Cannabinoid Hyperemesis Syndrome: Rare or Underdiagnosed?
AU ET AL.

Assessment of the Validity of Self-Report as a Measure of Smoking Status in Patients Post-Myocardial Infarction
GRANDI ET AL.
Join the many internists practising in British Columbia (BC), Canada who enjoy a quality of life that is envied around the world. Our physician services team can assist you with licensing and immigration, and match your skills and interests with exciting career opportunities. Visit our website to view current opportunities.

PRACTISE MEDICINE IN BRITISH COLUMBIA, CANADA
Enrich your career. Enhance your quality of life.

Join the many internists practising in British Columbia (BC), Canada who enjoy a quality of life that is envied around the world. Our physician services team can assist you with licensing and immigration, and match your skills and interests with exciting career opportunities. Visit our website to view current opportunities.

healthmatchbc.org

REGISTER TODAY!
healthmatchbc.org

PHYSICIANS
NURSES
ALLIED HEALTH

healthmatchbc.org

FIND A JOB IN BC

Health Match BC is a free health professional recruitment service funded by the Government of British Columbia, Canada.

Toll-Free: 1.877.867.3061 • TEL: +1.604.736.5920 • EMAIL: welcome@healthmatchbc.org

Photos: Picture BC & Tim Swanky
CONTENTS

Message from the Editor-in-Chief/Message du rédacteur en chef
124 Mitch Levine

Clinical Medicine
126 Acute Care SINS: Surgical Insights for the Non-surgeon
Chapter 9: Urology SINS
Adam Kinnaird MD PhD, Adrian Fairey MD MSc, Keith Rourke MD, Rachel G. Khadaroo MD PhD, Peter G. Brindley MD

134 Is ‘Gluten-Free’ Gluten-Free? Celiac Disease in Canada in 2015
Nauzer Forbes MD, Zain Kassam MD, David Morgan MD, MSc

Original Research
137 Assessment of the Validity of Self-Report as a Measure of Smoking Status in Patients Post-Myocardial Infarction
Sonia M. Grandi MSc, André Gervais MD, Lawrence Joseph PhD, Jennifer O’Loughlin PhD, Nauzer Forbes MD, Zain Kassam MD, Mark J. Eisenberg MD MPH

140 Medical Education and Duty Hours: Do We Really Care?
Peter G. Brindley MD

145 On the Shoulders of Giants
HMB

147 Cannabinoid Hyperemesis Syndrome: Rare or Underdiagnosed? Selena Au MD, Ali Rezaie MD

150 Sudden Bilateral Deafness
Florence Morriello MD, Enrico Granieri MD

153 A Forgetful Experience: A Case of Transient Global Amnesia
Kalpa Shah MD PGY2, Chris Sheasgreen MD PGY 3, Ameen Patel MB

156 Thoracic Aortic Dissecting
Aneurysm Presenting with Stroke, Disseminated Intravascular Coagulopathy (DIC), and Gastrointestinal Hemorrhage
Soraya Moghadam MD, Yoshitsugu Nakamura MD, Mackenzie Quantz MD, Raymond Kao MD

159 Rumpel-Leede Phenomenon: A Case Report
Rehman HU, MBBS, FRCPC; Kambo J, MD

Teaching and Learning
147 On the Shoulders of Giants
HMB

140 Medical Education and Duty Hours: Do We Really Care?
Peter G. Brindley MD

142 Resident Duty Hours: A Review
Lindsay Melvin MD, Sophie Corriveau MD, Aiman Alak MD, Ameen Patel MB

About the Cover
Photography by Jean-François Josso. It was taken in Gaspésie National Park of Île-Bonaventure-et-du-Rocher-Percé. Josso says he was fascinated by the hypnotic gaze of this Northern Gannet.

Copyright Jean-François Josso

For Instructions to Authors, please visit www.andrewjohnpublishing.com/pcjim.html
With the recent legalization of marijuana in a number of American states, there have been concerns expressed regarding the potential increase in medical problems associated with marijuana use. In Canada, unauthorized possession of marijuana is still a criminal offence although recreational use is substantial across the country. With the recent changes to the regulation of medical marijuana in Canada, it is anticipated that there will be an additional increase in the number of Canadians using the drug in the future. Further, one might anticipate that the use of medical marijuana would become a chronic treatment for many of the patients seeking this therapy.

In this edition of the *Canadian Journal of General Internal Medicine*, we have published a case report of an adverse effect that can occur with chronic marijuana use. Cannabinoid Hyperemesis Syndrome is an important adverse effect that physicians need to be aware of in the expanding medical marijuana environment. Its importance is not particularly driven by a high frequency of occurrence, but rather by the consequences associated with a failure to diagnose the situation correctly.

Unnecessary investigations and prolonged hospitalizations are two important consequences of missing the diagnosis, and they can result in patient harm and the inefficient use of finite medical resources. Further, a failure to inform the patient of the actions required to curtail the problem will permit the problem to perpetuate. Part of the difficulty in diagnosing this condition is the paradoxical nature of the symptom that is, vomiting, which is not readily associated with marijuana use when marijuana is perceived to be, and is used as, an antiemetic drug.

As internists, we often encounter patients who have proven to be diagnostically difficult to sort out by their primary care physicians. Patients with Cannabinoid Hyperemesis Syndrome can certainly fall into that category with its both chronic and peculiar pattern of occurrence. Making the correct diagnosis during a consultation will serve the patient well and will be an opportunity to inform the referring physician about a medical condition that is only likely to increase in the future.

References
Depuis la légalisation de la marijuana dans certains États américains, plusieurs s’inquiètent de la hausse potentielle des problèmes médicaux découlant de la consommation de marijuana. Au Canada, la possession non autorisée de marijuana constitue toujours un acte criminel quoique l’usage récréatif soit passablement répandu. Au vu des modifications récentes de la réglementation sur la marijuana utilisée à des fins médicales au Canada, l’on prévoit une hausse supplémentaire du nombre de Canadiens consommant du cannabis. De plus, l’on peut s’attendre à ce que, dans nombre de cas d’usage médical de la marijuana, ce traitement devienne chronique.

Dans le présent numéro de La Revue canadienne de médecine interne générale, nous publions un exposé de cas illustrant un effet indésirable de la consommation chronique de marijuana. L’hyperémèse cannabique ou syndrome cannabinoïde est un effet indésirable important que le médecin a intérêt à connaître dans l’environnement en expansion de l’usage médical de la marijuana. Son importance ne tient pas tant à son occurrence fréquente qu’aux conséquences de l’errance diagnostique, notamment les examens inutiles et la longue hospitalisation en raison d’un diagnostic difficile à établir, qui peuvent entraîner des effets néfastes chez le patient et l’utilisation inefficace de ressources médicales limitées. De plus, le fait de ne pas informer le patient des actions nécessaires pour régler le problème aura pour effet de perpétuer ledit problème. La difficulté que pose le diagnostic du syndrome réside en partie dans la nature paradoxale du symptôme, soit les vomissements, qui ne sont pas associés habituellement à l’incidence augmentera fort probablement à l’avenir.

En tant qu’internistes, il nous arrive souvent de nous pencher sur des cas difficiles à diagnostiquer, qui ont laissé perplexes le médecin de premier recours. L’hyperémèse cannabique ou syndrome cannabinoïde peut facilement se ranger dans cette catégorie en raison de son schéma d’occurrence à la fois chronique et singulier. Poser le diagnostic exact à la consultation sera bénéfique pour le patient et aussi l’occasion de renseigner le médecin traitant à propos d’une affection médicale dont l’incidence augmentera fort probablement à l’avenir.

Mitch Levine

Références
Acute Care SINS: Surgical Insights for the Non-surgeon
Chapter 9: Urology SINS

Adam Kinnaird MD PhD, Adrian Fairey MD MSc, Keith Rourke MD, Rachel G. Khadaroo MD PhD, Peter G. Brindley MD

About the Authors
Adam Kinnaird is a resident in urology; Adrian Fairey is an assistant professor of surgery; Rachel Khadaroo is an assistant professor of surgery and a member of the Divisions of Critical Care Medicine and General Surgery; Keith Rourke is an associate professor of surgery; and Peter Brindley is a professor of critical care medicine, all at the University of Alberta, in Edmonton, Alberta. Correspondence may be directed to peter.brindley@albertahealthservices.ca.

Summary
“Surgical Insights for the Non-surgeon,” or SINS, is composed of several short chapters intended to cover fundamental surgical knowledge for non-surgeons. The authors focus on surgical pearls, operative insights, and applied anatomy. In Chapter 9 of this series, the authors address the genitourinary system and Urology SINS.

Résumé

He who works with his hands is a laborer; he who works with his head and hands is a craftsman.
— Francis of Assisi

Anatomy
Urology is plumbing! Starting from the top of the genitourinary tract, urine is first concentrated in the collecting ducts of the renal medulla. From there, urine flows into the renal collecting system by passing from collecting duct to minor calyx, to major calyx, to renal pelvis, down the ureter, and into the bladder for storage. The fornix of a minor calyx can rupture (almost like a pressure-release valve) following severe obstruction (typically a ureteric stone). However, calyceal rupture is not an indication for emergent surgery.

The ureters run along the psoas muscle, cross over the iliac vessels at their bifurcation, and enter the posterior aspect of the bladder on opposite ends of the trigone muscle. The ureter is not the only tubular structure in the pelvis; the gonadal veins and the appendix also share that distinction. The bladder is an extraperitoneal muscular reservoir tucked behind the pubic symphysis. Continence relies upon the synergic relaxation of detrusor muscles and the simultaneous contraction of the bladder neck and pelvic floor muscles. Typically, urine does not reflux up a ureter, owing to a one-way flap valve created.
as the ureteral orifice is compressed while the bladder fills. The most common places for ureteric calculi are the ureteropelvic junction, the crossing of the iliac vessels, and the ureterovesical junction (UVJ). The UVJ is the most distal portion of the ureter, and it also happens to be the narrowest. The UVJ is where most stones get “stuck.”

The base of the bladder opens into the urethra, like the drain of a sink. When empty, the bladder is about the size and shape of a pear; when full, the typical adult bladder’s capacity is approximately 500 cc. A child’s bladder capacity is estimated by the following formula:

\[(\text{age} + 2) \times 30 \text{ cc}\]

For example, the bladder capacity of a 4-year-old is determined as follows:

\[(4 + 2) \times 30 \text{ cc} = 180 \text{ cc}\]

The bladder’s inside is covered by a watertight mucosal surface made up of transitional epithelial cells. These are the cells that most commonly transform into a bladder cancer.

The urethra has five anatomical sections: prostatic, membranous, bulbar, penile, and fossa navicularis. The prostate is a walnut-sized, conically shaped gland with four lobes. It is located between the bladder and the penis, just in front of the rectum (conveniently for digital palpation). It serves as a merging lane for urine and semen, which the latter enters via the ejaculatory ducts in the prostate. The urethra runs through the centre of the prostate, which is why benign prostatic hyperplasia (BPH) results in urinary obstruction, retention, and nocturnal bathroom trips. For a variety of non-urologic reasons, however, one or two episodes of nocturia per night is normal for many people.

Because it passes through the penis, the male urethra is longer (20 cm) than the female urethra (4 cm); the shorter separation from skin flora in females is why women are more likely to experience urinary tract infections. Urethral injuries associated with pelvic fractures occur at the junction of the membranous and bulbar urethra (and in approximately 5% of all pelvic fractures). The fossa navicularis, which is within 1 cm of the urethral meatus, is the narrowest part of the urethra. Therefore, if a catheter can pass the first 2 cm but then stops at any point, this is not due to a catheter of the wrong size; there is a pathologic reason (false passage, large prostate, urethral stricture, foreign object, etc.).

The remainder of this article discusses common reasons for urology consults.

