THIRTEENTH EDITION

SEWARD'S BEDSIDE DIAGNOSIS

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Churchill Livingstone

Seward's Bedside Diagnosis

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Preface

This present edition marks the fortieth year since the original publication of *Bedside Diagnosis* in 1949. It was written by Charles Seward during the closing stages of the Second World War when he was serving in the Royal Army Medical Corps in India. In it he classified the causes of a disease according to the symptoms and signs it produces rather than its aetiology and pathology, this being the way in which it presents in clinical practice. This novel approach was encouraged by the late Lord Cohen of Birkenhead, Professor of Medicine at the University of Liverpool and later President of the General Medical Council. In a foreword to the first edition he said that the student who masters the principles on which this handbook is based will have an intelligent and rewarding approach to the diagnosis of disease, and will have laid a foundation which will remain whatever stress the superstructure of later knowledge may impose upon it. And even the experienced practitioner will learn much from its text.

The almost unparalleled demand for *Bedside Diagnosis* over the past 40 years has resulted in thirteen English editions and its translation into Czechoslovakian, German, Greek, Portuguese and Spanish. Its continuing success is a tribute to the wisdom and vision of Charles Seward who died in November 1987 at the age of 89.

The ever-increasing advances in clinical medicine and diagnostic techniques by the seventh edition called for the help of a younger colleague in active hospital practice, and in 1964 he enlisted my help in the task of updating successive editions. Many of the chapters have been completely rewritten and none have gone unaltered. In the present edition there are new sections on such topics as the acquired immunodeficiency syndrome (AIDS), Alport's syndrome, the adult respiratory distress syndrome, anorexia nervosa, Cryptosporidia infection, diabetes mellitus, epiglottitis, the haemolytic-uraemic syndrome, hypertrophic cardiomyopathy, hypothyroidism, IgA nephropathy, the neuroleptic malignant syndrome, parvovirus infection and Reye's syndrome.

I am grateful to Mr Patrick Beasley and Mr Wilfred Selley for their advice on dysphagia and facial pain, and to Dr Harry Hall for his chapter on Coma. Dr Ian Cruikshank and Dr Dilwyn Morgan kindly supplied up-to-date statistics on the prevalence of infectious diseases and mortality rates in Britain. Thanks are also due to Dr John Barraclough, Dr Tom Hargreaves and Dr Miles Joyner for their help in compiling the tables of 'Normal Values' which complete the book.

I am particularly indebted to Mrs Mary Wood, secretary to the Postgraduate Medical School of the University of Exeter, who has spent many hours typing the manuscripts for this and previous editions of the book. Finally I remain obliged as always to Churchill Livingstone for the help and support that I have received from them.

Exeter, 1989 D. M.

Introduction

This book has been written to meet what is believed to be a need both of the medical student and the qualified practitioner. Its aim is to provide a link between the clinician's findings at the bedside or in the consulting room and the systematic description of diseases in the more orthodox textbooks of medicine.

The first concern of any doctor when confronted with an ill patient is to reach a diagnosis as soon as possible, for on this depends the management of the case. The means to this end are his or her clinical knowledge and skill in eliciting the history and physical signs, supplemented where necessary by laboratory and radiological investigations.

Some disorders may be recognized at the first encounter but if not recourse to textbooks may be of little immediate help — they tend to group and describe diseases under physiological systems. This presumes, of course, that a tentative diagnosis has already been made. A more rational approach is to determine the most significant symptoms or signs and to consider their most likely causes in the light of the age, sex and other circumstances of the individual patient. This is the approach adopted in this book. There are not many major symptoms and signs, and the most important are considered in separate chapters.

The starting point is to obtain a full and, if possible, accurate history. The student is sometimes instructed to let the patient tell his own story but one may be baffled by a garrulous, confused, nervous, forgetful or untruthful patient. In these circumstances it is necessary to guide him towards eliciting the primary symptom, rather than those arising from it. A few examples may clarify the principles involved.

A patient complains of dyspnoea, palpitations and retrosternal pain on exertion and presents an appearance of pallor. Anaemia is a possibility and the blood picture must therefore be ascertained. If anaemia is found it is likely to be the significant physical sign, and its causes should be considered rather than those of the other symptoms mentioned, which proceed from and are secondary to it.

