Braunwald
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Harrison's

PRINCIPLES OF INTERNAL MEDICINE

Eleventh Edition

Companion Handbook

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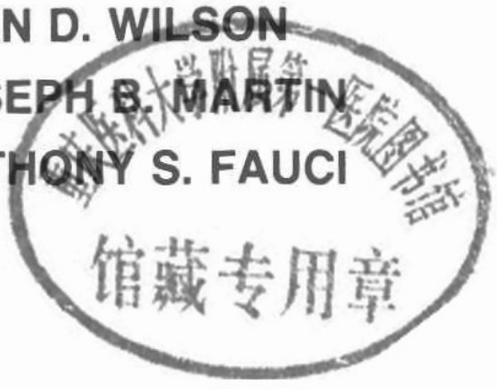

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NOTICE

Medicine is an ever-changing science. As new research and clinical experience broaden our knowledge, changes in treatment and drug therapy are required. The editors and the publisher of this work have made every effort to ensure that the drug dosage schedules herein are accurate and in accord with the standards accepted at the time of the publication. Readers are advised, however, to check the product information sheet included in the package of each drug they plan to administer to be certain that changes have not been made in the recommended dose or in the contraindications for administration. This recommendation is of particular importance in regard to new or infrequently used drugs.

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PREFACE

Most medical students and many residents are often overwhelmed by the sheer quantity of medical information potentially applicable to the diagnosis and treatment of their patients. The editors and authors summarize this vast amount of information in *Harrison's Principles of Internal Medicine,* which is thoroughly revised and updated every three to four years. Although *Harrison's* represents a distillate of the broad field of internal medicine, along with its deep roots in the basic sciences, the total information presented in the book grows steadily, along with the base of useful medical knowledge.

While it would be ideal to have a copy of *Harrison's* in one's pocket at all times, the sheer bulk and weight of the book make this impossible. The editors, with the aid of a few selected contributors, have now condensed the clinical portions of *Harrison's* into this pocket-sized *Companion Handbook* which residents and students can use on their trek through the inpatient, outpatient, and emergency services of a teaching hospital. The *Companion Handbook* consists of brief summaries of the key features of the principal diseases of patients which trainees are likely to encounter on a medical service. The blank pages interspersed in the book are to allow recording of additional information obtained during rounds and conferences to supplement the text. Following the text is a glossary spelling out the abbreviations used throughout the book.

It is important to point out that the *Companion Handbook* should not and cannot be a replacement for a textbook of internal medicine. Rather it is an extension of the Eleventh Edition of *Harrison's*. Each brief chapter in the *Companion Handbook* is referenced to the appropriate chapter(s) in *Harrison's*. The *Companion Handbook* is meant to be used when the resident or student requires a brief introduction to or reminder of an aspect of clinical internal medicine but does not have immediate access to or the time to consult *Harrison's*. Since the quantity of material presented is too brief to stand on its own, it is recommended that the relevant subjects in *Harrison's* be consulted as soon as time permits. Thus, we consider the two books, Harrison's and the *Companion Handbook*, as a single educational package.

Since this is the first edition of the *Companion Handbook*, the editors would be grateful to the readers for their comments concerning its usefulness.

THE EDITORS

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SECTION I
IMPORTANT SYMPTOMS AND SIGNS

1 PAIN AND ITS MANAGEMENT

Pain is the most common symptom of disease. Its management depends on determining its cause and alleviating triggering and potentiating factors.

ORGANIZATION OF PAIN PATHWAYS (See HPIM-11, Fig. 3-1.) Pain-producing (nociceptive) sensory stimuli in skin and viscera activate nerve endings of bipolar neurons of spinal dorsal root or cranial nerve ganglia. After synapse in cord or medulla, crossed ascending pathways reach thalamus and are projected to cortex. An indirect multisynaptic afferent system connects with brainstem reticular formation and projects to intralaminar and medial thalamic nuclei and limbic system. Pain transmission is regulated at level of dorsal horn by descending bulbospinal pathways that contain serotonin, norepinephrine, and several neuropeptides.

