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**Low rate of minor hypoglycemia observed**

- Episodes of minor hypoglycemia per patient per year: Victoza® 1.2 mg: 0.143; Victoza® 1.8 mg: 0.154; sitagliptin 100 mg: 0.137 (all in combination with MET)

Victoza® 1.2 mg: 50%; Victoza® 1.8 mg: 63%; sitagliptin 100 mg: 27% (all in combination with MET); p<0.0001 vs. sitagliptin + MET, p=0.0119 for Victoza® 1.8 mg + MET vs. Victoza® 1.2 mg + MET.

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CONTENTS

Message from the President
84 We Are General Internists
Benjamin Chen MD

Mot du président
85 Nous sommes des internistes généralistes
Benjamin Chen, MD

CSIM Announcement
87 Osler Award Winners 2013/
Lauréats des Prix Osler 2013

Health Promotion, Policy, and Advocacy
89 Restricting Marketing of Unhealthy Foods: Should General Internists Engage?
Norm Campbell MD, Tara Duhaney MHSc

Clinical Medicine
91 Medical Problems in Pregnant Women
Michèle Mahone MSc MD, Nadine Sauvé MD

97 Acute Care SINS: Surgical Insights for the Non-surgeon
Chapter 5: Small Bowel SINS
Rachel G. Khadaroo MD PhD, Kamran Fathimani MD, Peter G. Brindley MD

Original Observations and Research
101 Education Research Productivity of Academic Physicians
Liam Rourke PhD, Dale Storie MA MLIS

Case Reports
105 Scurvy in the Context of End-Stage Liver Disease
Amirrah Ajunrarai MSc, Michael Hackett CD CCPA, Kumanan Wilson MD MSc

107 Commentary: Lessons from History: Still Relevant in the “Information Age”
Peter G. Brindley MD

Professional Development/ Employment Opportunities
96 Sea Courses
110 Interior Health

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We Are General Internists

Benjamin Chen MD

About the Author
Benjamin Chen is a general internist in Napanee, Ontario, and an adjunct associate professor in the Division of General Internal Medicine at Queen’s University, in Kingston, Ontario. He does not have an RCPSC Certificate in GIM. Correspondence may be directed to Benjamin.Chen@generalinternist.ca.

The Canadian Society of Internal Medicine (CSIM) achieved a key milestone in December 2010: Royal College of Physicians and Surgeons of Canada (RCPSC) recognition of general internal medicine (GIM) as a distinct subspecialty. Much more work has been done since then, such as developing objectives of training for a new 2-year GIM training program, and additional challenges remain, for example, upgrading training programs across the country, developing a GIM examination for the new RCPSC Certificate in GIM, and attracting the best and brightest into careers in GIM. But it is clear that GIM in Canada has entered an exciting new era. Regular readers of this journal will appreciate that these successes have been through the tireless efforts of many general internists over many years.

With RCPSC recognition comes the opportunity for practising general internists to obtain an additional RCPSC certificate in GIM, through the PER-sub and GIM examination processes. The first GIM examination will be held in September 2014, with subsequent examinations offered yearly. A detailed Frequently Asked Questions page explaining these steps is posted on the redesigned CSIM.ca website, and I encourage everyone to consider the opportunity.

For those practising general internists who choose not to write the GIM examination at this time, it is clear that we may not claim to be RCPSC-certified in GIM. Nevertheless, several have asked whether we may continue to call ourselves general internists. In considering this issue, CSIM – as the national specialty society for internal medicine and for GIM – recently endorsed operational definitions of GIM and of general internist that are based solely on one’s competencies and scope of practice:

General Internal Medicine is a subspecialty of Internal Medicine which embraces the values of generalism, is aligned with population needs, and promotes the practitioners’ ability to adapt their practice profile when population needs change.

General Internists are prepared to diagnose and manage patients with common and emergency internal medicine conditions, and are able to do so when the individual has multiple conditions and with limited access to other subspecialists. General Internists provide comprehensive care of the adult patient in an integrated fashion as opposed to an organ-centred or disease-centred approach. They are prepared to maintain stability of patients with multisystem disorders over the long-term or during physiological stresses such as during pregnancy or the peri-operative period.