**Hematuria**
- Divided into microscopic versus gross (i.e., visible) hematuria
- In both cases, blood may come from any site along the genitourinary tract
- Causes include trauma, tumours (prostate, bladder, renal), BPH, stones, radiation cystitis
- Generally, hematuria is a urologic malignancy until proven otherwise
- The urinary tract is divided into two parts for evaluation
  - The upper tract (kidneys, ureters)
  - The lower tract (bladder, prostate, urethra)

**Microscopic Hematuria**
- Defined as >3 red blood cells per high-powered field
- Work up completely in patients >40 years old
  - Complete blood count (CBC)
  - Serum creatinine (Cr)
  - Urine microscopy and culture
  - Urine cytology
  - Cystoscopy
  - Upper-tract imaging (ultrasound kidneys, ureters, bladder)

**Gross Hematuria**
- Defined as bloody urine visible to the naked eye
- Work up for patients of all ages
  - CBC
  - Serum Cr
  - Urine culture
  - Cystoscopy
  - Upper-tract imaging (contrast-enhanced computed tomography of abdomen/pelvis)
- Gross hematuria can result in numerous blood clots in the bladder, which in turn can cause urinary retention (called “clot retention”)
- The best way to manage clot retention is to remove all blood clots from the bladder
  - Manually irrigate a three-way Foley catheter with normal saline
    - To decompress the bladder and remove clots
    - See below for technical instructions
  - Next, follow with continuous bladder irrigation (CBI) through the irrigation (large) channel of the three-way catheter
  - Titrate the CBI flow to keep the urine clear
  - If blockage reoccurs, manually irrigate again
- Remember, only manual irrigation clears bladder clots; CBI only prevents them from re-forming

**Manual Irrigation Technique**
- Equipment: one 60 cc Toomey syringe, one irrigation kit, 1 L normal saline (NS)
• Technique
- Place a large (22–24 French) three-way Foley catheter
- Instill 60 cc of NS through the middle (large) port, using a Toomey syringe
- Follow immediately with vigorous aspiration through the same port
- Repeat until 1 L of NS is used or the urine is clear
• If you can instill fluid but cannot aspirate
  - The catheter is against the bladder wall or malpositioned (i.e., not in the bladder!)
  - If so, instill 120 cc into the bladder prior to aspiration
  - If you still cannot aspirate, the catheter is likely malpositioned
    - So stop! Deflate the balloon and remove the catheter
    - Then call urology

**Troubleshooting the Difficult Urethral Catheter**

**General Tips**

• Urinary catheters are measured in “French,” abbreviated as “F”
  - “French” refers to the French scale, or French gauge
  - French is a measure of the external circumference of a catheter in millimetres (for example, a 16F catheter has an external circumference of 16 mm)
• Catheters range from 8F (pediatric) to 24F
• The typical adult catheter measures 14F to 18F
• Larger catheters (20F or greater) are used to irrigate a bladder or ensure that a bladder is well decompressed
• Look for urine return before inflating the catheter balloon; if you do not see urine draining, you may still be in the urethra
• If no urine returns but you believe that you are in the bladder (i.e., no urine due to renal failure or septic shock),
  - Aspirate the drainage port, or try manual irrigation (as above)
  - BUT, then only inflate the balloon if you see fluid return with aspiration
  - That is don’t inflate the balloon if fluid does not return with aspiration
• You do not need to test balloon inflation prior to insertion
  - This only makes the tip larger and more uncomfortable when inserted
  - Also, catheters are designed to be inflated only once
• Do not attach the sterile water syringe until the catheter is inserted; doing so only adds a useless counterweight that may pull the catheter out inadvertently
• Apply at least one tube of sterile xylocaine jelly to the urethra before insertion; if unavailable, ensure the catheter is at least well lubricated
• When inserting a catheter, make sure to “pull the penis to the sky” to straighten the angle between the penile urethra and bulb urethra
• Always return the foreskin back over the glans to prevent paraphimosis (see below)
• Bacterial colonization of an indwelling catheter is approximately 10% per day
  - Expect most patients to have (at least) asymptomatic bacteriuria by 1 week
  - Remove the catheter when no longer required

**Difficult Catheters**

• Difficulty with placement may be due to an enlarged prostate, urethral stricture, false passage, phimosis, bladder neck contracture, or hypospadias
  - A “false passage” is a tear in the urethral epithelium
    - Due to failed (usually multiple) attempts
    - The catheter enters under the flap
      - Causing a false passage and creating a one-way valve
      - Therefore you can push fluid in, but cannot aspirate fluid out
• Catheters may not pass due to an enlarged prostate or a steep angle between an enlarged prostate and the bladder
  - In such a case, the catheter may pass but not all the way to the bladder
  - Try a larger (18F or 20F) catheter to push the prostate lobes “aside,” or try a coudé-tip catheter (see below)
  - If you suspect a false passage, use a coudé-tip catheter
• A urethral stricture is scar tissue or fibrosis that narrows the urethra
  - Suspect a urethral stricture if the catheter passes initially but not as far as the prostate
  - Try a smaller (12F or 14F) catheter to try to “sneak” through the small urethral lumen
• A bladder neck contracture is scarring of the prostatic urethra, usually occurring after prostate surgery
  - When placing a urinary catheter, the non-urologist should
    - Try a regular 16F or 18F catheter with lots of lubrication and while keeping the penis stretched
    - If this fails, try a larger (18F or 20F) coudé-tip catheter to bypass a false passage or to push the prostate out of the way
    - If the above two steps fail, try a smaller catheter (12F or 14F)
      - A stricture could be the culprit
      - A smaller catheter may “negotiate” the narrowed urethra
  - If the above three steps fail, call urology
• Sometimes a catheter that doesn’t drain urine may be in the bladder, but the patient is anuric
  - When in doubt, irrigate the catheter
  - Then obtain a serum creatinine level, to check renal function
  - Also, do an ultrasound examination of the bladder to confirm that urine is present
Coudé-Tip Catheters
• A catheter (often coloured red) that is bent like a hockey stick at the end
• This catheter mimics the upward curve at the junction of the anterior and posterior urethra
• This catheter can aid passage through the prostatic urethra
• False passages usually occur posteriorly, and the coudé curve can bypass these; the curve causes the tip to pass over (but not into) the false passage
• The curved tip should always be pointing up
• Once the catheter is inside the urethra, you can no longer see the tip

Three-Way Catheters
• Colloquially called CBI catheters or “hematuria catheters”
• Sizes are 20F to 24F; typically, bigger = better for draining clots
• If indicated (i.e., gross hematuria), the technique is the same as with a usual catheter
  - Manually irrigate through the middle (large) port, as described above (Figure 2)
  - Connect inflow port (on the side opposite the balloon port) to a 4 L bag of NS
  - Proceed with CBI (as above)
  - Note: You will no longer be able to accurately track urine output while CBI is running

Suprapubic Catheters
• Placed directly through the abdominal wall into the bladder
• Typically used when a urethral catheter cannot be placed (i.e., a very difficult urethral stricture) or in some patients who require long-term (>3 months) catheterization
• Materials: local anaesthetic (with epinephrine), 22-gauge “seeker” needle, 10 cc syringe, suprapubic catheter kit, urometer
• Placement technique
  1. Prepare a wide sterile field
     - Top: level with the umbilicus
     - Bottom: below the pubic symphysis
     - Sides: lateral aspects of the rectus sheath
  2. Landmark two fingerbreadths in midline and above the pubic symphysis
  3. Inject local anaesthetic into skin, subcutaneous tissues, and down to fascia
  4. Using a seeker needle, advance into the anaesthetized tract until urine is aspirated
  5. Make a 2-cm stab incision down to fascia
  6. Nick the fascia with the blade
  7. Hold the suprapubic trochar so that your index finger acts as a guard
  8. Twist the trochar through the fascia and into the bladder
  9. Once urine flows, advance 1 cm more
  10. Detach the trochar, leaving a hollow plastic sheath in situ
  11. Insert a 16F Foley catheter through the plastic sheath into the bladder
  12. Inflate the Foley balloon prior to removing the plastic sheath
  13. Withdraw the sheath, but do not to pull the Foley with it
  14. Suture the skin with interrupted 3-0 Prolene superior to the Foley catheter
  15. Using this same closing stitch, secure the catheter as you would any drain
• Maintenance: Leave the catheter in for 4 weeks, prior to exchanging, in order for the tract to mature

Drainage Tubes
• Obstruction or stasis of urine in the collecting systems can cause renal failure or infection
• Therefore, urinary drainage must be maintained
• This is done via hollow plastic tubes
  - An enlarged prostate requires a urethral catheter (as above)
  - An infected ureteric calculus requires a ureteral (ureteric) stent or nephrostomy tube
  - A ureteric injury requires a nephrostomy tube

Ureteral Stents
• Commonly called “double-J” stents
• Usually 24–26 cm long, 6F–8F hollow plastic tubes
• They create a connection between the renal pelvis and the bladder, bypassing any obstruction along the length of the ureter
• At both ends, the stent curls in a pigtail (to hold it in place)
• Stents can be introduced in both directions
  - Retrograde (via cystoscopy [i.e., from the bladder up])
  - Antegrade (percutaneous [i.e., from the kidney down])
• Regardless of the direction, they are inserted over a wire, with a Seldinger-like technique
• Important to note that stenting rarely treats the underlying cause; rather, it temporarily alleviates the obstruction
• Postinsertion abdominal radiography is used to determine placement
• Proper placement is when the lower pigtail is in the bladder and the upper pigtail curls in the kidney (Figure 3)
• Note: If a patient presents with a presumed pyelonephritis and does not respond to treatment after 48 hours
  - Obtain urgent renal imaging to rule out an obstructing ureteral stone
  - This requires urgent decompression (i.e., stent) because acute obstructing ureteral calculus plus concurrent pyelonephritis is life-threatening

**Nephrostomy Tubes**
• A percutaneous urinary diversion
• Connects the renal pelvis to a bag on the patient’s back
• Used to emergently relieve obstruction and when a retrograde ureteric stent cannot be placed
• In Europe, nephrostomy tubes are the first-line treatment for infected ureteral stones; in North America, it is usually retrograde stenting (see above)
  - This indicates that stenting and nephrostomy tubes are merely two ways to decompress the genitourinary system
  - The most important points are that decompression is required and that either method works
• Nephrostomy tubes can be used prior to placing antegrade ureteric stents; once antegrade stents are placed, the nephrostomy tubes are typically removed within 24 hours

**Urinary Diversions**
• After cystectomy (bladder removal), restoration of urinary flow is needed
• To accomplish this, the ureters can be diverted to one of the following
  - An ostomy made of a 10–15 cm isolated segment of ileum (such as an ileal conduit)
  - A new intracorporal storage container made up of 60 cm of ileum (aka a neobladder)

**Ileal Conduit**
• A cutaneous “incontinent” urinary diversion
• Made by removing the ureters from the bladder and reconnecting them to a loop of 10–15 cm of detached bowel (also called an intentional ureteroenteric fistula brought out to the skin)
• Jejunum or colon can be used, but the ileum is simplest and most common
• The end of the bowel is brought through a stoma to the outside abdominal wall
• Urine drains continually into an ostomy bag
• Resorption of urine can occur via the bowel segment; this can cause hyperchloremic/nonanion-gap metabolic acidosis
• Postoperatively, patients have temporary stents in each ureter
  - This maintains patency and allows healing
  - These stents are visible (i.e., they stick out of the stoma)
• The duration of ureteric stenting is variable but typically ranges from 1 to 4 weeks
• The most common long-term complication is acute pyelonephritis, which occurs in approximately 18% of patients
Neobladders
• An orthotopic (i.e., grafted into the natural position) urinary diversion
• A new urinary reservoir is created and is connected with the native urethra
• The urethral sphincter is preserved; therefore, continence is maintained in 80–90% of cases
• A neobladder is made from ~60–75 cm of ileum detubularized and folded into a sphere
• Ureters are anastomosed to the neobladder and stented for 1–4 weeks (as above)
• Because they end in the neobladder, these stents may not be visible
• The patient will have a urethral catheter immediately post-operation
• Neobladder irrigation is critical to maintain function, due to the buildup of mucus secreted by the ileum
• While the patient is in hospital, irrigation should be done four times daily via the indwelling urethral catheter, using 60 cc of NS
• Perioperative complications are 20–30% higher as compared to ileal conduits, but neobladders improve long-term quality of life
• Neobladder leaks require percutaneous drainage and nephrostomy tubes
• Long-term complications include
  - Vitamin B12 deficiency (due to the large amount of terminal ileum used)
  - Bladder stones (both symptomatic and asymptomatic)

Unwanted Erection
Priapism
• Defined as (1) an erection lasting more than 4 hours beyond orgasm or (2) an erection unrelated to sexual stimulation
• An emergency that, if untreated, leads to corporeal fibrosis, permanent erectile dysfunction, and (in extreme cases) penile necrosis
• Subdivided into (1) ischemic, (2) stuttering, and (3) non-ischemic
  - Ischemic priapism
    o Analogous to compartment syndrome
    o Occlusion of venous outflow that subsequently prevents arterial inflow
    o Common causes include erectile dysfunction medications, hematologic dyscrasias, neurogenic injury, and recreational drugs
  - Stuttering priapism
  - Occurs most often in sickle cell disease
  - Non-ischemic priapism
    o Also known as high-flow priapism
    o Caused by straddle trauma and laceration of a cavernosal artery
    o Painless due to good vascular supply; therefore, low risk of permanent injury
• A corporal blood gas (i.e., a penile blood gas) is sometimes required to differentiate between ischemic and non-ischemic priapism
• Non-ischemic priapism has a blood gas profile most similar to arterial blood, whereas ischemic priapism will have a partial oxygen pressure (PO2) < 30 and pH < 7.25

