CLINICIAN’S HANDBOOK OF

ORAL AND

MAXILLOFACIAL SURGERY

Edited by

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This book is dedicated to Evie Laskin, my wife for over 63 years. She was always my best friend, my biggest supporter, my kindest critic, and my greatest love. —DML
Preface

Why develop a handbook of oral and maxillofacial surgery when there are already numerous texts available that can provide the clinician with extensive information about the various aspects of the specialty? The problem is that, in certain circumstances, textbooks can be too detailed. They are fine when one has the time to seek out the proper text and then sit and read through long chapters containing extensive information about specific problems or procedures; however, when one is faced with an urgent clinical situation and needs a quick answer, textbooks will not readily serve this purpose.

The main intent of this handbook is to provide important information in a concise and easily searchable format from areas of oral and maxillofacial surgery that can present situations in which immediate answers to clinical problems may be necessary. The authors of the various sections have been selected for their clinical expertise and therefore their ability to know the questions that may arise and the information that will answer these questions.

Although designed as a quick-reference source, this handbook can also serve many other functions. Reading the text in advance allows busy practitioners to easily review a considerable amount of clinically significant information. By doing so, they will not only increase their knowledge base, but also establish a familiarity with the text that will make it easier for them to find necessary information in an urgent situation. The book is also a handy compilation of relevant information for trainees in oral and maxillofacial surgery, as well as those in other hospital-based dental specialties, who are just beginning to learn this material. Finally, it is an organized resource for the review of important information pertinent to those preparing for the American Board of Oral and Maxillofacial Surgery.

I would like to express my sincere appreciation and thanks to all of the contributing authors who gave so freely of their time and effort. Without their willingness to share their knowledge and expertise, this book would not have been possible.
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**Table 11-1** Emergency cart drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Indication</th>
<th>Dosage</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosine</td>
<td>Tachycardia</td>
<td>6–12 mg</td>
<td>Rapid IV push</td>
</tr>
<tr>
<td>Albuterol</td>
<td>Bronchoconstriction</td>
<td>1–2 inhalations</td>
<td>Inhaled</td>
</tr>
<tr>
<td>Aminophylline</td>
<td>Asthma</td>
<td>5–7 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>Arrhythmias</td>
<td>150 mg/10 min</td>
<td>IV</td>
</tr>
<tr>
<td>Ammonia aromatic</td>
<td>Syncope</td>
<td>1 carpule</td>
<td>Inhaled</td>
</tr>
<tr>
<td>Atropine</td>
<td>Bradycardia</td>
<td>0.5–1.0 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Dextrose 50% (D&lt;sub&gt;50&lt;/sub&gt;)</td>
<td>Hypoglycemia</td>
<td>50 mL (25 g)</td>
<td>IV, oral</td>
</tr>
<tr>
<td>Diazepam</td>
<td>Seizures, anxiety</td>
<td>2.5–10 mg</td>
<td>IV</td>
</tr>
<tr>
<td>Diphenhydramine</td>
<td>Allergy, hypersensitivity</td>
<td>25–50 mg</td>
<td>IV, IM, oral</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Hypotension</td>
<td>3–20 µg/kg/min</td>
<td>IV</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Bronchoconstriction, hypersensitivity</td>
<td>0.2–0.5 mg</td>
<td>SC, IM</td>
</tr>
<tr>
<td></td>
<td>Cardiac arrest</td>
<td>0.5–1.0 mg</td>
<td>IV</td>
</tr>
<tr>
<td>Ephedrine</td>
<td>Vasopressor</td>
<td>2.5–25 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Hydrocortisone</td>
<td>Adrenal insufficiency</td>
<td>100 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Ventricular ectopy</td>
<td>1 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td>Morphine</td>
<td>Myocardial infarction</td>
<td>2–8 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Naloxone</td>
<td>Narcotic overdose</td>
<td>0.4 mg</td>
<td>IV, IM</td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>Angina</td>
<td>1/50–1/200 g</td>
<td>SL</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>Seizures</td>
<td>10 mg/kg</td>
<td>IV</td>
</tr>
<tr>
<td></td>
<td>loading</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Verapamil</td>
<td>Supraventricular tachycardias</td>
<td>5–10 mg</td>
<td>IV</td>
</tr>
</tbody>
</table>

IV = intravenous; IM = intramuscular; SC = subcutaneous; SL = sublingual.

- **Pulseless electrical activity (PEA):** The uncoupling of the electrical complexes and the physical contraction of the myocardium; although an electrical rhythm is present, there is no effective cardiac output.

