

SPINAL CORD INJURY



Spinal Cord Injury

Rehabilitation Medicine Quick Reference

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Rehabilitation Medicine Quick Reference

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Compositor: NewGen North America
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Visit our website at www.demosmedpub.com

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Library of Congress Cataloging-in-Publication Data

Spinal cord injury / editor, Thomas N. Bryce ; associate editors, Naomi Betesh ... [et al].
p. ; cm.—(Rehabilitation medicine quick reference)

Includes bibliographical references and index.

ISBN 978-1-933864-47-1

I. Spinal cord—Wounds and injuries—Handbooks, manuals, etc. I. Bryce, Thomas N.
II. Series: Rehabilitation medicine quick reference.

[DNLM: 1. Spinal Cord Injuries—rehabilitation—Handbooks. WL 39 S757 2010]

RD594.3.S6683 2010

616.7'3—dc22

2009024708

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*To the staff and patients of the Mount Sinai
Medical Center Spinal Cord Injury Unit*

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List of Acronyms

ADL	activity of daily living
AIS	ASIA impairment scale
ASIA	American Spinal Injury Association
CSF	cerebrospinal fluid
CT	computed tomography
CVD	cardiovascular disease
EEG	electroencephalography
EKG	electrocardiogram
EMG	electromyography
GI	gastrointestinal
LMN	lower motor neuron
MRI	magnetic resonance imaging
ROM	range of motion
SCI	spinal cord injury
UMN	upper motor neuron
UTI	urinary tract infection

Series Foreword

The Rehabilitation Medicine Quick Reference (RMQR) series is dedicated to the busy clinician. While we all strive to keep up with the latest medical knowledge, there are many times when things come up in our daily practices that we need to look up. Even more importantly . . . look up quickly.

Those aren't the times to do a complete literature search or to read a detailed chapter or review article. We just need to get a quick grasp of a topic that we may not see routinely, or just to refresh our memory. Sometimes a subject comes up that is outside our usual scope of practice, but that may still impact our care. It is for such moments that this series has been created.

Whether you need to quickly look up what a Tarlov cyst is, or you need to read about a neurorehabilitation complication or treatment, RMQR has you covered.

RMQR is designed to include the most common problems found in a busy practice, but also a lot of the less common ones as well.

I was extremely lucky to have been able to assemble an absolutely fantastic group of editors. They in turn have

harnessed an excellent set of authors. So what we have in this series is, I hope and believe, a tremendous reference set to be used often in daily clinical practice. As series editor, I have of course been privy to these books before actual publication. I can tell you that I have already started to rely on them in my clinic—often. They have helped me become more efficient in practice.

Each chapter is organized into succinct facts, presented in a bullet point style. The chapters are set up in the same way throughout all of the volumes in the series, so once you get used to the format, it is incredibly easy to look things up.

And while the focus of the RMQR series is, of course, rehabilitation medicine, the clinical applications are much broader.

I hope that each reader grows to appreciate the Rehabilitation Medicine Quick Reference series as much as I have. I congratulate a fine group of editors and authors on creating readable and useful texts.

Ralph M. Buschbacher, MD

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Preface

Having a spinal cord injury (SCI) can profoundly change a person's life as it can affect nearly all the body systems. It also affects the perception of that person by others. Clinicians who treat persons with SCI need to not only be able to treat the medical complications that can result, but also treat the whole person, helping those affected to return to a productive life integrated within society. This book was developed for all physicians and other health care professionals involved in the care of persons with SCI to provide knowledge to help facilitate this process.

The book addresses over one hundred varied topics related to SCI, ranging from psychological adjustment to treatment of vocal fold paralysis. It is organized into three sections, the first includes the medical and psychological conditions associated with SCI; the second includes common interventions; while the last outlines expected functional outcomes. I hope you find it useful.

Thomas N. Bryce, MD

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Conditions

Airway Issues: Tracheal Stenosis

Kenneth W. Altman MD PhD ■ Chih-Kwang Sung MD MS

Description

Tracheal stenosis is an abnormal narrowing of the tracheal lumen due to an intrinsic or extrinsic mass.

Etiology/Types

Etiology

- Intubation trauma
- Tracheostomy
- Neoplasm
- Infection
- External trauma
- Gastroesophageal reflux disease (GERD)
- Inflammatory/granulomatous/autoimmune disease
- Idiopathic

Types

- Congenital
- Acquired iatrogenic injuries are the most common cause
- Tracheostomy-related stenosis can be classified according to site of lesion: suprastomal, stomal, cuff, and cannula tip stenosis
- Cotton staging system: grade 1, <50% obstruction; grade 2, 50% to 70% obstruction; grade 3, 71% to 99% obstruction; and grade 4, complete obstruction

Epidemiology

- Incidence of postintubation or post-tracheostomy stenosis 10% to 22%, depending on duration and nature of intubation
- 1% to 2% are symptomatic or have severe stenosis

Pathogenesis

Acquired due to intubation tube cuff

- Excessive endotracheal (ET) tube or tracheostomy tube cuff pressure (>30 mm Hg) impairs mucosal capillary perfusion.
- Mucosal ischemia leads to ulcerations and chondritis.
- Lesions heal by fibrosis, resulting in progressive cicatricial stenosis.

