methods he described of preventing and correcting bodily deformities in children were, in his own words, within the reach of 'fathers, mothers, nurses, and others entrusted with the bringing up of children'. Andry's words need not be passed over lightly: prevention is always better than cure, and if the principles and practice of preventive orthopaedics were more liberally applied today, many of the severer degrees of flat foot, scoliosis, and similar deformities would disappear. But the timely institution of preventive measures demands the early recognition of loss of form or function.

The solution of the problems of an orthopaedic case depends on a clear understanding of the pathological nature of each lesion. Those who escape contact with the deformed do not appreciate the keen mental anguish which they suffer—a mental anguish that led Gloucester when bewailing his fate in his sad monologue in *Richard III*, to exclaim:

> Cheated of feature by dissembling nature,
> Deformed, unfinished, sent before my time
> Into this breathing world, scarce half made up,
> And that so lamely and unfashionable,
> That dogs bark at me as I halt by them.

## The scope of orthopaedic surgery

Orthopaedics as a specialized branch of surgery, though it has been growing progressively since the days of its great pioneers, has achieved its present prominence largely as a result of the casualties of the two wars and the accidents incidental to the present mechanical age, the management of acute infections of bones and joints, the result of poliomyelitis and other neurological disorders and the emergence of congenital and developmental disorders both of bone and of muscle.

Orthopaedic affections fall into one or other of six groups;

1. Congenital anomalies
2. Affections of joints
3. Affections of bones
4. Affections of muscles, tendons, and other soft tissues
5. Affections of the nervous system
6. Static deformities

While many of the lesions have a definite pathological basis, it is being realized more and more that a large number of orthopaedic disorders are the end result of postural or static anomalies. These are anomalies which are produced by either *postural* forces which result from

**Fig. 1.1** A standing radiograph of a young girl aged 6 years showing genu varum and over-growth of the medial condyles of both tibiae

habit, occupational attitudes or body carriage or *static* forces which are concerned with bodies at rest, or in equilibrium, or acting as weights which are not moving.

Peabody (1938) differentiated between a *postural* deformity which is dynamic in origin occurring in young children with the possibility of continuing growth aggravating it and a *static* deformity in which, because of weakened musculature, structural abnormalities are aggravated by the forces of gravity in all ages.

Posture is dependent upon the tone of the skeletal muscles which is under the control of the sympathetic and somatic nervous systems necessary to maintain position against the forces of gravity. Posture abnormalities arise from abnormal forces, particularly during growth. This is particularly seen in adolescent kyphosis or round shoulders, and is correctable before any bony and disc deformities develop. It is also seen in the genu valgum deformity which is accompanied usually by laxity of ligaments and quadriceps muscle insufficiency. Forces of gravity during growth will produce deformity of bone and this is commonly seen in the overgrowth of medial condyles of the tibiae (Fig. 1.1). Many cases of acquired flat foot deformity or pes planus are postural in nature.

Many of the following pages are occupied with this aspect of the orthopaedic problem; its intrinsic importance is great, and, further, it has an important lesson to teach—that, from the orthopaedic standpoint, the body must be viewed as a whole, even though the actual complaint is a local one.

## *THE CLINICAL EXAMINATION OF AN ORTHOPAEDIC CASE*

No part of orthopaedic training is more important than the acquisition of a systematized method of examination. It cannot be too strongly urged that a true knowledge of disease, which forms the basis of successful diagnosis and treatment, can be founded only on the careful and accurate study of individual cases. Scientific and orderly investigation is as essential in orthopaedic conditions as in any obscure internal malady.

### The history

At the first consultation it is necessary to elicit a complete and accurate history of the patient's complaint, the mode of its onset, and the order in which the symptoms were first observed.

(*a*) *The complaint*. The chief complaint may suggest to some extent the nature of the affection, while it always focuses attention on some definite part of the body.

(*b*) *Manner of onset*. The illness may begin suddenly, or it may be gradual and insidious in its development. Apart from trauma, the most likely cause of sudden derangement is acute infection. When the onset is insidious, it may be due to a low-grade inflammation, granuloma or tumour, a slow degenerative process, or a postural anomaly.

(*c*) *Typical symptoms*. The typical symptoms to be inquired for in any injury, disease, or deformity, congenital or acquired, of the musculo-skeletal system and its associated structures are: 1. Pain and its features; 2. Disturbed sensation, e.g. paraesthesia; 3. Deformity: its onset and progress; 4. Weakness or paralysis of muscle power; 5. Limitation of movement of a joint; 6. Instability of a joint; 7. Crepitus.