**Tachycardia with a pulse** (Fig 11-2): A tachyarrhythmia of atrial or ventricular origin; the patient may or may not be symptomatic. Includes paroxysmal supraventricular tachycardia and ventricular tachycardia.

**Bradydcardia** (Fig 11-3): Slowing of the heart rate below 50 to 60 beats/min (BPM) with inadequacy of clinical perfusion.
**Pulseless Arrest**

- **BLS Algorithm:** Call for help, give CPR
- **Give oxygen** when available
- **Attach monitor/defibrillator** when available

**Pulseless Arrest Algorithm**

1. **VF/VT**
2. Check rhythm
3. **Shockable**
   - Give 1 shock
     - Manual biphasic: device specific (typically 120 to 200 J; Note: If unknown, use 200 J)
     - AED: device specific
     - Monophasic: 360 J
   - Resume CPR immediately

4. Give 5 cycles of CPR
   - Check rhythm
   - **Shockable**

5. Continue CPR while defibrillator is charging
   - Give 1 shock
     - Manual biphasic: device specific (same as first shock or higher dose)
     - Note: If unknown, use 200 J
     - AED: device specific
     - Monophasic: 360 J
   - Resume CPR immediately after the shock

6. When IV/IO available, give vasopressor during CPR
   - **Epinephrine 1 mg IV/IO**
   - **Repeat every 3 to 5 min**
   - May give 1 dose of **vasopressin 40 U IV/IO** to replace first or second dose of epinephrine

7. Continue CPR while defibrillator is charging
   - Give 1 shock
     - Manual biphasic: device specific (same as first shock or higher dose)
     - Note: If unknown, use 200 J
     - AED: device specific
     - Monophasic: 360 J
   - Resume CPR immediately after the shock

8. Consider amiodarone (300 mg IV/IO once, then consider additional 150 mg IV/IO once) or lidocaine (1 to 1.5 mg/kg first dose, then 0.5 to 0.75 mg/kg IV/IO, maximum 3 doses or 3 mg/kg)
   - Consider magnesium, loading dose 1 to 2 g IV/IO for torsades de pointes
   - After 5 cycles of CPR, go to box 3 above

9. **Asystole/PEA**
   - **Assess shockable rhythm?**
   - **Give 1 shock**
     - Manual biphasic: device specific (same as first shock or higher dose)
     - AED: device specific
     - Monophasic: 360 J
   - **Resume CPR immediately**
   - **Repeat every 3 to 5 min**
     - May give 1 dose of **vasopressin 40 U IV/IO** to replace first or second dose of epinephrine

10. **Assess shockable rhythm?**
    - **Assess shockable rhythm?**
    - **Assess shockable rhythm?**
    - **Assess shockable rhythm?**
    - **Assess shockable rhythm?**

11. **Check rhythm**
    - **Shockable**

12. **If asystole, go to box 10**
    - **If electrical activity, check pulse, if no pulse, go to box 10**
    - **If pulse present, begin postresuscitation care**

13. **Go to box 4**

**During CPR**

- **Push hard and fast** (100/min)
- **Ensure full chest recoil**
- **Minimize interruptions in chest compressions**
  - One cycle of CPR: 30 compressions then 2 breaths, 5 cycles =2 min
  - **Avoid hyperventilation**
  - **Secure airway and confirm placement**
  - **After an advanced airway is placed, rescuers no longer deliver “cycles” of CPR. Give continuous chest compressions without pauses for breaths.**
  - **Give 8 to 10 breaths/minute.**
  - **Check rhythm every 2 minutes**
  - **Rotate compressors every 2 minutes with rhythm checks**
  - **Search for and treat possible contributing factors:**
    - Hypovolemia
    - Hypoxia
    - Hydrogen ion (acidosis)
    - Hypo-/hyperkalemia
    - Hypoglycemia
    - Hypothermia
    - Toxicosis
    - Tamponade, cardiac
    - Tension pneumothorax
    - Thrombosis (coronary or pulmonary)
    - Trauma

**Fig 11-1** Pulseless arrest algorithm. (Reproduced with permission. Advanced Cardiovascular Life Support Provider Manual, 2006. Copyright American Heart Association.)
Bisphosphonate therapy has been considered standard treatment in the management of cancer patients with metastatic bone disease and patients with osteoporosis. The efficacy of these drugs is due to their ability to inhibit osteoclast-mediated bone resorption. However, the postmarketing experience with intravenous and, to a much lesser extent, oral bisphosphonates has raised concerns about potential side effects related to profound bone remodeling inhibition and osteonecrosis isolated to the jaws. This chapter reviews the risk factors, incidence, pathogenesis, prevention strategies, and management of this new complication.