Acquired due to tracheostomy tube

- Poor surgical technique or poor visualization/palpation of landmarks
- Traumatic crush injury during procedure inverting cricoid cartilage or tracheal rings into airway

- Trauma of posterior tracheal wall during dilation and tube insertion
- Stomal stenosis secondary to bacterial infection and chondritis, which precipitate inflammation and granulation
- Distal tracheal stenosis from tracheostomy tube tip irritation or aggressive suction trauma
- Granulation tissue can progress and heal by fibrosis

Risk Factors

- Prolonged intubation
- Type and size of ET tube
- Stomal infection
- Hypotension
- Tight fitting tracheal cannula
- Excessive tube motion
- Corticosteroids
- High tracheostomy
- Cricothyrotomy (cricothyroidotomy)
- Excessive resection of anterior tracheal ring or poor tracheostomy procedure technique

Clinical Features

- Cough
- Inability to clear secretions
- Exertional dyspnea
- Stridor (usually biphasic)
- Inability to tolerate capping of tracheostomy tube
- Aponia

Natural History

- Prior history of neck trauma, intubation, or tracheostomy; symptoms may present within days.
- Symptoms may have sudden or gradual onset,
- Stenosis due to chronic inflammatory conditions or GERD is more likely to progress.
- Often asymptomatic until stenosis is >50% to 75% of lumen or diameter is <5 mm.

Diagnosis

Differential diagnosis

- Asthma
- Infection: supraglottitis, croup, bronchitis

History

- Trauma, intubation, or tracheostomy: days or weeks before onset of symptoms
- Dyspnea, increased work of breathing
- Aggravating factors: exercise, infection
- Recurrent pneumonia

Testing

- Helical computed tomography (CT) scan of neck and chest with fine cuts through the larynx and proximal trachea—preferred study
- Magnetic resonance imaging (MRI) to characterize soft tissue mass if present
- Pulmonary function testing (PFT)—may reveal fixed extrathoracic obstruction
- Flexible laryngoscopy
- Flexible or rigid bronchoscopy
- Direct microlaryngoscopy

Pitfalls

- Symptoms of asthma or wheezing may be confused with laryngotracheal stenosis.
- Asthma can be excluded based on PFT and successful response to treatment.

Red Flags

- Acute respiratory distress: requires securing of airway with tracheostomy or rigid bronchoscopy

Treatment

Medical

- Preventive measures: early tracheostomy; properly sized tubes; high volume, low-pressure cuffs
- Humidified air or oxygen
- Heliox therapy to temporarily reduce airway resistance
- Treatment of underlying medical causes of stenosis (eg, infectious or inflammatory)
- Antireflux management: dietary and lifestyle modifications, proton pump inhibitors, H2 blockers

Exercises

- General cardiovascular training to improve pulmonary requirements

Surgical

- Long-term tracheostomy
- Endoscopic repair: laser incision or excision, dilation
- Open repair: segmental tracheal resection with end-to-end anastomosis, laryngotracheoplasty with anterior or anterior-posterior cricoid split, and T-tube placement
- Stent placement

Consults

- Otolaryngology
- Cardiothoracic surgery

Complications of treatment

- Restenosis
- Migration or infection of stent
- Granulation tissue
- Mortality after end-to-end anastomosis up to 5% due to anastomosis breakdown and/or mediastinitis

Prognosis

- One-third of patients can be managed by laser excision, bronchoscopic dilation, or tracheal stenting.
- Two-thirds require open procedures.

Helpful Hints

- The goal of treatment is to improve airway function while retaining laryngeal function. Airway safety is paramount in all cases.

Suggested Readings

- Epstein SK. Late complications of tracheostomy. *Resp Care*. 2005;50(4):542–549.
- Sue RD, Susanto I. Long-term complications of artificial airways. *Clin Chest Med*. 2003;24(3):457–471.
- Zias N, Chroneou A, Tabba MK, et al. Post tracheostomy and post intubation tracheal stenosis: Report of 31 cases and review of the literature. *BMC Pulm Med*. 2008;8:18.