(*d*) *The question of preceding injury*. There is a distinct tendency to ascribe all orthopaedic symptoms and errors to some injury, often sustained at a date considerably remote. An attempt should always be made to ascertain the exact details of any alleged trauma, and to establish its exact relation to the actual lesion as this may have important medico-legal bearings. Such an inquiry should be directed towards discovering whether the symptoms arose at the time of the injury, existed previously, or only appeared subsequently. A good practical rule is to ascertain whether the patient was able to leave the scene of the accident unaided or whether he required immediate assistance.

PAIN AND ITS FEATURES

Pain is one of the more common features of many orthopaedic conditions, and therefore some understanding of its nature and its properties are important for their management.

Pain was defined by Sherrington as 'the psychical adjunct of an imperative protective reflex' and is the sensation one feels when injured. However any precise definition is very difficult because of the difficulty of describing where the afferent nocioceptive impulses produced by injurious agents arise before they pass up into the central nervous system to be given meaning by the emotional and psychological state of the individual based upon the past and present experience. Pain must be regarded as an '*experience*' rather than a sensory change in the strict neurological sense.

## The anatomy of pain

### (*a*) *The painful stimulus*

Pain was believed to have arisen by 'normal' stimuli exceeding the intensity threshold for sensory nerve endings. This is no longer accepted in the light of modern neurophysiological studies. Weddel suggested that stimulation of peripheral receptors by noxious agents produces a spatio-temporal pattern of nervous impulses, which is interpreted as pain within the higher cerebral centres. Such patterns of nervous activity may be produced by many physical phenomena such as pressure, puncturing, squeezing, tension, and by alteration in temperature or, by chemical effects, such as the alteration of pH or the concentration of histamine-like substances, serotonin, bradykinin, and other polypeptide compounds.

### (*b*) *Peripheral receptors*

There is no doubt that specialized receptor organs for pressure and stress do occur in skin, tendons, and ligaments. But the old concept of absolute *specificity* of end organs for pressure (Paccini), cold (Krause), and traction (Meissner and Ruffini), is no longer acceptable. Weddel and Winkelman have demonstrated in the dermis that most cutaneous sensory nerve endings are made up of a dense network of unmyelinated fibres. Sensations of pressure, touch, or pain, depending upon the impulse pattern invoked, may occur when these are stimulated. Similar networks of unmyelinated nerve fibres have been described in the walls of blood vessels, particularly arteries, in periosteum, bone, in synovium and joint capsule. In muscles, a similar role is conducted by small myelinated fibres. Cartilage has no sensory end organs. Also, end organs of Golgi, Vatter–Paccini corpuscles etc., can be identified in ligaments and tendons where they act as stretch receptors, for muscle control, and perhaps for pain. Bone and periosteum respond to pressure, percussion or tension. Identifiable nerve endings have been described in the annularis fibrosus of the intervertebral disc but not in the nucleus pulposus. When the annulus fibrosis is disrupted this is believed to result in the pain of 'lumbago' and paravertebral muscular spasm (Hirsch and Schajowicz, 1953). Capsule responds to both tension and traction and is certainly the most sensitive of all joint structures. Although synovium contains scattered free new endings in two plexuses—deep and superficial—it is difficult to elicit their function experimentally because of the proximity to capsule. In muscle,

TABLE 1.1 Articular receptor system

| *Type* | *Morphology* | *Location* | *Nerve fibres* | *Behaviour* |
|---|---|---|---|---|
| I | Encapsulated globular corpuscles (100 μm × 40 μm) | Fibrous capsule (superficial layers) | Myelinated (6 μm–9 μm) | Static and dynamic mechanoreceptors; low threshold, slowly adapting |
| II | Encapsulated conical corpuscles (280 μm × 120 μm) | Fibrous capsule, deeper layers, fat pads | Myelinated 9 μm–12 μm) | Dynamic mechanoreceptors; low threshold, rapidly adapting |
| III | Encapsulated fusiform corpuscles (600 μm × 100 μm) | Joint ligaments | Myelinated (13 μm–17 μm) | Dynamic mechanoreceptors; high threshold, very slowly adapting |
| IV | Plexuses and free nerve endings | Fibrous capsule, fat pads, ligaments, vessels | Myelinated (2 μm–5 μm) Unmyelinated (<2 μm) | Pain receptors; high threshold, non-adapting |

(After B. Wyke, 1969)

pressure and pain receptors are related to the presence of small myelinated fibres. Wyke and his co-workers over the past 10 years have been building up a very detailed picture of articular receptor systems (Table 1.1) as well as the articular nerves (Table 1.2) feeding back from

TABLE 1.2 Articular nerves

| *Group number* | *Diameter range (μm)* | *Structure* | *Function* |
|---|---|---|---|
| I | 13–17 | Large myelinated | Mechanoreceptor afferent (from ligaments) |
| II | 6–12 | Medium myelinated | Mechanoreceptor afferent (from capsule and fat pads) |
| III | 2–5 | Small myelinated | Pain afferent |
| | <2 | Unmyelinated | Pain afferent<br>Vasomotor efferent |

(After B. Wyke, 1969)

the system. In this they have identified 'pain' receptors of the high threshold, non-adapting type and impulses and from these nerves they pass along both the small myelinated and unmyelinated articular nerves.