Corporal Blood Gas Technique
• Insert an 18-gauge needle at either the 3- or 9-o’clock position into the corpus cavernosum, just distal to the penoscrotal junction
  - This position avoids nerves running along the dorsum of the shaft
  - Pinch the penile base between two fingers, and aspirate blood from the shaft
• Typical corporal blood gas findings in ischemic priapism
  - PO2 < 30
  - Partial carbon dioxide pressure (PCO2) > 60
  - pH < 7.25

Treatment of Ischemic Priapism
• Use the same initial technique used to assess corporal blood gas
• Attach an 18-gauge needle to a 10 cc syringe; aspirate the shaft until soft while pinching off the blood supply at the base of the penis
• With the needle still in place
  - Irrigate (with cold NS)
  - Use the same volume of NS as the volume of blood that was removed
  - Repeat three times
• If priapism persists, inject intracavernosal phenylephrine (see below)
  - Dilute phenylephrine to 200 µg/mL
    o Usual phenylephrine concentration is 1 mg/mL (1 mL in a vial)
    o Therefore, dilute with 4 mL cold NS (to create 5 mL in total)
- After aspirating the shaft (as above), inject 1 mL of this solution into the shaft
  - Release your grip at the base of the penis to allow blood to refill the penis
  - Repeat this every 5 minutes, up to a maximum of 5 times
  - If this fails, a surgical shunt is required
    o A shunt creates a vascular bypass between the corpora cavernosa and the glans penis
    o The shunt is achieved via bilateral transglanular incisions

**Penile Prostheses**
- Surgically implanted devices are used as last-line of treatment of erectile dysfunction
- Inserted into the corpora cavernosa
- Malfunction (or simple misunderstanding) may lead to misdiagnosed priapism
- Two major classes: malleable and inflatable
  - Malleable prostheses
    o Semirigid tubes: bent up for coitus (and bent down otherwise)
    o Penis is constantly rigid
  - Inflatable prostheses
    o Multicomponent devices
    o Two tubes in the penis, a scrotal pump, and an abdominal fluid reservoir
    o The scrotal pump permits on-demand erections and detumescence

**Penile Fracture**
- As the corpora cavernosa fill during erection, the blood is held by a strong collagenous outer layer called the tunica albuginea (which can withstand intracavernous pressures of up to 1,500 mm Hg!)
- A penile fracture represents rupture of the tunica albuginea
- Usually caused by “buckling”: most commonly, an erect penis slips out of the partner and hits the perineum or pubis, causing a sudden bend
- Fracture occurs laterally, where the tunica albuginea is thinnest
- Patients describe a cracking sound, followed by pain, and rapid detumescence
- The appearance is described as an “eggplant” deformity (swelling, bruising, dark discolouration)
- Approximately 20% of the time, the urethra is simultaneously injured, causing profuse bleeding through the urethral meatus
- Treat with prompt surgery (within 12 hours)
  - Hematoma evacuation and primary repair of the tunica
  - Timely surgery reduces erectile dysfunction and preserves penile curvature

**Surgical Pearl: Performing a Dorsal Penile Nerve Block**
- Indications: reduction of paraphimosis, dorsal slit for phimosis, circumcision
- Materials: 0.5% bupivacaine (without epinephrine), a 22-gauge needle, a 10-cc syringe
- Technique (Figure 4):
  1. Clean around the superior base of the penis and the pubic symphysis
  2. Locate the correct spot
    o Midline, inferior to the pubic symphysis and superior to the penile base
  3. Inject bupivacaine as you plunge the needle deeper
    o Final depth is the posterior inferior aspect of the pubic symphysis
  4. Without removing the needle, direct it to either side of the midline while injecting bupivacaine to a similar depth

Note: the dorsal penile sensory nerves run on either side of midline. This technique captures both of them with only one poke.

**Phimosis**
- The inability to retract the foreskin for cleaning, voiding, or sexual function
- Usually from a fibrotic band of tissue
- May result in recurrent infections (balanitis)
- May result in pain or tearing or bleeding with sexual intercourse
- Treatment may be conservative (e.g., topical corticosteroids [0.05% clobetasol] used in the pediatric population)
- If conservative measures fail, then surgery (dorsal slit or circumcision) is required
  - Dorsal slit
  - Circumcision
- Foreskin is incised dorsally through the area of phimosis
- Usually done under local anaesthetic
  - Circumcision
- Complete removal of the foreskin
- Completely treats phimosis
- Some men can develop penile cancer associated with phimosis
  - Phimosis increases penile cancer risk by five times
  - Usually squamous cell carcinoma
  - Usually associated with ulceration or induration
  - There may also be inguinal lymphadenopathy (from concurrent infection or metastases)
- For any penile ulcer persisting more than 3 months, a biopsy should be performed to rule out squamous cell carcinoma
**Paraphimosis**
- Occurs when the foreskin is trapped behind the glans penis and cannot be reduced (i.e., cannot be pulled back to its normal position covering the glans)
- May occur in patients with a pre-existing phimosis
- May occur iatrogenically, when a health care professional forgets to place the foreskin back after a catheter insertion or physical examination
- If it persists for hours
  - It results in pain, penile edema, or even gangrene
  - Therefore, it is a medical emergency
- Treatment includes compressing the glans and edema while pulling the foreskin back to its normal position; usually done with lubricant, cold compression, and local anaesthesia
- If above measures fail, an emergency dorsal slit or circumcision is required

**Miscellaneous Urologic Clinical Pearls**
- Not all flank pain is caused by a “kidney stone”
  - Don’t forget to think of ruptured abdominal aneurysm, diverticulitis, and perforated viscera
- Kidney stones cause pain by causing ureteral obstruction, so look for hydronephrosis on imaging
- Not all pyuria is a urinary tract infection (UTI)
  - Urinalysis requires clinical context
  - Both pyuria/bacteruria and clinical symptoms are needed for the condition to be a true UTI
- Not all scrotal swelling is a urologic problem; the scrotum also swells from generalized disease (i.e., congestive heart failure, anasarca)
- Renal ultrasonography usually overestimates the true size of a kidney stone
  - Up to two-fold size miscalculations
  - Also can completely miss ureteral calculi
- The ureter is not the only tubular structure in the right lower quadrant; do not forget the gonadals, appendix, etc.
- Maintain high clinical suspicion for spinal cord compression (cauda equina syndrome) in metastatic prostate cancer with new back pain, especially with lower-extremity weakness and/or urinary retention

**Reference**
Is ‘Gluten-Free’ Gluten-Free?
Celiac Disease in Canada in 2015

Nauzer Forbes MD, Zain Kassam MD, David Morgan MD MSc

About the Authors
Nauzer Forbes is a 5th-year gastroenterology resident completing his training at McMaster University in Hamilton, Ontario. Zain Kassam is pursuing his master’s of public health at Harvard University in Cambridge, MA, USA, having completed his gastroenterology training at McMaster University. David Morgan is a faculty member of the Division of Gastroenterology at McMaster University and is a past president of the Canadian Association of Gastroenterology. Correspondence may be directed to nauzer.forbes@gmail.com.

Summary
Our understanding of celiac disease (CD) has improved dramatically in recent years. Once considered a rare childhood malabsorption syndrome, we now recognize CD as a complex enteropathy mediated by immune, genetic, and environmental factors. CD affects a large proportion of the Canadian population, and its disease burden is substantial in terms of proven reduction in quality of life. Symptom recognition and referral to a gastroenterologist for diagnosis are crucial. As CD is caused by intolerance to gluten, the only effective treatment is strict gluten avoidance. Canadian food product guidelines have only recently become sufficiently rigid, incorporating newer evidence that suggests lower limits of gluten tolerability in this patient population than previously thought.

Résumé
Dans les dernières années, nous en avons appris énormément sur la maladie cœliaque. Il fut un temps où nous pensions qu’il s’agissait d’un syndrome de malabsorption infantile rare, mais nous savons désormais que la maladie cœliaque est une entéropathie complexe où entrent en jeu des facteurs immunitaires, génétiques et environnementaux. La maladie touche une grande proportion de la population canadienne et a des répercussions considérables sur la qualité de vie. La détection des symptômes et l’aiguillage du patient en gastroentérologie en vue de l’établissement du diagnostic sont des éléments cruciaux de la prise en charge. Étant donné que la maladie provient d’une intolérance au gluten, le régime alimentaire strict sans gluten constitue le seul traitement efficace. Ce n’est que récemment que les lignes directrices canadiennes sur les produits alimentaires ont été resserrées à la lumière de nouvelles données probantes indiquant que la tolérabilité au gluten dans ce groupe de patients est moindre que ce que l’on pensait auparavant.

Background and Pathogenesis
Gluten, a composite protein found in wheat (as well as in the related species barley and rye), is responsible for the histopathologic and clinical changes observed in celiac disease. The degree to which oats may be contaminated with gluten and whether they are safe for consumption by CD patients are issues currently being studied.1–3 At any rate, a diet intentionally lacking in gluten — a gluten-free diet — has become established as the standard therapy for CD patients; adhering to a gluten-free diet has consistently been shown to induce remission in this population.4 The exact pathogenesis of CD is not fully understood, but there appears to be an interplay between immune, genetic, and environmental factors.

Canadian Epidemiology
CD continues to be under-diagnosed, yet prevalence is nevertheless high, at approximately 1% of the population.5,6 The national case load, according to the Canadian Digestive Health Foundation, lies between 110,000 and 330,000.7 This range represents the disparity between the population currently labelled with CD (and managed accordingly) and the proportion that remains undiagnosed. Rates of CD have roughly doubled in the Western world in the last 25 years. This comes primarily as a result of heightened clinical suspicion and improvements in diagnosis.
Clinical Presentation and Disease Burden
The clinical spectrum of CD is vast. It comprises gastrointestinal symptoms of varying severity (diarrhea, bloating, abdominal pain, constipation, and weight loss, among others) as well as several extraluminal presentations. These include anemia, dermatitis herpetiformis (pruritic papulovesicular eruptions most commonly found on extensor surfaces), arthralgia, dysmenorrhea, neuropathy, and osteopenia, among others. The most severe process connected with untreated CD is enteropathy-associated T-cell lymphoma (EATL). There are also well-known associations between CD and endocrine conditions, such as insulin-dependent diabetes, thyroid dysfunction, and adrenal insufficiency. As with other immune conditions, there appear to be higher rates of CD in the female population, although this may partially be due to a higher incidence of osteoporosis and anemia in this population, which leads to increased diagnosis of CD. There should be increased clinical suspicion for patients with a documented family history of CD.

There is a substantial reduction in quality of life associated with a diagnosis of celiac disease. Observing a gluten-free regimen is challenging by any account, both for economic and personal reasons. In a recently published cohort study, 73% of CD patients avoided dining outside of their homes at least some of the time, while 78% reported feeling left out of social functions at least some of the time. Strikingly, 81% stated they had difficulty purchasing gluten-free foods at least some of the time. Perhaps most importantly, a staggering 93% of CD patients described at least some difficulty determining whether a particular food was truly gluten-free. Thus one can imagine that CD takes its toll on patients not only via direct symptoms, but also indirectly on quality of life. The same study demonstrated that 47% of CD sufferers feel depressed at least some of the time as a result of their disease. Meanwhile, 53% described alterations in their social activity because of CD, and only 37% described feeling energetic all or most of the time.

Diagnosis and Management
Diagnosing CD can be challenging by virtue of its broad clinical presentation. Not all CD patients will present with gastrointestinal symptoms, and there are patients who have histopathologic changes and mild to absent symptoms, leading to under-diagnosis. The gold standard of diagnosis remains histologic sampling via small bowel biopsy. However, there is a role for serologic testing in patients in whom there is appropriate clinical suspicion of CD. A recent well-designed systematic review evaluated the properties of blood tests readily available to primary care practitioners. IgA anti-tissue transglutaminase (IgA-tTG) was shown to have a pooled sensitivity of 89% and a specificity of 98%. A second serological marker, IgA anti-endomysial antibodies (EmA), had a pooled sensitivity of 90% with a specificity of 99%. Other serology and clinical symptoms were found to be less helpful in establishing diagnosis.

CD serology is expensive and often comes at personal cost to the patient, depending on the laboratory. Referral to a gastroenterologist should be made if the diagnosis is being considered. IgA deficiency can affect test results, and therefore, quantitative immunoglobulin measurement should also be performed for patients in whom clinical suspicion exists. As mentioned, endoscopic biopsy of the small bowel, coupled with clinical and histologic remission on a gluten-free diet, remain the definitive diagnostic parameters. A gluten-free diet is the only means of achieving remission in CD, and compliance is difficult due to the socioeconomic and personal issues listed above. A recent Canadian study found that gluten-free products, on average, were 242% more expensive than their equivalent gluten-containing counterparts.