**Indications for Bisphosphonate Use**

**Malignancy**
Based on clinical practice guidelines established by the American Society of Clinical Oncology, the use of bisphosphonates is considered the standard of care for treatment of (1) moderate to severe hypercalcemia associated with malignancy and (2) metastatic osteolytic lesions associated with breast cancer and multiple myeloma, in conjunction with antineoplastic chemotherapeutic agents. Recently, the US Food and Drug Administration (FDA) has broadened the indications for intravenous bisphosphonates to include bone metastases from any solid tumor.

**Osteoporosis**
As a potent suppressor of osteoclast activity, bisphosphonates slow the remodeling process and increase bone mineral density, thereby reducing the risk of fracture in women with osteopenia and osteoporosis. The World Health Organization has established criteria for bisphosphonate therapy that are based on bone density values. Patients with scores between –1.5 and –2.5 (osteopenia) or scores less than –2.5 (osteoporosis) are candidates for antiresorptive therapy.

**Paget Disease**
Paget disease is characterized by osteoclast hyperplasia coupled with compensatory osteoblast hyperactivity. This results in exuberant abnormal bone formation and skeletal deformities. Suppression of osteoclast function with bisphosphonates has emerged as an effective FDA-approved treatment strategy for patients with Paget disease.
Diagnosis of Nerve Injuries

Primary Diagnostic Steps
Patient evaluation following trigeminal nerve injury involves a series of key diagnostic measures:

- Neuropathic symptom assessment
- Assessment of general and orofacial functional impairment
- Maxillofacial clinical examination and imaging findings
- Quantitative sensory test responses

Assessing neuropathic symptoms
Patients should be asked to describe the anatomical location(s) of their altered sensations and to estimate their current level of discomfort or pain on a scale where:

- 0 = no discomfort
- 25% = mild discomfort
- 50% = moderate discomfort/pain
- 75% = severe pain
- 100% = intolerable pain

Patients should then be prompted to characterize their altered sensations by circling terms from a list of neuropathic terms such as: constant, intermittent, rhythmic, steady, brief, triggered, spontaneous, numb, itching, dry, tickling, twitching, wet, rubbery, stretched, swollen, woody, crawling, moving, quivering, vibrating, cool, warm, cold, hot, burning, pricking, stinging, electric, tender, sore, painful, aching, excruciating, cramping, shocking, bitter, sweet, sour, salty, tasteless, other.

These baseline patientsymptom responses are used for assessment of recovery at future clinic visits.

Assessing general and orofacial functional impairment
Patients are asked to estimate their current (baseline) levels of functional impairment following injury, where:

- 0 = no impairment
- 25% = mild impairment
- 50% = moderate impairment
- 75% = severe impairment
- 100% = complete impairment

Patients are then prompted to characterize their specific impairments following injury by selecting from a list of terms such as: eating, talking, swallowing, tasting, toothbrushing, dental care, face washing, smelling, smiling, lovemaking, sleeping, working, socializing, other.

Maxillofacial imaging
Neural imaging has not yet reached the levels of accuracy that precisely define anatomical details of nerve injuries. Nevertheless, postinjury imaging using plain films, tomography, digital, and computed tomography (CT) techniques can be used to assess:
- Paraneural foreign bodies (broken instruments, implants)
- Paraneural bone or dental root impingement
- Irregularities of nerve canal or foramina

Maxillofacial clinical examination

Following nerve injury, a maxillofacial examination is carried out in order to:

- Rule out nonneural sources of noxious pathoses (temporomandibular joint pain, active odontogenic or periodontal disease, sinusitis, sialadenitis)
- Detect sources of secondary nerve injury (mobile bone fractures, osteotomy segments, infection, invasive or compressive pathology)
- Reveal signs of traumatic neuroma formation (pain and tingling responses to digital palpation of nerve trunk distribution (Tinel sign).

Quantitative sensory testing (QST)

The objectives of clinical QST are to determine:

- Loss of sensory detection (hypoesthesia) in the injured nerve distribution
- Presence of neuropathic sensitization (hyperesthesia)
- Overall level of neurosensory recovery toward normalcy

Measuring sensory loss (hypoesthesia) (Fig 22-1)

Clinical neurosensory testing involves the application of graded stimuli to an uninjured (control) nerve distribution, comparing the patient's normal detection capacities to the detection thresholds found within the injured nerve distribution. This is done using:

- Fine touch stimuli such as thin von Frey filaments, cotton, or brush strokes; these stimuli test for integrity of large myelinated nerve fibers (level A)