Airway Issues: Vocal Fold Paralysis

Kenneth W. Altman MD PhD ■ Melin Tan-Geller MD

Description

Vocal fold paralysis after spinal cord injury (SCI) is most commonly due to injury to the recurrent laryngeal nerve (RLN) and often leads to a weak or absent voice and can lead to aspiration.

Etiology/Types

- Congenital
- Traumatic
- Postsurgical
- Neoplastic
- Neuromuscular
- Infectious
- Vascular
- Idiopathic

Epidemiology

- Postsurgical paralysis occurs in approximately 2% to 6% of anterior cervical spine surgeries.
- True paralysis due to endotracheal intubation is rare and likely related to hyperinflation of the cuff.
- Paralysis due to brain stem stroke can occur but is rare.

Pathogenesis

- RLN injury is the most common cause of vocal fold immobility causing true paralysis or paresis.
- Superior laryngeal nerve injury can cause paresis.
- Immobility can occur from scarring, ankylosis, or dislocation of the arytenoids.
- A mass or neoplasm can limit vocal fold movement.

Risk Factors

- Surgery in proximity to RLN
- Endotracheal intubation

Clinical Features

- Unilateral paralysis: weak or breathy voice, change in pitch, weak cough and poor pulmonary secretion management, cough with eating or drinking, aspiration, choking
- Bilateral paralysis: stridor and airway obstruction if adducted position; aphonia and aspiration if abducted position

Natural History

- Postsurgical neuropraxia of the RLN has about a 30% to 50% likelihood of meaningful recovery by 6 months.
- Injuries due to intubation have about a 50% likelihood of recovery at 6 months.
- Complete nerve transactions are unlikely to recover.
- Without treatment, aspiration is the greatest risk.

Diagnosis

Differential diagnosis

- Vocal fold paresis
- Arytenoid dislocation/subluxation or fixation
- Laryngeal scar, web
- Posterior glottic stenosis
- Mass or neoplasm
- Degenerative neuromuscular disorder

History

- Neck or chest surgery
- Prolonged intubation
- Trauma to the neck
- Aspiration
- Choking
- Weak, breathy, or absent voice
- Change in pitch of voice
- Weak cough
- Cough with eating or drinking

Exam

- Asymmetric movement of the vocal folds
- Vocal fold bowing
- Tilting of the posterior larynx
- Incompetent closure of vocal folds on phonation
- Stridor on auscultation
- Aphonia

Testing

- Laryngeal electromyography (EMG) confirms impaired muscular recruitment, and determines presence of polyphasic potentials (reinnervation) or spontaneous activity (denervation).
- Aspiration risk should be assessed with bedside swallow evaluation, flexible endoscopic evaluation of swallowing, or modified Barium swallow.

- An unexplained new-onset vocal paralysis should be assessed with CT neck/fine cuts of larynx with intravenous contrast to evaluate the course of the RLN.

Pitfalls

- Vocal fold immobility due to scar, stenosis, arytenoid dislocation, or ankylosis

Treatment

Medical

- Treatment is focused on improvement of voice quality if aspiration not present.

Exercises

- Voice therapy can offer patients useful compensatory exercises that improve voice quality and improve aspiration protection.

Surgical (for unilateral paralysis)

- Medialization for unilateral paralysis is performed either by injection of material into the vocal fold or with an implant augmenting the vocal fold.
- Reinnervation to the ansa cervicalis, phrenic nerve, preganglionic sympathetic neurons, hypoglossal nerve, and nerve-muscle pedicles are also employed.

Surgical (for bilateral paralysis)

- Tracheotomy may be required if airway patency is jeopardized by adducted bilateral vocal paralysis
- Cordotomy and arytenoidectomy with or without suture lateralization of the vocal fold for bilateral vocal paralysis.

- Unilateral or bilateral augmentation implants or injection for bilateral vocal paresis.

Consults

- Otolaryngology
- Speech and swallow therapy

Complications of treatment

- Airway edema and hematoma are major complications of airway surgery
- Implant migration
- Poor voice

Prognosis

- Reduction of aspiration and improved quality of voice can be attained with appropriate therapy or surgical intervention.

Helpful Hints

- While treatment is indicated immediately upon diagnosis, it is wise to delay irreversible surgery for about 6 months in cases in which recovery of the recurrent laryngeal nerve is a possibility.

Suggested Readings

- Merati AL, Heman-Ackah YD, Abaza M, Altman KW, Sulica L, Belamowicz S. Common movement disorders affecting the larynx: a report from the neurolaryngology committee of the AAO-HNS. *Otolaryngol Head Neck Surg.* 2005;133(5):654–655.
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Autonomic Nervous System Issues: Autonomic Dysreflexia

Jung-Woo Ma MD ■ Thomas N. Bryce MD

Description

Autonomic dysreflexia (AD) is a condition of imbalanced reflex sympathetic discharge in response to noxious stimuli after SCI.