Agents that modify pain perception may act to reduce tissue inflammation (corticosteroids, NSAIDs, prostaglandin synthesis inhibitors), to interfere with pain transmission (narcotics), or to enhance descending modulation (tricyclic antidepressants). Anticonvulsants may alter aberrant pain sensations arising from neurogenic sources, e.g., demyelination of peripheral nerves.

EVALUATION Pain may be of *somatic* (skin, deep tissues, joints, muscles) or *neuropathic* (injury to nerves, spinal cord pathways, or thalamus) origin. Characteristics of each are summarized in Table 1-1.

Sensory symptoms and signs in neuropathic pain are described by the following definitions: *neuralgia:* pain in distribution of a single nerve, as in trigeminal neuralgia; *dysesthesia:* spontaneous

TABLE 1-1 **Characteristics of somatic and neuropathic pain**

Somatic pain:
Nociceptive stimulus usually evident.
Usually well localized; visceral pain may be referred.
Similar to other somatic pains in patient's experience.
Relieved by anti-inflammatory or narcotic analgesics.
Neuropathic pain:
No obvious nociceptive stimulus.
Often poorly localized.
Unusual, dissimilar from somatic pain.
Only partially relieved by narcotic analgesics.

Modified from Maciewicz R, Martin JB: HPIM-11, p. 15.

TABLE 1-2 **Drugs used to relieve pain**

Nonnarcotic analgesics: equivalent doses and intervals

Generic name	Dose, mg	Interval
Aspirin	750–1250	q 3 h
Phenacetin	750–1000	q 3 h
Acetaminophen	600–800	q 3 h
Phenylbutazone	200–400	q 4 h
Indomethacin	50–75	q 4 h
Ibuprofen	200–400	q 4 h
Naproxen	250–500	q 4 h
Nefopam	60–120	q 4 h

Narcotic analgesics compared to 10 mg morphine sulfate (MS)

Generic name	IM dose, mg	PO dose, mg	Differences from MS
Oxymorphine	1	6	None
Hydromorphine	1.5	7.5	Shorter acting
Levorphanol	2	4	Good PO-IM potency
Heroin	4		Short-acting
Methadone	10	20	Good PO-IM potency
Morphine	10	60	
Oxycodone	15	30	Short-acting
Meperidine	75	300	None
Pentazocine	60	180	Agonist-antagonist
Codeine	130	200	More toxic

Anticonvulsants

Generic name	PO dose, mg	Interval
Phenytoin	100	q 6–8 h
Carbamazepine	200	q 6 h
Clonazepam	1	q 6 h

Antidepressants

Generic name	PO dose, mg	Range, mg/day
Doxepin	200	75–400
Amitriptyline	150	75–300
Imipramine	200	75–400
Nortriptyline	100	40–150
Desipramine	150	75–300
Amoxapine	200	75–300
Trazodone	150	50–600

Reproduced from Maciewicz R, Martin JB: HPIM-11, p. 16.

background pain of aching, burning quality; *hyperalgesia* and *hyperesthesia:* exaggerated responses to nociceptive or touch stimulus, respectively; *allodynia:* perception of nonnociceptive stimulus as painful, as when vibration evokes painful sensation. Reduced pain perception is called *hypalgesia* or, when absent, *analgesia. Causalgia* is continuous severe burning pain with indistinct boundaries and accompanying sympathetic nervous system dysfunction (sweating, vascular, skin, and hair changes—sympathetic dystrophy) which occurs after injury to a peripheral nerve.

MANAGEMENT Acute somatic pain: Usually effectively treated with nonnarcotic analgesic agents (Table 1-2). Narcotic analgesics are usually required for relief of severe pain.

Neuropathic pain: Often chronic; management is particularly difficult. The following drugs, in combination with careful assessment of underlying factors that contribute to pain (depression, "compensation neurosis"), may be beneficial:

1 Anticonvulsants: In patients with neuropathic pain and little or no evidence of sympathetic dysfunction; diabetic neuropathy, trigeminal neuralgia (tic douloureux).