However, the anaemia may in turn be due to a more fundamental cause, such as haemorrhage. Thus a patient may consult the doctor because of giddiness or faintness. On being interrogated he describes what has evidently been a loss of blood in his stools. Clearly this is the significant condition which calls for investigation. Another patient may complain of debility and loss of weight but if abdominal pain, vomiting, diarrhoea,

pyrexia or anaemia are present the causes of these should be considered first.

No consultation is complete without an enquiry as to the taking of drugs prescribed or bought over the counter. Drug-induced symptoms have been reported in as many as 20% of hospital patients, and in one general practice in Britain it was found that 87% of patients over the age of 75 were on medication. In old people then, in whom there may be a slowing of metabolism and impaired renal excretion, the possibility of side-effects should always be borne in mind.

Arrangement of chapters

Most chapters are concerned with a symptom or sign and begin with a synopsis of its more important causes. The pathophysiology is then considered. The diagnostic approach which follows is intended to show how the clinician, with the synopsis of causes in mind, should analyse it. This analysis will narrow down the field of possible causes and allow the doctor to select the most likely.

Diseases are important not on account of their rarity or scientific interest but in the degree to which they affect the life and health of the population. The student should cultivate a preference for the probable; a small bird on a chimney top may certainly be a canary, but it is much more likely to be a sparrow.

In the text the aetiology and clinical features of each cause or disease are discussed, followed by the relevant investigations. Many disorders present with more than one symptom or sign and a given entry may be brief to avoid unnecessary repetition. Where a fuller description is given in another chapter this is indicated by a reference to the appropriate page.

The alternative to some such diagnostic approach as is given in this book is a haphazard consideration of the most likely causes that come to mind. This is no way to undertake that most fascinating, responsible and rewarding of all forms of detection — the diagnosis of disease.

The index

At the bedside or in the consulting room the clinician is confronted not with diseases but with symptoms and signs. In this book a score of these is considered and the synopsis of each chapter analyses each symptom or sign under discussion into its causes or the diseases giving rise to it.

The index, which is chiefly one of diseases, is the reverse of the structure of the book in that it refers back from a disease to the chapters where each symptom or sign to which it may give rise is discussed. It has the additional purpose of listing the more important ways by which a disease may manifest itself. Bold type is used to indicate the fullest description of the condition.

Contents

CHAPTER 1

The physiology of pain

We all experience pain at some time or another — it is the commonest and therefore the most important symptom of disease. The term encompasses a wide variety of unpleasant sensations associated with tissue damage caused by trauma, inflammation, neoplasia, ischaemia or extremes of temperature. Pain may be described as dull, aching, colicky, stabbing, constant, burning or agonising depending upon the site and nature of the injury. Moreover, the response to pain varies greatly in different individuals and is modified by temperament and psychological factors. This adds to the diagnostic difficulties because, in addition to the objective analysis of pain in the various parts of the body which is the subject of this book, the clinician will have to assess the patient's psychological make-up.

Peripheral receptors

Peripheral pain receptors in most tissues consist of three-dimensional plexuses of unmyelinated fibres passing between the cells in all directions. Similar plexuses are present in the walls of arteries, arterioles, veins and venules and encircle each blood vessel embedded in the adventitial sheath. Free nerve endings appear to be largely confined to the cornea, teeth, tendons and ligaments.

Pain impulses arise from the depolarization of these sensory nerve endings which occurs in response to mechanical deformation or to a change in the composition of the surrounding fluid. The latter may be caused by an alteration in the hydrogen ion concentration or by the release of compounds such as histamine, bradykinin, serotonin (5-hydroxytryptamine) and prostaglandins from damaged cells. Prostaglandins are particularly important in this context as they appear to enhance and prolong the effect of other pain-producing substances. Prostaglandins are long-chain fatty acids derived from arachidonic acid which is liberated from membrane-bound phospholipids. The analgesic properties of aspirin and other non-steroidal anti-inflammatory drugs stem from their ability to act as prostaglandin synthetase inhibitors.

Peripheral pathways

'Somatic' pain originates in the skin, adjacent mucous membranes, subcutaneous tissues, musculoskeletal system and the parietal serous membranes.