Etiology/Types

- Reactive to noxious stimuli below the level of injury
- Reactive to central nervous system abnormality such as syringomyelia
- Idiopathic

Epidemiology

- All individuals with complete injuries above T6 can have symptoms of AD with a sufficient stimulus
- Occurs in persons with either complete or incomplete SCI
- Symptoms are less common and severe in persons with incomplete SCI
- Rarely occurs in persons with lesions below T6
- Does not occur until spinal shock has resolved

Pathogenesis

- A noxious stimulus activates nociceptors below the level of the lesion setting off a barrage of afferent impulses.
- Sympathetic neurons are activated in the spinal cord below the level of the lesion producing a generalized sympathetic response.
- The generalized sympathetic response generates increased peripheral resistance, circulating blood volume, and an elevation in blood pressure.
- Inhibitory signals generated by brain stem vasomotor centers are unable to descend (when the injury level is at T6 or above) to the splanchnic vascular beds, which could accommodate the increased blood volume.
- Parasympathetic output prevails above the injury level leading to nasal congestion, flushing, and sweating above the level of the injury.
- Vasomotor brain stem reflexes attempt to lower blood pressure by increasing vagal parasympathetic stimulation to the heart, causing bradycardia.

Risk Factors

- Bladder distension (75% to 85%)
- Fecal impaction (13% to 19%)
- Pressure ulcers
- Urinary tract infection (UTI)
- Ingrown toenails
- Menstruation
- Labor and delivery
- Cholecystitis
- Gastric ulcers or gastritis
- Hemorrhoids
- Sexual activity
- Constrictive clothing
- Fractures or other trauma
- Detrusor sphincter dyssynergia
- Syringomyelia

Clinical Features

- Pounding headache
- Hypertension
- Profuse sweating and flushing above the level of injury
- Blurry vision

Natural History

- A noxious enough stimulus can produce AD in almost anyone with a high thoracic or cervical SCI.
- Untreated severe hypertension accompanying AD may lead to retinal hemorrhage, cerebral hemorrhage, seizures, and death.

Diagnosis

Differential diagnosis

- Essential hypertension
- Pheochromocytoma
- Migraine and cluster headaches
- Toxemia of pregnancy

History

- Acute onset of severe headache, blurry vision, nasal congestion, and sweating

Exam

- Sudden rise in blood pressure, generally >20 mm Hg

- Profuse sweating, piloerection, and flushing above the level of lesion
- Bradycardia

Testing

- Blood pressure and pulse monitoring
- Abdominal flat plate to look for impaction
- Urinalysis and urine culture if urinary tract infection is suspected
- MRI of cervical spine if no cause can be found

Pitfalls

- Failure to recognize mild symptoms, especially headache, as AD
- Failure to recognize that baseline blood pressure can be quite low in persons with tetraplegia and resultant increase to a seemingly modest absolute blood pressure may be quite a significant increase

Red Flags

- Chest pain
- New neurologic dysfunction

Treatment

Medical

- Sit patient upright
- Loosen clothing
- Relieve obstruction to drainage of an indwelling urinary catheter
- If no indwelling catheter present then catheterize patient
- If the systolic blood pressure is still ≥ 150 mm Hg, administer rapidly acting and easily reversible antihypertensives, such as topical nitroglycerin ointment
- If the systolic blood pressure is < 150 mm Hg, then the rectum should be manually disimpacted

- Search for other precipitants if symptoms persist
- Prophylactic treatment for patients with recurrent episodes of AD can include alpha blockade or ganglion blockade
- Use of a local anesthetic gel can help prevent AD triggered by digital stimulation or urethral catheterization

Consults

- Physical medicine and rehabilitation
- Neurourology
- Gastroenterology

Complications of treatment

- Hypotension from antihypertensives

Prognosis

- The condition can nearly always be managed successfully if underlying cause can be identified and eliminated.
- Mortality is rare.

Helpful Hints

- Patients should be educated about the clinical findings and treatment for this condition.

Suggested Readings

- Blackmer J. Rehabilitation medicine. 1. Autonomic dysreflexia. *CMAJ*. 2003;169(9):931–935.
- Bryce TN, Ragnarsson KT. Spinal cord injury. In: Braddom RL, ed. *Physical Medicine & Rehabilitation*. 3rd ed. Boston: Saunders Elsevier; 2007:1325–1326.
- Bycroft J, Shergill IS, Choong EAL, Arya N, Shah PJR. Autonomic dysreflexia: a medical emergency. *Postgrad Med J*. 2005;81:232–235.
- McLachlan EM. Diversity of sympathetic vasoconstrictor pathways and their plasticity after spinal cord injury. *Clin Auton Res*. 2007;17:6–12.