The complexity of pain receptor systems in the thoracic spinal tissues has been described and summarized by Wyke (1968) and are applicable to elsewhere in the vertebral column. Pain-sensitive nerve endings have been found in fibrous capsule of apophyseal joints, in the longitudinal (particularly the posterior) flaval, and interspinal ligaments, in the periosteum of vertebral bony structures, in the dura mater and epidural adipose tissue, and in the walls of blood vessels—arteriolar as well as venous.

*(c) Nerve pathways*

The conduction velocity and frequency of impulses in afferent nerve fibres is dependent upon fibre diameter and have been described in at least four groups of sensory pathways:

TABLE 1.3

| *Type* | *Velocity* (m/sec) | *Fibre diameter* (μm) | *Fibre function* |
|---|---|---|---|
| A | 120 | 15–25 | Proprioceptive afferents from the skin and joints |
| B | | Less than 2 | Unmyelinated pain afferents accompanying sympathetic fibres from muscle and bone |
| C | 10 | 5–15 | Afferents from muscle and tendon |
| D | | | Myelinated fibres in association with visceral nerves |

These afferent impulses are carried within the peripheral nerves to spinal root ganglia and then to the cord where they synapse within one or two segments of the dorsal column before crossing the midline to form the contralateral, lateral spino-thalamic tract. At the site of dorsal column synapse the pathway of pain fibres is regulated by fibres descending in the ipsilateral corticospinal tract. Melzack has described a controlling mechanism acting at every junction at which nerve impulses are relayed from one neurone to the next on their cerebral ascent utilizing psychological or emotional events in order to appreciate the quality and quantity of ultimate perception of pain. There are five links in the path by which pain reaches the cerebrum:

1. The spino-thalamic tract is excitatory in function, carrying the majority of pain impulses and can be cut off by division or reduced by analgesic drugs.

2. The central tegmental tract is inhibitory in function but its division actually increases pain sensitivity, although analgesia depresses it.

3. The central grey pathway. Its division will decrease pain sensitivity, as will analgesic drugs.

4. The ascending reticular system whose division will alert the entire brain, but cannot be depressed by analgesic drugs.

5. The medial lemniscal tract—for proprioception and light touch and is not affected by division or analgesic drugs.

It has been shown that the initiating afferent input impulses are influenced by descending fibres from supraspinal cells at cord level.

*Pain may be:*

(*i*) *Local:* when felt at the site of pathological processes in superficial structures. It is usually associated with local tenderness to palpation or percussion.

(*ii*) *Diffuse:* appears to be more characteristic of deeply lying tissues and has a more or less segmental distribution.

(*iii*) *Radicular:* commonly expressed as sciatica or brachalgia. This paroxysmal pain is characterized by its radiation from the centre to the periphery in a strict anatomical sense but not necessarily in continuity. It is often associated with paraesthesia and tenderness along the nerve root. Clinical examination will frequently show neurological disturbances of sensory loss, reflex depression or loss and muscle weakness. This will be described in greater detail under various clinical conditions. However it does differ from the pain due to a neuritis (from an infective agent, e.g. Herpes Zoster, from a metabolic disturbance, e.g. lead or mercury poisoning, etc.) which is more continuous until relief of the disease process or total destruction of the neural tissue occurs.

(*iv*) *Referred pain;* or pain which is experienced in other areas, besides that felt in the area of initial stimulation. This is seen when there is injury or disease affecting either somatic or visceral structures, and results from 'misplaced pain projection because of cortical mis-representation'. This occurs because of the convergence of sensory pathways onto a single cell within the cord or higher centres. Gaze and Gordon (1954) and McLeod (1958) demonstrated such confluence of visceral and cutaneous impulses in the thalamus of the cat. Whitty and Hockaday (1967) have suggested recently that there is possible influence upon such convergence points of stored 'pain experience', from cells within the brain, rather than only at the spinal-cord level.

The early experiments of Kellgren and Samuel (1950), and of Whitty and Willison (1958) have helped to define some characteristics of referred pain in different body tissues particularly arising from spinal disturbances. This will be described in Chapter 13.