Defining a Safe Gluten Threshold in 2015
Importantly, the amount of daily gluten intake that CD patients are able to tolerate varies considerably within the population. The so-called gluten threshold, or lower limit of daily exposure that results in pathology and/or symptoms, therefore becomes important to establish. The gluten threshold is a patient-specific value that represents the maximum allowable daily consumption of the protein and is generally represented by the unit mg per day (gluten content of specific foodstuffs is denoted by parts per million [ppm or mg/kg], a value that can then be translated into mg/day by approximating the average daily intake of CD patients). There is an area of research concerned with the subset of patients for whom even a so-called “gluten-free diet” may be contaminated with enough gluten to cause symptoms or histopathologic change.

Several studies have sought to define a safe gluten threshold for CD patients; these have been summarized in two recent systematic reviews. Unfortunately, these studies are heterogeneous in terms of study design, patient age, exposures, measurement techniques, and outcomes. These factors prevented the data from being meta-analyzed in any meaningful way. Despite this, best current evidence suggests that consumption of even small amounts of gluten (between 10 and 50 mg daily) is sufficient to reproduce symptoms as well as induce villous change in most CD patients. These thresholds are primarily driven by arguably the most robust study available, a randomized trial performed in 2007. If one
uses an average consumption of gluten-free foods of 300–500 g per day for CD patients (a typical range in most European and North American countries), one calculates an allowable upper limit for gluten in gluten-free foods of 20 ppm (or mg/kg). The method that has been studied and validated most extensively in gluten quantification is the enzyme-linked immunosorbent assay (ELISA), several subtypes of which exist.16

Despite these data, industrial and governmental standards for gluten content vary substantially among countries. In 2008, the World Health Organization (WHO) and Food and Agriculture Organization (FAO) offered an updated version of the Codex Alimentarius, proposing that 20 ppm should be the maximum allowable ratio of gluten for a product to be labelled “gluten-free.”17 Up until recently, however, the Canadian Celiac Association (CCA) stated simply that “gluten-free in Canada means that the food does not contain wheat, spelt, kamut, rye, barley, oats, or triticale, or any parts thereof.”18

Fortunately, 2012 marked the start of a new era of food labelling in Canada, with important changes made to national labelling regulations. As a result, “labels for all food products sold in Canada will have to carry clear identification of the priority allergens, gluten, and added sulphites at a level greater than 10 ppm.”18 Health Canada has stated that “industry has been given 18 months to implement the new allergen labelling regulations.” (from August 2012).19 This legislation is crucial in providing confidence to consumers.20

Conclusion

There is substantial disease burden associated with CD. Primary care physicians and specialists alike must maintain a high degree of clinical suspicion for this disease and employ early referral to a gastroenterologist for formal diagnosis. Adhering to a strict gluten-free diet is the only known treatment for CD, and historically, this way of life has been difficult, as well as potentially misleading, for consumers. Armed with new labelling directives, the GFCP initiative, expanding arrays of gluten-free products, and advanced techniques in diagnosis, Canadians with CD should now be recognized and protected, as they should. As Canadians in 2015, we proudly lead by example in being able to accurately label our gluten-free products “gluten-free.”

References

Assessment of the Validity of Self-Report as a Measure of Smoking Status in Patients Post-Myocardial Infarction

Sonia M. Grandi  MSc, André Gervais MD, Lawrence Joseph PhD, Jennifer O’Loughlin PhD, Gilles Paradis MD MSc, Louise Pilote MD PhD, Stéphane Rinfret, MD MSc, Mark J. Eisenberg MD MPH

Summary
Self-report is the standard method for assessment of smoking status in the outpatient setting for myocardial infarction (MI) patients. However, the validity of self-report in this patient population has not been previously investigated. Using data from a double-blind, placebo-controlled, randomized trial we examined the validity of self-report for assessment of smoking status in an outpatient setting for MI patients. Smoking was assessed by self-report and biochemical validation by expired carbon monoxide (CO). Abstinence was defined by a self-report of no cigarettes smoked in the past week and a CO level of less than or equal to 10 parts per million (ppm). At 12 months, number of cigarettes smoked was positively correlated with CO level ($r = 0.70$). Results show that biochemical validation by CO does not substantially increase the likelihood of detecting smokers in MI patients. However, it may discourage patients from denying their smoking status and therefore should be considered for routine assessment of smoking status in the outpatient cardiac setting.

Current clinical guidelines for secondary prevention in myocardial infarction (MI) patients recommend routine assessment of smoking status.1 In clinical trials, biochemical validation in conjunction with self-report is the conventional method for assessment of smoking status.2-4 In the outpatient cardiac setting, self-report alone is used for routine assessment of smoking status.

Résumé
En consultation externe, l’on évalue habituellement le tabagisme selon les dires du patient, et il en va ainsi pour le patient ayant subi un infarctus du myocarde (IM). La validité de cette méthode (autoévaluation) dans ce groupe de patients n’a pas été étudiée. Nous reprenons les données d’un essai clinique randomisé, comparatif avec placebo et à double insu pour examiner la validité de l’autoévaluation dans la détermination du tabagisme en consultation externe chez des patients ayant subi un IM. Le tabagisme est évalué selon les dires du patient.
Methods
Data from a double-blind, placebo-controlled, randomized trial examining the efficacy of bupropion following an acute MI was used to evaluate the validity of self-report for assessment of smoking status. The full details of the study have been described elsewhere. Briefly, patients had to have smoked 10 or more cigarettes per day over the past year, be aged 18 years and over, and be motivated to quit smoking. A total of 392 patients hospitalized for an acute MI were randomized to receive bupropion or placebo for 9 weeks. Patients returned for clinic visits at 4 and 9 weeks and at 6 and 12 months. Both groups received motivational support at baseline and all follow-up visits. Motivational support consisted of a brief (two-minute) session with a physician prior to randomization followed by a session (≥ 5 minutes) with a research nurse or smoking cessation counsellor (if available) at baseline (prior to discharge) and at all follow-up visits.

Smoking status was determined at each clinic visit by self-report and biochemical validation by expired CO. Expired CO levels were measured using a Micro 4 Smokerlyzer monitor (Bedfont Scientific Ltd., USA) and are expressed in parts per million (ppm). In the original study, abstinence was defined by a self-report of no cigarettes smoked in the past week and an expired CO reading fewer than or equal to 10 ppm. Data from patients who returned for clinic visits and provided self-report and CO measures were included in the analyses. A total of 1,046 paired measures were obtained from each of the four follow-up visits (291, 287, 236, and 232, at 4 and 9 weeks and at 6 and 12 months, respectively).

The agreement between self-report and expired CO was calculated using the Cohen’s kappa statistic. Pearson’s correlations were performed to determine the relationship between number of cigarettes per day and CO levels. Data were analyzed using the PASW statistical software program (Version 18.0) and the OpenEpi Epidemiologic Calculator.

Results
Patients were predominantly male (84%) and the mean age was 54 years (standard deviation [SD] 10). Patients smoked an average of 33 years (SD 12) and 36% reported having another smoker at home. On average, patients reported smoking 23 cigarettes per day (SD 11) and 63% reported low-to-moderate nicotine dependence at baseline.

At 12 months, biochemical validation correlated with self-reported smoking status for 182 of 232 patients (kappa = 0.54; 95% confidence interval, 0.44–0.57). This pattern was consistent across all follow-up visits (data not shown). Of the discordant pairs, two (0.9%) included a self-report of non-smoking but a positive CO reading. These patients had a mean CO level of 24.5 ppm (SD 5.5). The remaining 48 discordant pairs included a self-report of smoking and a negative CO reading (false negatives, n = 48). These patients had a mean CO level of 5.9 ppm (SD 2.6) and reported smoking a median of 4 (interquartile range 1.4, 8.5) cigarettes per day. Patients with both a positive CO and self-report had an average CO of 18.7 (SD 7.7) and reported smoking a mean of 11.9 (SD 7.6) cigarettes per day.

At 12 months, the number of cigarettes smoked per day was positively correlated with expired CO levels (r = 0.70, Figure 1). The strength of the correlation increased across the 12-month follow-up (data not shown). Among patients who reported smoking more than 14 cigarettes per day, 90% had a CO reading greater than 10 ppm.

Discussion
Our study was designed to examine the validity of self-report for outpatient ascertainment of smoking status in post-MI patients. We found that biochemical validation by CO was only able to increase the likelihood of detecting smokers by less than 1% in patients. However, it may have discouraged patients from denying their smoking status and therefore should be considered for routine assessment of smoking.
status in the outpatient cardiac setting.

Self-report remains the conventional method for ascertaining smoking status in the outpatient post-MI setting. However, it has been found to be an inaccurate marker of smoking status. Moreover, biochemical validation has been shown to reduce the social desirability bias that often occurs in patients who suffer a major health event. A study investigating the validity of self-report found that patients with acute coronary syndrome (ACS) were more likely to deny their smoking status versus healthy individuals. Our finding that CO was only able to increase the likelihood of detecting smokers by less than 1% does not support these previous findings. However, knowledge of having to undergo biochemical validation may have deterred patients from denying their smoking status.

Our study had several limitations that should be noted. First, individuals who participated in the original study had to be motivated to quit smoking. This may explain the minimal effect of CO above that of self-report. Second, the knowledge of using a CO monitor at each clinic visit might have discouraged patients to deny their smoking status. Finally, we did not collect data on factors that could potentially affect the ability to detect biological levels of expired CO in the body (e.g., levels of physical activity, time since last cigarette, puffing habits). This could potentially explain the inability of CO to detect individuals who reported having returned to smoking.

**Conclusion**

Our study was designed to examine the validity of self-report for outpatient ascertainment of smoking status in post-MI patients. We found that expired CO does not substantially increase the likelihood of detecting smokers who would otherwise deny smoking. However, it may discourage patients from denying their smoking status and therefore should be considered for routine assessment of smoking status in the outpatient cardiac setting.

**References**

The Canadian Society of Internal Medicine has a noble mission statement that bears repeating. The goal is to “go beyond simple transmission of information, and to make a lasting impact on the knowledge, skills and attitudes of clinicians and future clinicians; to narrow the theory-to-practice gap; to improve the health of our patients and of all Canadians.” I agree wholeheartedly; in fact, I presume we all agree. How could you not? But the devil is always in the details. What does this really mean for how we prepare future doctors? More contentiously, what does this mean for duty hours? This (reactionary?) author believes it is worth routinely examining whether we live up to our lofty mission. If not, then we should accept a few inconvenient truths.

Workloads are not decreasing, but resident duty hours clearly are. Diminished resident hours means more routine clinical work done by fewer, more senior, and more expensive staff. Strangely, few cry foul over the fact that these “seniors” have less time to recuperate, manage their health, maintain their learning, promote quality control, or otherwise advance their field. In an increasingly stressed system, we should not be surprised if formal education becomes the first victim; next is the “luxury” of informal learning, followed by the working relationship between educators and trainees. In short, there are fewer staff members available to teach and fewer learners to receive their teachings. In what way is this beneficial to learners, let alone patients?

Even more concerning to this physician is that studies suggest that despite reduced trainee hours, errors are not decreasing, and time off is not being used for reading or writing. Trainees are not allowed to remain post-call as learners, nor do they stay to teach (each other, the nurses, or their students). In short, reduced on-call hours have not been shown to offer measurable benefits to patients. When I have fewer clinical hours, I increase my reading, writing, and reviewing (even if I exceed duty hours). This is because I have the privilege of a respected profession, coupled with the responsibility for vulnerable patients. That may sound atavistic, even quaint. But to return to that old medical litmus test: “What would your mother want?”

Others retort that they want their doctor to be rested, and reduced work hours clearly means fewer errors. However, not so fast. Less fatigue is associated with fewer errors, but the benefit is limited to that solo practitioner at that specific time; medical errors do not appear to lessen either overall or for individual patients. This is because errors are transferred to those practitioners who remain. In addition, errors may increase with increased sign-over (aka sign-off or hand-over) from one practitioner (or team) to another. Errors may increase because “juniors” attain less volume-based competence (i.e., weaker “clinical reflexes”). Moreover, those juniors remain junior (at least in skill set) for longer; they commit their errors at a more senior level and with less supervision. If reduced trainee duty hours were a drug, I doubt it would get Health Canada approval.

If trainees are doing less but we award the same qualifications, then simple mathematics shows that residencies need to be lengthened. Notably, few have called for this. Instead, competency-based training and simulation are enthusiastically promoted, although less enthusiastically funded. Both may play an important role, but they are not panaceas. After all, coupled with reduced hours, we should admit (and I would suggest we admit it to the public) that nowadays almost no trainee is
actually failed. Therefore, declaring competency means nothing if you cannot be declared otherwise. “Incomplete” trainees (we’ve even lost the courage to use the word “failed”) are blamed on inadequate efforts (or inadequate documentation) by their supervisors, strangely not on the individuals themselves. While seasoned practitioners are quite rightly expected to be responsible for their own education and outcomes, this responsibility is now de-emphasized in training. Accordingly, this (curmudgeon) physician is nervous for the future.