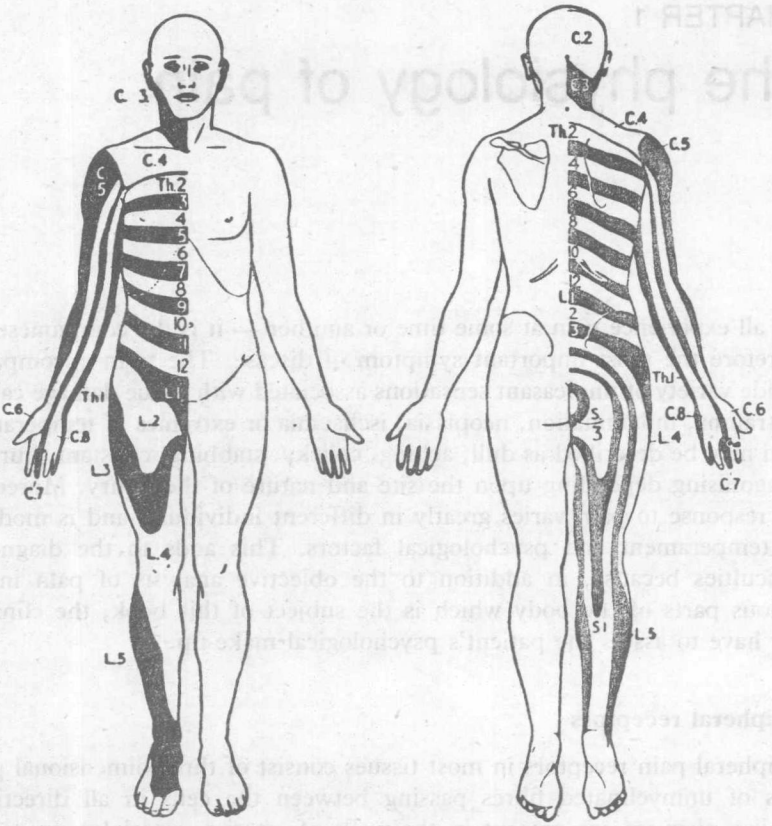


FIGURE 1

The impulses that give rise to somatic pain are carried by fine myelinated and unmyelinated sensory nerves to the spinal cord via the posterior root and trigeminal ganglia. The parietal serous membranes lying within the body cavity include the outer coverings of the dura, pleural spaces, pericardium and peritoneum, and pain arising from these structures is interpreted as coming from the corresponding dermatome. Similarly, when a sensory nerve is damaged anywhere along its course, the pain produced is felt in the peripheral tissue from which it comes. Examples of such 'referred' pain are the pain felt in the ring and little finger when the ulnar nerve is compressed at the elbow, or the sciatic pain that arises from pressure on posterior nerve roots in the lumbar spine.

Pain impulses generated in the viscera and the membranes associated with them follow a different route to the posterior root ganglia. They travel in afferent sympathetic nerves to the corresponding ganglia of the sympathetic trunk before joining nerves from the rest of the body. Visceral and deep somatic pain are more diffuse and less well-localized than pain arising from more superficial structures. This is possibly because there are fewer sensory

receptors in the deeper tissues. Nearly all the sensory nerves enter the spinal cord via the posterior roots and terminate in the grey matter of the dorsal columns.

Spinal pathways

On entering the cord the pain fibres divide into many branches and form synapses in the superficial layers of the dorsal horns. Most of the noxious impulses which eventually reach consciousness in the cerebral cortex enter the central nervous system via the substantia gelatinosa. Here an intricate network of interconnecting neurones begins the task of filtering and modulating the signals before transmitting them to the brain.

The nature of the neurotransmitters in the spinal cord is still uncertain. However they may include excitatory aminoacids such as L-glutamate and L-aspartate, or one of the many polypeptides that have been isolated from nervous tissue. Substance P is the most promising of these polypeptides. Opioid peptides such as enkephalins are present in the dorsal horns and probably play a major role in weakening pain signals at this level by binding to the opiate receptors there. Epidural injections of small doses of morphine in man have been shown to produce complete analgesia for many hours.