Feinstein *et al.* (1954) described how the injection of muscle and site of intervertebral articulations produced gripping, boring and cramp-like pain in a segmental plane but often with overlapping accompanied by muscle spasm and autonomic effects such as hypotension, nausea, and bradycardia. They found that in such cases there was less segmental distribution because of extensive arborization of nerve endings and the excitation within the internuncial pools which allowed extensive overflow into neighbouring segments. Whitty and Hockaday (1967) in repeating these experiments in 28 normal subjects did manage to produce referred deep pain although not common and usually only specific to each of the individuals and not to the group as a whole. Anaesthetizing the site of the stimulus by local anaesthetic agents did abolish the referred reflexes. Skin hyperalgesia was not a constant feature.

There still remains much uncertainty about the true nature of referred pain especially if one believes that pain is part of a specialized system, or that the convergence is only at the cord level or only in the cortex or central area of the brain.

## Specific types of pain

*Bone pain* has a deep boring quality usually attributable to the stimulus of internal tension as seen in osteomyelitis, expanding tumours, and vascular lesions of bone such as Paget's disease. The deep boring night pain of osteoarthritis is thought to be of vascular origin—particularly an increase on the venous side. However, one must differentiate this type of pain in osteoarthritis from that due to soft tissue disturbances of capsular fibrosis and muscle spasm which can be often aggravated by unguarded movements. Pain of a similar 'boring' nature but maybe more diffuse occurs in generalized osseous diseases such as osteomalacia, osteoporosis and hyperparathyroidism or metastatic lesions (myelomatosis, carcinomatosis) of the vertebral column, ribs, pelvic, and shoulder girdles. Such pain may become more severe due to pathological fractures either macro or micro in severity.

Fracture pain has a different character and is often sharp or piercing and is characteristically relieved by rest. Pain which is unrelieved is ominous and suggests serious disease processes.

*Muscle and tendon pain*

Many unmyelinated nerve fibres are associated with the rich blood supply to muscle. In addition there are many efferent motor fibres to the muscle end plate which can subserve a sensory function by virtue of their control over muscle contraction, which when abnormal, is experienced as pain.

Muscle 'cramps'—a typical type of pain—may be the result of direct injury or the effect of chemical irritants such as calcium deficiency, lactic acid, and to an increased factor P—with or without tissue anoxia. This appears to result in abnormal contraction patterns and is often recorded in elderly people at night. Calcium as lactate or gluconate, quinine sulphate all appear to ease it.

Pain due to direct injury is usually described as 'tearing' and sharp and is followed by a soreness aggravated by movement. The cramp-like pain characteristic of intermittent claudication secondary to atherosclerosis, or Volkmann's ischaemia and in the anterior tibial syndrome is aggravated by muscle movement, either passive or active. It results from oedema secondary to tissue necrosis and the release of chemical irritants, with alteration in the muscle circulation.

Muscle spasm refers to sustained muscular contraction and is felt as deep diffuse persistent pain often described as like 'toothache'. Characteristically, in sciatica it produces a scoliosis and in brachialgia a torticollis due to lumbar and cervical nerve root irritation. It is accompanied by local tenderness and a feeling of hardness of the muscles.

Paroxysmal cramp-like pain accompanied by rigidity or excessive muscle spasm is seen in tetany, caused by increased sensitivity of the neuromuscular unit to the hypocalcaemia or the alkalosis. Muscle cramps are also felt in sodium depletion due to hypermotility of muscle cells, a condition rapidly reversed by restoration of the electrolyte balance. Peripheral neuritis may also present cramping of muscle masses as well as paraesthesia. The pain of a fibrositis is related to a specific muscle group with tenderness but its pathology is ill-understood. One should also note that cramp-like pains in generalized skeletal muscles in adolescents after exercise can be due to rare myoglobinuria specially if the urine contains the deep brown pigment.

*Joint pain* is difficult because of the complexity and number of tissues involved in its structure which have been already described in those of the spinal column.

In addition the synovium is known to have two plexuses of nerve fibres—a superficial plexus lying in close proximity to the capsule and a deeper one running in the synovial villi, where it is intimately related to the blood vessels.

The capsule has a rich supply of somatic sensory fibres and proprioceptive fibres in the form of specialized Golgi apparatus, Vatter–Paccini corpuscles and Ruffini-like endings. Leriche clearly demonstrated that the capsule is obviously concerned in joint pain appreciation by showing that local anaesthetic when injected into the capsule was more effective than an intra-articular injection.

Coomes (1963) by injecting hypertonic saline into joint capsule, and the periosteum of the femoral neck produced patterns of pain around the hip. Irritation of the anterior capsule produced pain in the groin, buttock, anterior thigh, and knee, whereas irritation of the posterior capsule produced pain in the buttock, posterior aspect of the thigh, and heel. Articular cartilage has been shown to be insensitive to stimuli. Bone contains many sensory endings in the periosteum and entering blood vessels.

Numerous pathological states can be a source of stimuli to the various receptor systems, e.g. (1) vascular—especially arteriolar hyperemia or venus engorgement. The deep burning