Patients need clinical decision making that is bespoke and compassionate. This requires experience and wisdom, which in turn come from following many patients through many illnesses. Reduced hours can mean that decisions are avoided, consults are increased, and more tests are ordered, even though unnecessary and harmful. All of this affects efficiency, throughput, cost, and safety. Reduced hours also decrease “patient ownership” and threaten generalism. Fragmented care (“I look after only the bowel lumen, not the whole gut”) is one thing, but more concerning is end-of-life decision making. Eighty percent of Canadians die in hospital but rarely because nothing more can be done. Instead, a decision is required (and before patients become moribund)—a decision that the patient’s dignity, comfort, and wishes are better served by palliative care, not escalation. In short, patient-focused care requires your presence and experience. I wish it were otherwise (I also have a home life), but wishing will not make it so.

The journey from “I don’t do it” to “therefore, I can’t do it” to “therefore, I won’t learn it” is perilously short. You may not forget how to ride a bicycle, but you can forget how to manage a complex patient or how to talk with families. However, with reduced hours, trainees might never learn to ride that bike. Moreover, if you believe (as my teachers taught me) that “education is what is left after you have forgotten what you were taught,” then we must accept the idea that trainees need to learn more than just facts. The hospital is where you learn attitudes, behaviours, and adaptability, as well as how to work with others. Only at the bedside do you learn that a “team of experts” is not an “expert team” and experience the perils of the “ninja consult” (notes hidden in a chart but nothing communicated) and the dangers of the “opaque consult” (no clear plan; just a repeat of the history, followed by those noncommittal words, “will review with staff”).

Presumably, the goal is for trainees to become—and for educators to help produce—graduates “fit for task” (i.e., competent and capable) as well as “fit for life” (i.e., balanced and psychologically resilient). Whether I liked or understood it, I attained competence by completing my “ten thousand hours” and following unambiguous feedback. I heard that “10% more effort can make you appear 50% better” and that getting hired is about “the three A’s”: availability, affability, and ability, in that order. I heard these things not because I was special but because I was there. I graduated from the University of British Columbia, whose motto, Tuum Est, means that your education is ultimately “up to you.” To update this motto, I would suggest it is “up to us.” This includes ensuring that our methods match our mission.

Peter G. Brindley MD
Associate Editor

“The hospital is where you learn attitudes, behaviours, and adaptability, as well as how to work with others.”
Residents are physicians undertaking further training to become independently licensed practitioners. Historically, resident duty hour periods were long and intense. The goal was to maximize learning through high patient volume and to teach doctors how to take responsibility. Recently, concerns over patient and resident safety have led to restricted trainee work hours. The putative justification is to improve resident education, resident well-being, and patient care. In light of this recent shift in the medical culture, resident duty hours have become a controversial topic.

Restricted duty hours take many forms. In the United States, the Accreditation Council for Graduate Medical Education (ACGME) mandated junior residents work no longer than 16 consecutive hours, while senior residents could work up to 26 hours. In Canada, no nationwide mandate exists and the issue falls within provincial jurisdiction. In Ontario, under the Professional Association of Residents of Ontario agreement, call-periods are no more than 26 consecutive hours in-house, no more than one in four nights in-house, or no more than one in three nights of home-call. After a 2011 Quebec court ruling, resident duty hours were restricted to 16 consecutive hours in that province. This resulted from the court concluding that traditional hours violate the Canadian Charter of Rights and Freedoms. Regardless, the Quebec ruling prompted other Canadian programs to further reduce resident duty hours and consecutive hours on-call.

To better understand this complex issue, the following review discusses resident safety, resident performance, resident education, and patient safety. Our goal is to present a balanced, evidence-based discussion, addressing both patient safety and resident fatigue management.
Effect of Duty-Hour Restriction on Patient Outcomes

Overall, there is no clear signal that reducing resident duty hours impacts patient safety. For example, a systematic review in 2004 examined patient mortality, adverse events, and medication errors. This systematic review, though limited by the quality of the studies available, found no change in patient mortality after the implementation of reduced working hours for residents.

A systematic review published in the *Annals of Internal Medicine* examined studies involving residency shifts with defined length, night float, or protected sleep. Only one of three studies found that six-month patient mortality reduction was associated with limited duty hours. However, all studies demonstrated reduced medical errors where resident duty hours were limited.

The most recent systematic review of 135 studies by Ahmed and colleagues examined both night-float and 16-hour shift systems separately. There was no overall improvement in patient outcomes attributable to resident duty hour reduction; however, several studies found increased complication rates with reduced resident duty hours for high acuity.

Effect of Duty-Hour Restriction on Education

Exposure and intensity is crucial for learning. However, the growing ease with which information can be accessed, and the growth of simulation may mitigate this concern. Goitein and colleagues studied the overall effect of duty hour restriction on resident education and found that more residents reported a negative effect (47%) than positive (32%) or neutral (21%). Seventy percent of residents felt there was not enough time for teaching by attending physicians and formal education was frequently missed. Overall, senior residents were less likely to approve of duty hour restrictions and more likely to report negative effect on education. These findings were confirmed by a systematic review by Ahmed and colleagues.

Effect of Duty-Hour Restriction on Resident Well-Being

Overall, studies into the associations between resident duty hours and resident well-being are inconclusive. In the general population, the 12-month prevalence of any mood disorder is 9.5%. By comparison, studies consistently demonstrate a depression prevalence of 27–30% within 12 months of residency training. Reduced duty hours can lead to work compression, namely, trying to accomplish the same tasks in a reduced period of time. This, in turn, can increase rather than alleviate pressure and stress for residents. While the implementation of the 80-hour work-week with the first ACGME mandate in 2003 was followed by improvement in resident wellness, reducing the shift length to 16-hour maximums in 2011 was not associated improved resident experiences.

The well-publicized study by Sen and colleagues further illustrates the conflicting nature of duty hour reform. The group studied the effects of the 2011 duty-hour reforms in over 2000 United States residents regarding sleep, work hours, and medical errors. There was no change in hours slept, depression scores, or subjective well-being scores following a limited change in resident duty hour conditions (16-hour maximums). However, the average weekly duty hours only decreased from 67 hours to 64 hours.

Duty-Hour Restriction and Handover

One possible reason that patient safety has not improved by reducing resident duty hours is because this creates more handovers and between relatively junior practitioners. Many studies have shown the importance of performing a thorough and standardized handoff. It is unclear who provides safer care to patients: a well-rested resident who has to rely upon a handover, or a fatigued resident but with first-hand patient knowledge.

Conclusion

Residency training can be associated with reduced quality-of-life, increased burnout, and mental stress. However, reduction in resident duty hours has not been demonstrated to lead to harm or benefit for residents or for patients. In fact, reduced resident duty hours highlights new problems: work compression, increased handover, and decreased education.

Consecutive or total duty hours are not the only contributor to resident fatigue and burnout. Outside of clinical work, residents have research responsibilities, educational endeavours, and the opportunity to work extra shifts for additional income. Independently licensed physicians do not have restrictions on their work hours; as such, changes in duty hours in isolation do not reflect “real world” practice. Accordingly, residency training programs need to develop well-rounded fatigue management plans, not just duty-hour restriction.

Fatigue management includes ensuring a safe and supportive resident work environment, along with graded responsibility and adequate back-up. Time for sleep is important, as is attention to diet, exercise, support system, and mental health. These changes may require changes to how we configure our teams and deliver our education. This may include shifting towards focused exposure rather than sheer volume and a greater role for medical simulation. We must also recognize those at risk for burnout and in need of better coping strategies. There is clearly much work to be done.
References

“W e see further” … for many reasons: better teaching, better access to information, better diagnostic tools. Sometimes we are fortunate to meet—and learn from—giants in the profession, whose accumulated wisdom and experience are shared with junior learners. Usually this advice is gleaned with great difficulty; it is not written down or etched in stone. It is a passing remark, a knowing smile, a saying that just sticks in the mind.

There have been many “condors” and “eagles” that have shown me the updrafts of medicine, whose vision has been inspiring, and whose integrity and insight are beacons for those that follow.

I remember the day I told a nurse: “Communication is nine-tenths of medicine.” She turned quietly, thought for a moment, and said: “Communication is nine-tenths of life.” Of course, she was correct. When we hear patients breathing heavily or shuffling hesitantly down the corridor behind us, the dialogue has begun. You turn and hold their cold, frail hands, and a link is forged. You eye their lined visage; their dark, knowing eyes say, “Hear my story.” You disarm their anxiety with well-worn pleasantries and seek—through measured inquiry—what it is that brings them to your office. Sometimes you just hit it off, as if you are at some sort of social get-together. Sometimes you have to earn your fee, cutting through a bland defensive visage to a colourful spirit beneath.

I sometimes say: “Well, that’s the interrogation over.” My new acquaintance sighs and her shoulders relax. A smile creases her face, and she knows things will be okay. The cadence of question and answer is as much a part of medicine as the scalpel is to surgery. A “gatling gun” repartee gives the recipient the impression you are short of time and short of interest. Wait a moment (say “one hundred” to yourself) before moving on, and consider what might to you be a simple answer. When you get good at this job, ask less and learn more. The pregnant pause behind “You don’t sleep well; why is that?” may bring out a tearful disclosure, an admission of worrisome facts, or even an accurate self-diagnosis.

I frequently have to remind myself that I am privileged to share in this patient’s life story, to be a character in her play or even a chapter in her book. She teaches me my job, since much of learning comes from experience. Her illness may be her burden, but it is my textbook. I tread carefully here, because one day I will be on her side of the desk, worried about a lump, anxious about chest pain, or challenged by arthritis.

Sometimes things don’t go well; responses are vague, slow, and poorly formed. I forget that illness dulls the intellect, that age can limit vision and hearing and memory, and that the patient’s life experiences may vastly outrank mine. Or I feel hurt by an unexpected challenge, a threat to my integrity. It is a mark of maturity to suppress the rapid pulse, the surging blood pressure, the tunnel vision, and the urge to make a cutting riposte—to stop, to breathe, to “reset,” and to salvage a difficult situation. Every day, I come to work and say to myself: “How many strangers are going to come into my life today? How many friends will leave?” I hope all will have more understanding, more hope, and better health after a consultation. This is what interns can offer: a listening ear, a thoughtful mind, a guiding voice, and above all compassion.

If I had to describe my role in this complex profession, I would liken it to that of a tugboat captain: I seek out ships that are cast adrift, swept close to rocks by unfavourable tides, much like the Russian tanker Simushir, helpless off Western shores for want of a heat pump to put things right. Sometimes all it takes is a strong haul on a sturdy rope, and disaster is averted.

Sometimes, the ship is boarded and the course redrawn, the cargo jettisoned, or the crew removed. If I can’t save the vessel and know that the captain will not survive, I have a life jacket and a sailor’s prayer. We offer hope and strive to afford a degree of peace and dignity that all of us deserve at the end.

When I started out as a young doctor, I learned the words of Hippocrates: “With purity and holiness I will live my life and practice my art.” It has taken me decades of experience to truly understand these words. I hope to have come close to living his values. And I hope that those who follow will bring more to the profession than they take away.
APPLY FOR A CSIM AWARD!

**DR. DAVID SACKETT SENIOR INVESTIGATOR AWARD**  
(Deadline: May 12, 2015)  
To recognize excellence in research by a senior Canadian general internist. The award is intended to increase awareness of high quality research by general internists in Canada and to foster such research.

**NEW INVESTIGATOR AWARD**  
(Deadline: May 12, 2015)  
In recognition of excellence in the field of internal medicine research by a young investigator.

**THE HUI LEE HEALTH PROMOTION SCHOLARSHIP**  
(Deadline: June 10, 2015)  
To promote excellence in health promotion; and to honour and remember the life of Dr. Hui Lee, a cherished member of the Canadian Society of Internal Medicine.

**CALL FOR ABSTRACTS**  
(for trainees) (Deadline: June 10, 2015)  
- Quality Improvement Abstracts  
- Ted Giles Clinical Vignettes  
- CSIM/CAPM Research Abstracts

**THE CSIM OSLER AWARDS**  
(Deadline: June 22, 2015)  
These awards are to demonstrate excellence in achievement in the field of general internal medicine (GIM), either in clinical practice, research, medical education or specialty development.

**GIM FACULTY RESEARCH SHOWCASE**  
(Deadline: July 13, 2015)  
The GIM Faculty Research Showcase highlights the research programs of Canadian general internists. This Showcase is largely intended for Full Members of the CSIM. Research from senior trainees in GIM (PGY4, PGY5, or greater) is also eligible for consideration.