According to the 'gate control' theory of Melzack and Wall the perception of pain is regulated by some kind of neurological gate in the substantia gelatinosa which can be opened or shut by appropriate stimuli. Incoming signals from large tactile sensory fibres and descending spinal pathways 'close the gate' by presynaptic inhibition of the neurones carrying the pain traffic, thereby reducing it to acceptable levels.

Whether this mechanism involves the release of enkephalins is still debatable but the theory would explain why vigorous massage relieves pain by 'rubbing it better' and provides a physiological basis for pain relief by transcutaneous electrical nerve stimulation. It has been suggested that the apparent analgesic effect of acupuncture works in a similar way. The afferent impulses from the acupuncture point may inhibit the pain traffic either locally in the spinal cord or — more likely — by activating the descending inhibitory pathways from the brain-stem nuclei. It may be relevant that the Chinese have claimed that acupuncture is more successful the higher up the body one goes, being particularly effective when applied to the neck.

From the dorsal horns the pain impulses ascend by two main routes, a multisynaptic short fibre system which communicates with other segments of the cord before reaching the reticular formation in the brainstem, and oligosynaptic long-fibre tracts in which the axons cross the midline and ascend directly to the reticular formation or the thalamus itself. The lateral spinothalamic tract is largely concerned with carrying discriminative somatic sensations which will help to locate the source of the pain. The spinoreticulothalamic tract conveys the messages which determine the quality and intensity of the pain.

Pathways in the brain

The reticular formation in the brain stem contains a number of nuclei which serve as relay stations on the spinoreticulothalamic pathways. From here the painful messages are passed on to the thalamus to be further processed before reaching consciousness in the cerebral cortex. The autonomic manifestations of sweating, peripheral vasoconstriction, tachycardia, nausea and vomiting which may accompany any severe pain are probably mediated by the fibres which radiate from the reticular formation to other centres in the brain stem and the hypothalamus.

In addition to these ascending pathways fibres descend from the grey matter adjacent to the aqueduct and floor of the third ventricle to the raphe nucleus in the reticular formation. This nucleus is the origin of the descending dorsolateral tract which terminates in the substantia gelatinosa of the spinal cord. All these neural junctions contain opiate receptors and high concentrations of endogenous opioid peptides. Morphine and other opiate drugs probably exert their main analgesic effect by binding to these receptors, thus reducing synaptic transmission in the ascending pathways and at the same time activating descending inhibitory impulses which close the 'gates' to incoming noxious signals in the dorsal horns.

The influence of the psyche on an individual's response to a particular painful situation may be exerted at any level in the central nervous system. Whilst it may not be possible to 'wish a pain away' there is no doubt that mechanisms do exist which allow some control by the mind over the physical discomforts of the body.

Neuroendocrine interactions

This brief and inevitably simplified account of the physiology of pain would be incomplete without some reference to the presence of endogenous opioid peptides in the pituitary gland. Soon after the discovery of the enkephalins in brain tissue and the gut it was realised that the met-enkephalin sequence of groups within the molecule was present in beta-lipotrophin, a polypeptide secreted by the anterior lobe of the pituitary. It was also present in three distinct fragments of this hormone which have been found in the peripheral blood; these are now known as endorphins. All these substances have opiate-like activity but differ from the enkephalins in having a longer-lasting action. Beta-lipotrophin and its related endorphins are derived from the same precursor molecule as corticotrophin and their plasma concentrations also rise in response to stress. The role of these opiate hormones in modulating pain perception has still to be determined, but their increased secretion during stressful situations might explain the transient freedom from pain which has been reported immediately after a severe injury. That they are potentially capable of achieving this is shown by the recent demonstration that the intrathecal injection of a few milligrams of a synthetic endorphin produced total analgesia for more than 24 hours in some patients with intractable cancer pain.

DIAGNOSTIC APPROACH

The distinctive features of any pain are of great diagnostic importance and it is recommended that they should be analysed in the following order.

Position, radiation and character

The site of the pain must be ascertained as precisely as possible. Somatic pain is usually well localised to the affected area but referred pain from the deeper tissues may be misleading. This particularly applies to pain arising from the spine and parietal serous membranes. For example, the dome of the diaphragm is supplied by the phrenic nerve (C4) and inflammatory lesions above or below the diaphragm may cause pain in the shoulder.