2 Antisympathetic agents: In patients with causalgia and sympathetic dystrophy, surgical or chemical sympathectomy may be tried (see HPIM-11, Chap. 3).

3 Tricyclic antidepressants: Pharmacologic effects include facilitation of monamine neurotransmitters by inhibition of transmitter reuptake. Are useful in management of patients with chronic pain, postherpetic neuralgia, atypical facial pain (see Chap. 4), chronic low back pain (see Chap. 5).

For more detailed discussion of this topic, see Maciewicz R, Martin JM: Pain: Pathophysiology and Management, Chap. 3 in HPIM-11, p. 13

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2 CHEST PAIN

There is little correlation between the severity of chest pain and the seriousness of its cause.

POTENTIALLY SERIOUS CAUSES

MYOCARDIAL ISCHEMIA **Angina pectoris:** Substernal pressure, squeezing, constriction, with radiation typically to left arm; usually on exertion, especially after meals or with emotional arousal. Characteristically relieved by rest and nitroglycerin.
Acute myocardial infarction: Similar to angina but more severe, of longer duration (≥30 min), and not immediately relieved by rest or nitroglycerin. S_3 and S_4 common.

PULMONARY EMBOLISM May be substernal or lateral, pleuritic in nature, and associated with hemoptysis, tachycardia, hypoxemia.

AORTIC DISSECTION Very severe, in center of chest, a "ripping quality," radiates to back, not affected by changes in position. May be associated with weak or absent peripheral pulses.

MEDIASTINAL EMPHYSEMA Sharp, intense, localized to substernal region; often associated with audible crepitus.

ACUTE PERICARDITIS Usually steady, crushing, substernal; often has pleuritic component aggravated by cough, deep inspiration, supine position, and relieved by sitting upright; one-, two-, or three-component friction rub often audible.

PLEURISY Due to inflammation; less commonly tumor and pneumothorax. Usually unilateral, knifelike, superficial, aggravated by cough and respiration.

LESS SERIOUS CAUSES

COSTOCHONDRAL PAIN In anterior chest, usually sharply localized, may be brief and darting or a persistent dull ache. Can be reproduced by pressure on chondrosternal and/or costochondral junctions. In Tietze's syndrome (costochondritis), joints are swollen, red, and tender.

CHEST WALL PAIN Due to strain of muscles or ligaments from excessive exercise or rib fracture from trauma; accompanied by local tenderness.

ESOPHAGEAL PAIN Deep thoracic discomfort; may be accompanied by dysphagia and regurgitation.

EMOTIONAL DISORDERS Prolonged ache or dartlike, brief, flashing pain; associated with fatigue, emotional strain.

OTHER CAUSES

(1) Cervical disk; (2) osteoarthritis of cervical or thoracic spine; (3) abdominal disorders: peptic ulcer, hiatus hernia, biliary colic;

(4) tracheobronchitis, pneumonia; (5) diseases of the breast (inflammation, tumor); (6) intercostal neuritis (herpes zoster)

APPROACH TO PATIENT

- Obtain a meticulous history of the behavior of pain, what precipitates it and what relieves it.
- When localized pain can be reproduced by pressure, it usually originates from chest wall.
- Electrocardiogram *during* chest pain helpful in diagnosis of ischemia (ST segments may be elevated or depressed).
- Workup for angina (Chap. 65) in patients with episodic pain.
- Serum enzymes and evolution of ECG are diagnostic of myocardial infarction (Chap. 64) in patients with prolonged pain.
- Echogram or CT scan of aorta in patients with sudden, severe pain helpful in diagnosis of aortic dissection (Chap. 69).
- Esophageal pH, acid perfusion test, and barium esophagram useful in diagnosis of esophageal pain (Chap. 95).
- CXR for pleurisy, pneumonia, mediastinal emphysema, pneumothorax.

For more detailed discussion of this topic, see Braunwald E: Chest Discomfort and Palpitation, Chap. 4, in HPIM-11, p. 17