**CSIM EDUCATION AND RESEARCH FUND**  
(Deadline: November 2, 2015)  
- Encourage residents to pursue careers in both community and university-based General Internal Medicine (GIM) to do an elective at another Canadian site to pursue a scholarly activity in medical education, clinical research project or QI project.  
- Encourage the development of GIM (community and university-based) as a specialty by facilitating scholarly activity, knowledge exchange and pursuit of new knowledge.

**EUROPEAN SCHOOL OF INTERNAL MEDICINE CONFERENCE**  
(Deadline: April 1, 2015)  
The Canadian Society of Internal Medicine offers one travel award to help defray travel and registration costs of a Canadian general internal medicine resident or fellow interested in attending the European School of Internal Medicine, an international conference for trainees in internal medicine.

Open to CSIM Members and CSIM Residents and Medical Students, visit www.csim.ca for terms of reference.

---

**CSIM ANNUAL MEETING 2015**

Join us at the CSIM Annual Meeting October 14-17, 2015.  
Visit www.csim.ca to reserve your hotel room at the special conference rate at the Delta Prince Edward in Charlottetown.

**JOIN THE CSIM**

Visit www.csim.ca for a list of membership benefits - including eligibility for awards and scholarships, discounted CSIM Meeting registration fees, access to CPD events and more. Membership is FREE for residents and medical students!

Watch www.csim.ca and join us on Twitter and Facebook for updates!
Cannabinoid Hyperemesis Syndrome:
Rare or Underdiagnosed?

Selena Au MD, Ali Rezaie MD

Case
A 38-year-old male presented to the emergency department with severe acute-on-chronic abdominal pain. For the previous 20 years, he had experienced daily sharp epigastric pain with generalization to the entire abdomen. The pain typically began late in the morning and could last from hours to days. It was associated with nausea, vomiting, diaphoresis, and occasionally fever, diarrhea, and fatty or bloody bowel movements. These symptoms were not associated with food consumption. Over the previous five years, the patient had lost over 20 kg in weight and had noticed a marked decline in his performance at work and in the quality of his leisure time. He found symptom relief by smoking rolled marijuana cigarettes (“joints”), which he had been using three times per day since his teens. His pain crises were alleviated only with soaking in a tub of “scalding hot” bath water which he drew up ritualistically every morning.

The patient’s symptoms had previously been investigated by his family physician, who suspected acute intermittent porphyria (AIP), but it is unclear what medical diagnostics had been performed. Quetiapine (50 mg qhs) had been prescribed and started one year prior to presentation, but during a review of systems, the patient denied having had any psychiatric symptoms. His medical history was significant only for smoking and lactose intolerance. Review of systems and family history were negative for autoimmune, infectious, malignant, or inflammatory bowel disease. When questioned as to why he chose to come to the emergency department just then, the patient replied that his family physician had suggested that he present during one of the more severe pain crises in order to allow laboratory work-up to confirm the diagnosis of AIP. At the time of presentation, the patient had been ill for the previous four days.

On examination, the patient was febrile (38.6°C), hypertensive (195/113), and markedly dehydrated. He was of thin habitus and had normal results on cardiorespiratory examination. His abdomen was nondistended but diffusely tender. Abdominal rigidity with guarding was noted by the emergency physician but was absent on reassessment by internal medicine.

The patient’s laboratory investigation results were consistent with a stress response and dehydration, including hemoglobin = 193 g/L, white blood cell count = 20 × 109 g/L, creatinine = 164 µmol/L, and lactate = 2.7 mmol/L. Liver enzymes and the results of tests of hepatic synthesis were normal. Urine porphobilinogen at the time of crisis was negative for AIP.

Upon a review of the literature, a diagnosis of cannabinoid hyperemesis syndrome was made. To rule out other organic etiologies, the patient underwent abdominal computed tomography, which was unremarkable. Upper gastrointestinal endoscopy with gastric biopsies revealed no pathology.

Discussion
Cannabinoid hyperemesis syndrome (CHS) is a previously described but underdiagnosed adverse effect of marijuana use. It was first characterized in a group of nine patients by Australian investigators in 2004. Clinical features of the syndrome include the following:

1. long-term, excessive use of marijuana;
2. a cyclical vomiting pattern that often begins years after the initiation of substance use;
3. abdominal pain;
4. compulsive hot showering or bathing, with symptom relief; and
5. the resolution of symptoms upon cessation of marijuana use.1

About the Authors
Selena Au is a member of the Department of Critical Care Medicine at the Cumming School of Medicine in Calgary, Alberta and holds a Masters in Science in Quality Improvement and Patient Safety. Ali Rezaie is a gastroenterologist at Cedars Sinai Medical Center in Los Angeles. Correspondence may be directed to selena.au@albertahealthservices.ca.
Since this initial description, approximately 50 case reports and series have appeared internationally. The largest case series to date includes 98 patients reviewed at the Mayo Clinic from 2005 to 2010 and characterizes the typical features of patients with CHS. Common features of cases described in the literature include long-standing undiagnosed symptoms; multiple unnecessary diagnostic tests; and major surgical intervention for symptoms, with no relief. Table 1 lists the typical features of CHS patients in addition to the cardinal features listed above.

<table>
<thead>
<tr>
<th>Clinical Presentation of a Patient with Cannabinoid Hyperemesis Syndrome&lt;sup&gt;3&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Essential for diagnosis</td>
</tr>
<tr>
<td>Major features</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Other typical features</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

### Pathophysiology

The pathophysiology of CHS remains poorly understood. Cannabinoids exert their psychoactive and antiemetic properties centrally on cannabinoid type 1 (CB1) receptors; cannabinoid type 2 (CB2) receptors are typically found peripherally in nonneuronal tissues. Disturbances of the hypothalamus-pituitary-adrenal axis and the presence of autonomic instability have been described as the framework for symptoms in CHS. It has been postulated that the chronic stimulation of CB1 may cause receptor downregulation and internalization, leading to the paradoxical vomiting reaction. CB1 receptors are also located on peripheral enteric nerves, where their stimulation is known to slow gastrointestinal transit, potentially overwhelming the antiemetic central CB1 effects. The behaviour of compulsive hot bathing and showering remains an area of much speculation but likely relates to the effect of cannabinoids on CB1 receptors in the temperature-regulating centres of the hypothalamus.

### Treatment

Long-term follow-up in the Mayo Clinic case series was poor due to the majority of its patients’ being referred from abroad. However, of the 10 patients who had complete follow-up (10%), 7 stopped using cannabis, and 6 of the 7 (86%) noted a complete resolution of their symptoms. The only patient who did not notice any improvement had stopped consuming marijuana only one month previously. Because of the lipophilic nature and long half-life of cannabinoids, prolonged abstinence must be encouraged. Although typical antiemetics and analgesics seem to provide little relief from acute symptoms, successes with intravenous haloperidol, risperidone, and lorazepam have been described.

In our patient’s case, he was given information on his diagnosis and was advised to abstain from marijuana use with concurrent down-titration of his quetiapine. A follow-up telephone call six months after diagnosis revealed that his pain had continued in the setting of ongoing marijuana use. Three months after diagnosis, he had presented to the emergency department again, at which point he was off quetiapine but back on marijuana. He had abstained for one month but resumed use because of the lack of rapid symptom improvement.

### Implications

In September 2013, Statistics Canada reported that more Canadians experienced symptoms of cannabis abuse or dependence in their lifetime (6.8%), compared with other drugs (4.0%). Of Canadians, 1.3% met the criteria for cannabis abuse or dependence in the past year. While these statistics pertain to recreational marijuana use, 37,000 licensed medical marijuana users receive treatment for chemotherapy-induced nausea and vomiting and other conditions. This number is expected to reach 400,000 in the next decade. Effective April 1, 2014, Health Canada’s new medical marijuana regulations shift the responsibility from federal regulators to doctors and nurse practitioners to write “medical documents” similar to prescriptions and authorizing patients to obtain marijuana from a federally licensed provider. As with any other safe prescribing practice, the physician must have an adequate understanding of adverse reactions and must disclose them to the patient. However, as demonstrated by our case, the literature on the adverse effects of chronic marijuana use is incomplete. Because the general public thinks of marijuana as having antiemetic properties, patients may not disclose their use of it during history taking unless specifically probed by a medical professional. Similarly, symptom relief by hot baths may be the tipoff for CHS, but only for a physician primed on this condition.
References


Basilar artery occlusion is associated with a high mortality rate and poor functional outcome in survivors. The most common prodromal symptoms are motor and oculomotor deficits. Hearing loss is not a major prodromal symptom.

Sudden deafness usually results from either circulatory disturbances or inflammation. Deafness of vascular etiology generally occurs unilaterally. Occlusion of basilar arterial origin suggests that thrombosis is the primary mechanism of stroke, especially in elderly patients. This case highlights the importance of hearing loss as either a main manifestation or a warning of impending brainstem ischemia. Clinicians should be aware of the possibility of vertebrobasilar ischemia in patients with bilateral sudden deafness, even when classic brainstem or cerebellar signs are mild or absent. This case also highlights the importance of prodromal signs and symptoms.

Case Report

A 65-year-old right-handed woman with long-standing hypertension presented to the emergency room with sudden bilateral deafness and difficulty communicating. Collateral history was obtained from her family, as the patient was unable to communicate because of her bilateral deafness. Twelve to 24 hours previously, the patient had had an acute onset of intense vertigo-associated vomiting and had become bedridden because of ongoing vertigo and dizziness, with rapid deterioration of her clinical status over the previous two hours.

She now had slurred speech and was unable to ambulate independently. The patient’s family denied her having any convulsions, incontinence, or loss of consciousness. The patient’s only medication was hydrochlorothiazide (25 mg daily). She had no cerebrovascular risk factors except hypertension. She was a nonsmoker and a nonalcohol user. Her family history was noncontributory. She had no known drug allergies.

On arrival at hospital, her Glasgow Coma Scale score was 9 (E2, M5, V2). She was afebrile and in sinus tachycardia with a heart rate of 115 beats per minute (BPM), her blood pressure was 177/85, and her oxygen saturation was 99% on 5 litres of oxygen. Her pupils were equal and reactive to light. Funduscopy revealed hypertensive eye changes but no papilledema. Cranial nerve VIII was compromised; the remaining cranial nerves could not be fully assessed as the patient was unable to follow commands. Her neurologic examination was limited due to her hearing impairment. The right upper and lower extremities were flaccid relative to the left, and plantar reflexes were bilaterally extensor. Cerebellar testing could not be performed, as the patient was not fully able to cooperate.

Her respiratory examination was unremarkable. There was no evidence of volume overload. Her jugular venous pulse was flat, and heart sounds were normal. She had a 2/6 holosystolic murmur at the apex that radiated to the axilla. Her abdomen was soft, and no obvious guarding was noted. Her level of consciousness was fluctuating, and she was intubated and mechanically ventilated for airway protection.

On admission her hemoglobin level was 87 g/L, with a mean corpuscular volume of 90 fl, white blood cell counts were 19,900
g/L with 92% neutrophils, 4% lymphocytes, 2.5% monocytes, and 0% eosinophils. Her blood film showed no evidence of hemolysis. Platelets were 150,000 g/L. Her serum sodium level was 136 mEq/L, serum potassium level was 3.2 mmol/L, and serum chloride level was 111 mEq/L. Serum creatinine was 99 µmol/L, urea was 33 mmol/L, and a random glucose level was 95 mg/dL. Her serum transaminases, amylase, and lipase were all within normal limits, but cholesterol values were extremely elevated, with low-density lipoprotein at 4.2 mmol/L and high-density lipoprotein at less than 1.0 mmol/L, total cholesterol/HDL greater than 5, and triglycerides at 2 mmol/L. Cardiac work-up and autoimmune and vasculitic screens were negative except for an elevated C-reactive protein and erythrocyte sedimentation rate. Her transthoracic echocardiogram revealed a normal ejection fraction and mild-to-moderate mitral regurgitation. Her electrocardiogram showed sinus tachycardia at 140 BPM, with evidence of left ventricular hypertrophy but no ST changes and a normal QT interval of 420 ms. Noncontrast brain computed tomography showed high attenuation of the basilar artery, consistent with a thrombus and bilateral cerebellar peduncle infarction, and T2-weighted magnetic resonance imaging of the brain confirmed hyperintensities involving both inferior cerebellar peduncles and pontomedullary junction. Magnetic resonance angiography of the brain revealed complete occlusion of the basilar artery at its origin and severe stenosis of the distal vertebral artery bilaterally.

Due to the delay in presentation, the patient was not eligible to receive intravenous thrombolysis. In fact, in order to be effective, patients should be treated with intravenous tissue plasminogen within 4.5 hours of the onset of stroke symptoms. Instead she was started on an antiplatelet agent, clopidogrel 75 mg daily and simvastatin.