Visceral pain, on the other hand, is usually dull, diffuse and poorly localised, being felt mainly in the midline of the body. It too may radiate to involve other structures; thus the pain of renal calculi may pass from loin to groin whilst pain from an ischaemic heart or inflamed pericardium may be referred to arms, jaw or back.

The terms in which a patient describes his pain reflect both the character of the pain and the patient's temperament. A pain may be described as stabbing or colicky in nature but it may be disabling to one patient and merely inconvenient to another. Colicky or griping pain is caused by spasm of smooth muscle in the gut or genito-urinary system whilst one which pulsates in time with the heart beat is usually vascular in origin.

Duration and incidence

The time a pain has lasted from its first appearance should be ascertained. Acute pain is relatively short-lived but there may have been previous attacks of a similar kind. The frequency of these should be determined and it should be noted whether they are increasing or decreasing in severity and duration. Examples of recurrent pain include trigeminal neuralgia, migraine, angina pectoris and attacks of gallstone or renal colic. Pain which recurs daily but does not last all day is termed 'diurnal'. The patient should be asked for an account of its occurrence from the time of waking onwards. An example is the pain of peptic ulceration which may recur daily for weeks with intervals of freedom from pain between the bouts.

Chronic pain persisting for weeks or months is often only partially relieved by the milder analgesics. It may be constant, as in cranial arteritis and the later stages of invasive carcinoma, or it may fluctuate in intensity without disappearing completely. In severe rheumatoid arthritis, for example, the pain and stiffness in the joints are usually at their worst on waking but may ease off during the day.

Aggravating and relieving factors

These will be referred to where they are relevant to the disorder under

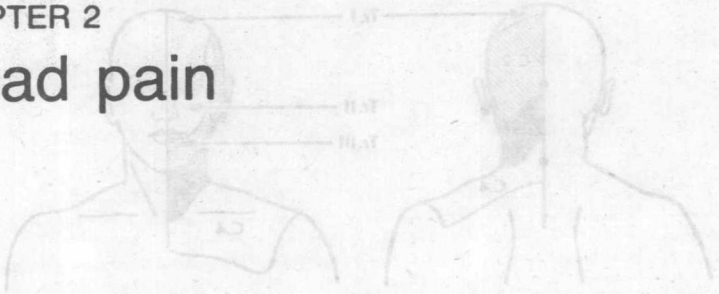
discussion. They include the emotional state of the patient, the menses, taking of meals or any particular food, evacuation of the bowels or bladder, coughing and breathing, posture and movements of the body. The effect of any drugs, particularly analgesics, upon the symptoms should be noted.

Accompanying symptoms

These may not be volunteered and should be sought. Thus migraine may begin with scotomata and end with vomiting. Chest pain accompanied by some shortness of breath is likely to arise from the heart or pleura, and in patients with oesophageal disease there may be a history of dysphagia. In the abdomen renal pain is often accompanied by dysuria, haematuria and vomiting. Finally it should not be forgotten that any severe pain, particularly if it arises from the viscera, may be associated with extreme anxiety, sweating, tachycardia and nausea from stimulation of the autonomic nervous system.

CHAPTER 2

Head pain



The term head pain is used to cover the causes of facial pain as well as the more familiar headache.

SYNOPSIS OF CAUSES*

INTRACRANIAL

INFLAMMATORY

Meningism; **Meningitis**; Encephalitis; Poliomyelitis; Malaria; Cerebral abscess; Cranial arteritis.

NON-INFLAMMATORY

Migraine; Cluster headache; Concussion; Extradural haemorrhage; Subdural haemorrhage; **Subarachnoid haemorrhage; Stroke**; Neoplasm; Benign intracranial hypertension.

CRANIAL

Dental disease; Otitis and mastoiditis; Sinusitis; Disease of skull.

EXTRACRANIAL

Trauma; Cervical spondylosis; **Glaucoma; Corneal ulcer; Iritis**; Scleritis; Trigeminal neuralgia; Temporo-mandibular neuralgia.

GENERAL

Fever; Hypertension; Drugs; Psychogenic causes.

* Bold type is used for causes more commonly found in Europe and North America.