The patient was admitted to the intensive care unit for further monitoring and care. During her stay, she remained dysarthric and unable to obey commands consistently. She had recovered very little of her motor function; her balance had mildly improved and she continued to be wheelchair dependent. Otoscopic examination confirmed sensorineural hearing loss (SHL). Urgent audiometric testing and brainstem auditory evoked potentials confirmed almost complete hearing loss bilaterally. Eventually the patient was extubated, and she was supplied with hearing aids and referred to a rehabilitation centre, where she participated in a graded exercise rehabilitation program. A follow-up pure tone audiogram confirmed 80% bilateral deafness, while her motor deficits (including coordination and gait) improved steadily with rehabilitation over several weeks. At a three-month follow-up post-discharge, bilateral deafness persisted.

**Discussion**

Idiopathic sudden SHL has an incidence of 5 to 30 cases per 100,000 cases per year. The hearing loss is unilateral in most cases; bilateral involvement is reported in less than 5% of cases. Tinnitus occurs in about 80% of patients, and vertigo indicating an associated peripheral vestibular dysfunction occurs in about 30%. Up to 80% of patients report a feeling of ear “fullness.” In 2.8% of patients, sudden SHL is due to vascular or hematologic causes, particularly ischemia involving the cochlear or the ascending pathways. The cochlea is supplied by an end artery, and vascular occlusion has been postulated to be a cause for sudden SHL. In our case there was evidence of basilar artery infarction, suggesting that thrombosis was likely the cause. This is in keeping with previous studies that have shown that local atherothrombosis is the major etiology of vertebrobasilar occlusions in the elderly population.

Despite limited large-scale studies, the literature has shown that sudden SHL is associated with a significant increase in the hazard of stroke during the subsequent five years. From this we can conclude that patients with sudden hearing loss should be examined for additional brainstem symptoms, since this can be the presenting sign of a life-threatening basilar artery thrombosis.

**Conclusion**

Vertebrobasilar or posterior circulation territory stroke accounts for 20% of all strokes. Basilar artery occlusion is generally associated with a high mortality rate and poor functional outcomes in survivors (i.e., major disabilities, including tetraplegia and coma). Sudden SHL can present as a warning signal of an acute basilar artery stroke. Therefore, an early diagnosis is essential if direct or supportive measures are to be of benefit.

This case highlights the importance of hearing loss as either a main manifestation or a warning of impending brainstem ischemia. Clinicians should be aware of the possibility of vertebrobasilar ischemia in patients with bilateral sudden deafness, particularly in older patients, even when classic brainstem or cerebellar signs are mild or absent.
Sudden Bilateral Deafness

References

A Forgetful Experience: 
A Case of Transient Global Amnesia

Kalpa Shah MD, PGY2, Chris Sheasgreen MD, PGY 3, Ameen Patel, MB

About the Authors
Kalpa Shah is a postgraduate PGY3 resident in Internal Medicine, Christopher Sheasgreen is a PGY4 resident in his Gastroenterology fellowship, and Ameen Patel is a staff member of the Division of General Internal Medicine and holds the William J. Walsh Chair in Medical Education, all at McMaster University, in Hamilton, Ontario. Correspondence may be directed to Ameen Patel patela@mcmaster.ca

Summary
We present a case of a 67-year-old man with transient global amnesia, a clinical syndrome that presents with an acute onset of temporary amnesia lasting less than 24 hours, without impairment of consciousness or cognition. In this article, we discuss the clinical criteria for diagnosis, the pathophysiology of the condition, and an approach to its diagnostic work-up and prognosis. It is important to differentiate TGA from other entities because it is a benign condition that does not require treatment.

Résumé
Nous présentons le cas d’un homme de 67 ans en ictus amnésique, syndrome clinique caractérisé par un trouble mnésique transitoire d’installation subite qui dure moins de 24 heures, sans altération de la conscience ou de la cognition. Nous examinons les critères cliniques du diagnostic, la physiopathologie du trouble, une méthode d’investigation diagnostique et le pronostic. Il est important de distinguer l’ictus amnésique d’autres troubles étant donné que ce syndrome bénin ne nécessite pas de traitement.

Transient global amnesia (TGA) is a clinical syndrome that presents with an acute onset of temporary amnesia lasting less than 24 hours, without impairment of consciousness or cognition. It has an incidence of 3 to 8 per 100,000 people per year.1 Approximately 75% of TGA episodes occur in individuals between 50 and 70 years of age.2 It is uncommon in patients younger than 40 years of age. Although various pathophysiologic explanations have been put forth, the etiology of TGA remains uncertain.

Case Presentation
A 67-year-old man presented to the emergency department with confusion and amnesia. Earlier that day, he had awoken feeling completely normal. He met some friends for coffee at a mall, as per his usual routine. His friends reported no unusual behaviour or confusion. The last thing he remembered is having bid goodbye to his friend at 12:30 p.m., and he had no recollection of events after having left his friend.
His son had found him at home approximately two hours later, in his underclothes, repeatedly opening and closing the automated garage door and asking whether his son knew where the car was. His son reports having found his father’s clothes strewn across the main floor of their home. The patient did not recollect that his wife had taken the car after dropping him off at the mall. During the episode he was alert and responsive. He was oriented to person but disoriented to place. He was subsequently brought to the emergency department.

He had no recent travel history, sick contacts, or infectious symptoms. He had no bowel or bladder incontinence, no smell or taste sensations, and no visual or visceral (epigastric rising) auras. There were no complaints of focal weakness, numbness, tingling, slurred speech, vertigo, diplopia, chest pain, shortness of breath, nausea, vomiting, diarrhea, or fevers and chills. There was no history of prior similar episodes.

He had no medical conditions, was in excellent general health, and did not take any prescription or over-the-counter medications. There was no history of tobacco or recreational drug use, and he used alcohol rarely. He had no family history of neurologic or psychiatric disease.

On examination he had a heart rate of 82 beats per minute and a blood pressure of 152/97 mm Hg. He was afebrile and had an oxygen saturation of 96% on room air. He was oriented to person, place, and time. The examination of his head and neck, heart, lungs, abdomen, and extremities were normal. A neurologic examination revealed anterograde amnesia from approximately 12:30 p.m. Long-term memory was preserved. There was no evidence of hallucinations or delusions, and he was able to correctly identify his family members. He had no focal motor, sensory, coordination, or cranial nerve deficits. His reflexes were 2+ in the upper and lower extremities. Plantar reflexes were down going bilaterally. His speech was fluent and without evidence of aphasia or dysarthria. During the examination, he repeatedly asked what had happened to him. Laboratory investigations showed a normal complete blood count, urea, creatinine, electrolytes, glucose. A liver panel consisting of total bilirubin, alanine transaminase (ALT), alkaline phosphatase, and international normalized ratio (INR) were within normal ranges.

Venous blood gas analysis showed a pH of 7.35 (7.32–7.42) and a partial carbon dioxide pressure (Pco₂) of 52 (38–50) mm Hg. Additional blood work revealed a thyroid-stimulating hormone (TSH) level of 1.2 (0.3–4.2) mIU/L and a C-reactive protein (CRP) level of 2.7. The result of a urine toxicology screen was negative, and serum levels of acetaminophen and salicylate were undetectable. Electrocardiography revealed normal sinus rhythm. The results of chest radiography, brain computed tomography (CT), magnetic resonance imaging, and electroencephalography were normal.

His amnesia resolved over 24 hours, and he had no ongoing symptoms of anterograde amnesia. There have been no recurrences in the six months since his discharge.

**Discussion**

The term “transient global amnesia” was originally used by Fisher and Adams in 1964 to describe a clinical syndrome characterized by the abrupt onset of anterograde amnesia accompanied by repetitive questioning lasting minutes to hours.³ With the exception of amnesia, there were no other neurologic deficits, and patients were alert, responsive, and aware of their personal identity during the attacks. Since then, diagnostic criteria for TGA have been developed by Caplan and later validated by Hodges and Warlow in a prospective study.⁴ Hodges and Warlow followed 153 patients who met the diagnostic criteria for TGA (Table 1) and found that the rate of vascular events (a vascular event being defined in the study as myocardial infarction, stroke, or death) in the TGA group was not increased compared to the general population at 6 months after the episode of TGA. These findings are in keeping with those of a multicentre study in Denmark and prospective follow-up studies of TGA patients, published by the Mayo Clinic.⁵,⁶ For a diagnosis of TGA to be made, all criteria listed in Table 1 must be met. If only some of the criteria are met—particularly if there are neurologic deficits—the likelihood of alternative diagnoses is greater.

<table>
<thead>
<tr>
<th>Table 1. Clinical Criteria for the Diagnosis of Transient Global Amnesia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presenecne of an attack of anterograde amnesia (most of the attack should be witnessed)</td>
</tr>
<tr>
<td>- No clouding of consciousness or loss of personal identity</td>
</tr>
<tr>
<td>- Cognitive impairment limited to amnesia (no apraxia, aphasia, etc.)</td>
</tr>
<tr>
<td>- No focal neurologic or epileptic signs during or after the attack</td>
</tr>
<tr>
<td>- No recent history of head trauma or seizures</td>
</tr>
<tr>
<td>Resolution of symptoms within 24 hours</td>
</tr>
</tbody>
</table>

Source: Adapted from Hodges JR, Warlow CP.⁴

**Etiology**

The clinical symptoms of difficulty in the formation and retrieval of memories in TGA implicate the hippocampus and the temporal lobe as potential areas of involvement. Several pathophysiological explanations have been proposed, such as migraine-related mechanisms, ischemia, and epileptic phenomena, as well as venous flow abnormalities.⁷ Different modalities of neuroimaging studies have been used to investigate the area of the brain and mechanism involved in TGA. Everal studies using single-photon emission CT show a decrease in
cerebral blood flow in the temporal lobe and hippocampal region in most patients. Studies with diffusion-weighted imaging (DWI) have yielded conflicting results, some studies showing no abnormalities on DWI and other studies describing small lesions in the hippocampal region. The etiology of TGA continues to be shrouded in controversy.

**Differential Diagnosis**

The differential diagnosis of TGA includes complex partial seizure, transient ischemic attack, acute confusional state, transient epileptic amnesia, dissociative amnesia, intoxication, and Wernicke-Korsakoff syndrome.

It is often difficult to differentiate TGA from transient ischemic attack (TIA); some data suggest that TGA is a result of an ischemic process involving the hippocampus. Findings of focal neurologic deficits, such as motor weakness or sensory abnormalities, favour the diagnosis of TIA. In addition, the presence of risk factors such as smoking, hypertension, and dyslipidemia should increase the clinical suspicion for TIA.

In patients with TGA, self-identity is preserved, which differentiates TGA from some psychiatric disorders such as dissociative amnesia, in which personal identity is lost. Furthermore, attention is intact in episodes of TGA, in contrast to drug-related confusional states in which patients may present with attention deficits.

Patients who are experiencing complex partial seizures often display automatisms, staring spells, and impaired consciousness; whereas in TGA, patients are alert. It is more challenging to differentiate TGA from a complex partial seizure when patients present in a postictal state.

Patients with Wernicke-Korsakoff syndrome will have a history of heavy alcohol use, and the clinical presentation will be characterized by anterograde as well as retrograde amnesia and confabulation. There may also be other clues on examination and laboratory data to support excessive alcohol use.

**Work-Up**

It is important to note that TGA is a clinical diagnosis and remains a diagnosis of exclusion. The diagnostic criteria as outlined by Hodges and Warlow act as a tool to help guide clinicians. If all the criteria are met, the probability of a diagnosis of TGA is increased. An initial work-up should include serum electrolytes, glucose, oxygenation status, and a toxicology screen. It is often useful to perform brain imaging to rule out other serious causes such as trauma and stroke. In the absence of recurrent episodes, risk factors for seizures (such as a prior stroke), or a history suggestive of epileptic activity, it is not necessary to perform electroencephalography.

**Treatment and Prognosis**

A benign condition with no long-term sequelae, TGA requires no treatment. The reported annual rate of recurrence varies between studies but is within the range of 5% to 8%. There is no published literature that indicates a subsequent risk of delirium or neurodegenerative diseases in patients who have experienced TGA.

**Conclusion**

Transient global amnesia (TGA) is a syndrome of reversible amnesia of unclear etiology. It occurs most frequently in individuals between 50 and 70 years of age. It is important to differentiate TGA from other causes of neurologic impairment, as it is a benign condition that does not require treatment. Applying the diagnostic criteria can help clinicians determine the probability of a diagnosis of TGA. For cases that do not meet all of the diagnostic criteria, further investigations should be considered to rule out more serious conditions that require intervention. Diseases may not necessarily present as described in the literature and oftentimes will help unveil the diagnosis.