PHYSIOLOGY

Most people suffer from headache from time to time. Pain in the head, like pain elsewhere, is conveyed to the cerebral cortex by sensory nerves; it may be localised in their surface distribution or felt diffusely in the head as a whole. The nerves mainly concerned are:

1. The fifth or trigeminal nerve which supplies the face and underlying structures, the anterior two thirds of the scalp and the underlying perios-

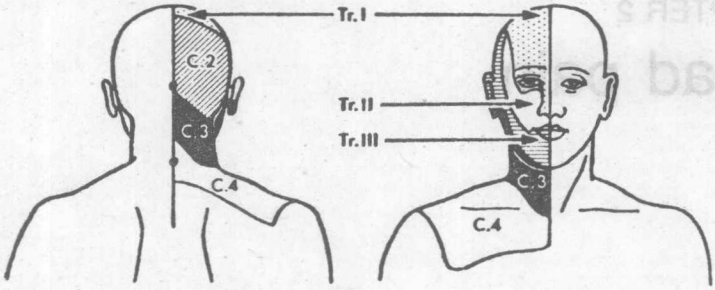


FIGURE 2

teum outside the skull. Within the skull it supplies the dura and vessels of the anterior and middle fossae in front of the tentorium cerebelli.

2. The first three cervical nerves which supply the posterior third of the scalp and periosteum and the trapezius muscle outside the skull. Within the skull they supply the dura posterior to the tentorium and the vessels of the posterior fossa.

CRANIAL PAIN

The skull itself is insensitive to pain; bony lesions such as metastases or Paget's disease seldom give rise to any discomfort. Pain may be due to dental disease, acute sinusitis, otitis or mastoiditis.

INTRACRANIAL PAIN

The brain itself is insensitive to pain. The pain-sensitive tissues are the cerebral and dural arteries, the large veins and the venous sinuses. Pain may arise from:

1. Inflammation of cerebral arteries, as in cranial arteritis
2. Dilatation of arteries as in migraine, fever or due to the effects of drugs, including alcohol
3. Traction or displacement of cerebral blood vessels, such as may occur in tumour, abscess or haemorrhage
4. Inflammation of the dura, as in meningitis

EXTRACRANIAL PAIN

Sustained spasm of the scalp or neck muscles is a common cause of pain in tension headache or cervical spondylosis. It is often accompanied by local

tenderness felt mainly in the frontal and trapezius muscles. Pain may arise from inflammation, dissection or dilatation of extracranial arteries. For example, the superficial temporal artery is frequently involved in cranial arteritis. Ocular disease such as acute glaucoma or iritis causes varying degrees of extracranial pain.

Finally it may be said that the great majority of headaches are due to vascular disturbance or to sustained muscular contraction in the extracranial muscles.

DIAGNOSTIC APPROACH

The complaint of headache calls for four preliminary enquiries:

1. *Is the headache a 'casual' one?* By 'casual' is meant the occasional headache which many people get for no apparent reason. It is discussed at the end of this chapter under Psychogenic causes, but the possibility of an underlying biochemical disturbance cannot be excluded.
2. *Did head injury precede the onset of the headache immediately or by weeks or even months?* Such a headache may be a sequel to concussion or subdural haemorrhage.
3. *Is fever present?* If so, its causes must be considered. In certain infections, notably typhoid and infections caused by arboviruses, headache may be of such severity as to overshadow the presence of fever.
4. *Are drugs being taken which might account for the headache?* Women should be specifically asked whether they are taking the contraceptive pill.

Position, radiation and character

Descriptions of the character of pain vary so much with the personality of the patient that it is more useful to try to determine the degree of severity. Is this such as to render physical or mental work impossible? The pain of such catastrophes as acute meningitis or subarachnoid haemorrhage is so intense as to be outside the patient's previous experience. Vascular headaches are throbbing in character and are made worse by coughing, jolting and straining.

The position of the pain may be of considerable value in determining the likely pathology. Frontal headaches are by far the commonest and most are casual in type and respond to mild analgesics. Unilateral pain, on the other hand, occurs in migraine, cluster headache, cranial arteritis, trigeminal neuralgia, sinusitis, dental disease and inflammation of the ear. Vertex headache, especially if resistant to mild analgesics, suggests a psychogenic cause.

Duration and incidence

The time the headache first appeared should be established. The tendency