**References**

Thoracic Aortic Dissecting Aneurysm Presenting with Stroke, Disseminated Intravascular Coagulopathy, and Gastrointestinal Hemorrhage

Soraya Moghadam MD, Yoshitsugu Nakamura MD, Mackenzie Quantz MD, Raymond Kao, MD

About the Authors
Soraya Moghadam is an internist/nephrologist with the Canadian Forces Health Services and is based in Ottawa; Raymond Kao is with the Canadian Forces Health Services and staff intensivist; Yoshitsugu Nakamura is a cardiac surgery fellow; and Mackenzie Quantz is a staff cardiac surgeon – all at London Health Sciences Centre, London, ON. Correspondence may be directed to rkao3@uwo.ca

Case Review

Summary
Acute ischemic stroke secondary to vessel occlusion from an aneurismal dissection is an uncommon presentation. Disseminated intravascular coagulation (DIC) can present as a consequence of aortic dissection, although this is also rare. In some cases, the laboratory diagnosis of DIC uncovers a vascular abnormality or bleeding diathesis. This article describes a patient presenting with three sequential complications of a dissecting thoracic aortic aneurysm: ischemic stroke, upper gastrointestinal bleeding, and consumptive coagulopathy.

Résumé
L’accident vasculaire cérébral (AVC) ischémique aigu secondaire à l’occlusion vasculaire découlant d’une dissection anévrismale est rare. La coagulation intravasculaire disséminée (CIVD) peut se produire à la suite de la dissection aortique, quoique cela soit rare également. Dans certains cas, le diagnostic de CIVD en laboratoire dévoile une anomalie vasculaire ou un phénomène de diathèse sanguine. Nous présentons le cas d’une femme de 73 ans qui a subi trois complications successives de la rupture d’un anévrisme de l’aorte thoracique : un AVC ischémique, un saignement digestif haut et une coagulopathie de consommation.
Case
A 73-year-old woman experienced shortness of breath and an inability to walk while at church. She then had a sudden decrease in level of consciousness and was brought to the local hospital. Her medical history included hypertension, osteoarthritis, and a history of smoking 20 packs of cigarettes a year. Her medications on presentation were enalapril, aspirin, and meloxicam. Her decreased level of consciousness improved with intravenous fluid resuscitation. Then she complained of headache and left-sided weakness. Her heart rate was 68 beats per minute (BPM) and regular, blood pressure was 89/45 lying, and temperature was 37.8°C. A computed tomography (CT) head followed by angiography was completely normal. A CT chest showed a Type A aortic dissecting aneurysm extending downwards, from the aortic root to the mid-abdominal aorta and upwards into the right common carotid artery, which was occluded proximally (Figure 1).

She was transferred to the London Health Sciences Centre, Cardiac Surgery Recovery Unit (CSRU). En route, the patient had a gastrointestinal hemorrhage with melena and bright red blood per rectum. Upon arrival, her heart rate was 76 BPM, blood pressure was 115/50 lying, respiratory rate was 20 breaths per minute, and temperature was 32.3°C. Fluid resuscitation was continued and a nasogastric tube was inserted, resulting in drainage of a significant amount of bright red blood. Initial laboratory results revealed a hemoglobin of 136 g/L (subsequently 99 g/L), white blood cell count of 22.2 x 10^9/L, platelets decreased at less than 20 x 10^9/L with some clumping, international normalized ratio (INR) 2.0, partial thromboplastin time (PTT) 59, fibrinogen 0.41 g/L, lactate 1.0 mmol/L, alanine aminotransferase (ALT) 59, fibrinogen 0.41 g/L, lactate 1.0 mmol/L, alanine aminotransferase (ALT) 37 U/L, aspartate aminotransferase (AST) 52 U/L, sodium 142 mmol/L, potassium 3.5 mmol/L, chloride 119 mmol/L, bicarbonate 19 mmol/L, urea 7.6 mmol/L and creatinine 48 µmol/L. She was aggressively warmed and resuscitated throughout the night with one unit pooled platelets and two units pooled cryoprecipitate, four units of fresh frozen plasma, and two units of packed red blood cells. The gastroenterology service was not consulted because she stabilized by early morning.

The cardiac surgeon and intensive care physician felt this stabilization provided a window of opportunity for operative repair of her Type A aortic dissection. Prior to the operation her hemoglobin was 121 g/L, white blood cell count 7.8 x 10^9/L, platelets 129 x 10^9/L, INR 1.2, PTT 30, fibrinogen 1.91 g/L, lactate 1.1 mmol/L, ALT 40 U/L, AST 61 U/L, sodium 145 mmol/L, potassium 3.4 mmol/L, chloride 114 mmol/L, bicarbonate 19 mmol/L, urea 7.6 mmol/L and creatinine 48 µmol/L. She was aggressively warmed and resuscitated throughout the night with one unit pooled platelets and two units pooled cryoprecipitate, four units of fresh frozen plasma, and two units of packed red blood cells. The gastroenterology service was not consulted because she stabilized by early morning.

At surgery, an intima tear in the posterior middle portion of the ascending aorta was noted, and the hemi-arch was replaced with a 29-mm Gel weave conduit. The root of the aortic valve was frail and required repair with BioGlue. She tolerated the surgery well and there was no post-operative bleeding. Her platelet count dropped progressively over the next 48 hours to a nadir of 26 x 10^9/L; her INR and PTT were normal, and her fibrinogen level was >1 g/L. A follow-up magnetic resonance imaging (MRI) and magnetic resonance angiogram (MRA) of the head and neck revealed multiple small acute infarcts in the right cerebral hemisphere; both carotid vessel bifurcations had normal appearances with no evidence of carotid or vertebral artery dissection. The carotid occlusion noted earlier on CT scan was now patent. She continued to do well with resolution of her disseminated intravascular coagulation (DIC). She was transferred to the ward, and later to her home hospital, for continuing physiotherapy and rehabilitation of her mild left-sided motor deficit. Her medications on transfer were metoprolol 50 mg twice daily and lansoprazole 20 mg once daily.

Discussion
Thoracic aortic aneurysms can cause cerebral infarction by dissecting to the carotid vessels, leading to perfusion deficits from the dissection and from branch vessel occlusion.1,2 The dissection can also extend inferiorly, involving the descending aorta and the superior mesentery artery, leading to bowel infarction and gastrointestinal bleeding.3 In uncomplicated arterial aneurysms and in spontaneous aortic dissection, the
extensive thrombus formation can trigger DIC, with a reported incidence of 4%. In this report we describe a patient who presented with all three complications — stroke, DIC, and gastrointestinal hemorrhage, resulting from Type A aortic dissection.

In a post-mortem study in 1819, Laennec described two significant factors that can cause aortic dissection: 1) a predisposing structural weakness of the aortic wall and 2) an initiating event. This is usually in the intima, but based on autopsy studies, it occurs in the media in 4% of the cases. The common causes of aortic structural weaknesses are arterial hypertension, aortic dilatation, bicuspid aortic valve, coarctation of the aorta, aortic arch hypoplasia, Marfan’s syndrome, pregnancy, and aging. The initiating event can be a sudden increase in either systemic vascular resistance or systemic vascular pressure, or trauma to the aorta. However, the understanding of aortic aneurysm formation and dissection has evolved since Laennec, and we now know the genetic implications of extracellular matrix proteins, smooth muscle cells, and growth factors as they relate to aortic wall homeostasis. These predeterminants are also influenced by smoking, hypertension, and atherosclerosis, which result in an inflammatory response coupled with an accelerated proteolytic cascade that disrupts elastin and collagen in the arterial wall. Our patient had hypertension and smoked a half-pack of cigarettes per day for 40 years; these factors may have contributed to her aortic structural weakness. We believe a sudden increase in her systemic vascular pressure may have led to her aortic dissection.

DIC is a life-threatening syndrome associated with several clinical conditions, including sepsis, cancer, severe burns, prosthetic devices, liver disease, trauma-related injuries, obstetric complications, transplant rejection, transfusion reactions, and vascular abnormalities. The association of DIC with aortic aneurysms was first reported by Fine and colleagues in 1967. This association is clinically relevant from two perspectives: first, it represents a source of perioperative complication in a major surgical procedure; and second, it provides a lesson to consider vascular abnormalities in the differential diagnosis of DIC, since its early detection can potentially reduce significant morbidity and mortality.

Acute ischemic stroke secondary to vessel occlusion from an aneurismal dissection is an uncommon presentation, but it has been described secondary to DIC. In a case series of 272 patients with aortic dissection, one patient presented with a stroke, and seven patients suffered preoperative stroke. Only one reported DIC case resulted in brain and spinal cord infarction. Aortic dissections can lead to other vascular structural complications, such as superior mesenteric artery (SMA) dissection with bowel infarction, and lower gastrointestinal bleed: our patient’s SMA was not dissected and continued to be fed by the true lumen of the aorta. Our patient’s upper gastrointestinal bleeding appeared to be associated with her consumptive coagulopathy from the aortic dissection. This bleeding could have been further exacerbated by the non-steroidal anti-inflammatory drug use and the hypothermia developed during transport to the Cardiac Surgery Recovery Unit.

**Conclusion**

Our case illustrates that one should consider the possibility of a dissecting aortic aneurysm in any patient presenting with neurological symptoms and acute gastrointestinal bleed, with or without DIC. DIC induced from an aneurysm is a rare presentation that is most frequently diagnosed perioperatively; however, it can be a useful clue to the diagnosis of a vascular abnormality. The preoperative incidence of DIC associated with an aneurismal dissection is estimated to be 3–4% and is managed in a similar fashion, that is, by treating the underlying disorder.

**References**

A 72-year-old woman presented with a one-week history of fever, non-productive cough, and three unwitnessed syncopal episodes, with no other associated symptoms. She had poor recollection of the episodes, but denied urinary/fecal incontinence, tongue biting, numbness, parasthesias, or weakness.

Her medical history was significant for hypertension, dyslipidemia, leg ulcers, psoriasis, stress incontinence, and gastroesophageal reflux disease. She did not have diabetes, coronary artery disease, or peripheral vascular disease. Her medications included Atorvastatin, Enalapril, Hydrochlorothiazide, Lansoprazole, and Calcium. She did not smoke or drink.

On examination, her vital signs were stable and she was afebrile. Chest auscultation revealed crackles in the right lower base. She developed an erythematous, lacy sharply demarcated rash on her left hand that lasted for 3–4 hours after her blood pressure was taken on the same arm (Figures 1 and 2). Blood tests were unremarkable and included glucose, complete blood count, electrolytes, urea, creatinine, liver function tests, and coagulation studies.

Discussion

The case illustrates a well-demarcated, non-blanching erythematous rash arising after BP measurement. This is presumed to be the result of mechanical trauma to the dermal capillaries from non-invasive blood pressure monitoring. Although our patient was not diabetic, this phenomenon has been most often described in patients with underlying diabetic retinopathy and after non-invasive ambulatory blood pressure monitoring. In one study, 68% of 72 diabetic patients compared to 35% age- and sex-matched non-diabetic control subjects had a positive Rumpel-Leede sign. High prevalence of hypertension and increased capillary fragility in diabetic patients may predispose them to Rumpel-Leede phenomenon, which is positively correlated with duration of diabetes and the presence of microvascular complications of diabetes, including neuropathy, retinopathy, and nephropathy.

Rumpel-Leede phenomenon is not specific to diabetes and can occur with other causes of increased capillary fragility, such as Ehlers-Danlos syndrome and thrombocytopenia. The exact mechanism is not known, but increased capillary fragility in the presence of high intravascular pressure created by the sphygmomanometer cuff would be a plausible mechanism. In our nondiabetic patient, the likely cause of the Rumpel-Leede sign was the fragility of her small blood vessels as skin and blood vessels are more fragile in old age. The prominent demarcation of the Rumpel-Leede sign at the wrist rather than forearm (well below the area of the BP cuff, where one might expect to see it) was likely due to the tourniquet causing intravascular pressure, greatest in the distal, dependent areas.

There were no clinical implications caused by this condition in our particular patient. However, a lack of awareness of this condition and its causes can lead to unnecessary investigations.

References

Join us in PEI for the CSIM Annual Meeting 2015!

The focus of this year’s meeting is “GIM: Excellence in Hospital and Community Care.”

As general internists responsible for delivering complex care to patients spanning the inpatient and ambulatory settings, issues of healthcare quality, patient safety, and stewardship of finite resources are core to our everyday practice. Throughout this year’s meeting, we will hear from internationally recognized researchers, educators, and clinicians. These presentations will open a dialogue about how we as general internists can lead in incorporating the best available evidence to deliver high quality care to our patients.

October 14-17, 2015
Delta Prince Edward Charlottetown, PEI

Visit www.csim.ca for program, registration and hotel information.