General Internists advocate for their individual patients as well as for all patients within complex healthcare delivery systems, by aiming to optimize and not maximize...
care, including prevention of other conditions. General Internists recognize that the practice of medicine is tightly linked to the art and science of health care delivery and, by virtue of their pivotal role, are uniquely placed to engage in quality improvement, patient safety, and healthcare systems initiatives.

Or to paraphrase the duck test: if an internist considers himself/herself a general internist, practises like a general internist, cares for patients like a general internist, and communicates like a general internist, then he/she must be a general internist. CSIM has been celebrating general internists for decades, notwithstanding a lack of RCPSC recognition or certification, and CSIM will continue to represent all general internists in Canada.

To continue this conversation, please write to this journal, contribute or comment on the new CSIM.ca blog, or contact me directly at Benjamin.Chen@generalinternist.ca.

Nous sommes des internistes généralistes

Benjamin Chen MD

L’auteur
Interniste généraliste, Benjamin Chen exerce sa profession à Napanee en Ontario; il enseigne également à titre de professeur agrégé adjoint pour le compte de la division de médecine interne générale de l’Université Queen’s à Kingston en Ontario. Il n’est pas titulaire du certificat de médecine interne générale délivré par le Collège royal des médecins et chirurgiens du Canada. Prière d’adresser la correspondance à Benjamin.Chen@generalinternist.ca.

La Société canadienne de médecine interne (SCMI) a franchi une étape marquante en décembre 2010 : le Collège royal des médecins et chirurgiens du Canada (CRMCC) a décrété que la médecine interne générale était une surspécialité à part entière. Beaucoup a été fait depuis lors, notamment la détermination des objectifs de la formation de ce nouveau programme d’études de deux ans en médecine interne générale, quoique des défis demeurent, dont la mise à niveau des programmes de formation au pays, la conception de l’examen de médecine interne générale menant au nouveau certificat délivré par le CRMCC et la promotion de la discipline dans l’espoir de recruter des médecins talentueux. Nul doute qu’une nouvelle ère prometteuse attend la médecine interne générale au Canada. Le lecteur de la revue sait bien que la discipline doit cette réussite aux nombreux internistes généralistes qui n’ont rien ménagé pendant des années pour défendre cette cause.

La surspécialité étant désormais reconnue par le CRMCC, les internistes généralistes ont la possibilité d’obtenir le certificat de médecine interne générale en empruntant la Route d’évaluation par la pratique pour les surspécialistes (REP-sur) et en se présentant à l’examen de médecine interne générale. Le premier examen aura lieu en septembre 2014, et une séance d’examen se tiendra chaque année par la suite. Le site Web rénové de la SCMI présente une foire aux questions précisant la démarche d’obtention du certificat. Le certificat est un atout, c’est à bien y penser!

Bien entendu, l’interniste généraliste qui ne souhaite pas se présenter à l’examen de médecine interne générale pour le moment ne peut se dire titulaire du certificat délivré par le CRMCC. Néanmoins, plusieurs se demandent si nous pouvons nous dire des internistes généralistes. À ce propos, la SCMI – société d’envergure pancanadienne représentant la médecine interne et la médecine interne générale – a adopté dernièrement une définition opérationnelle de la médecine interne générale et d’interniste généraliste en fonction exclusivement des compétences professionnelles et du champ de pratique :

La médecine interne générale est une surspécialité de la médecine interne qui intègre les valeurs du généralisme, est sensible aux besoins de la population et préconise l’adaptation du mode de pratique du médecin lorsque les besoins de la population changent.
Les internistes généralistes sont préparés à diagnostiquer et à prendre en charge les problèmes de santé courants ou émergents qui relèvent de la médecine interne, que le problème se présente seul ou en compagnie d’autres troubles de santé et lorsque l’accès aux autres surspécialités est limité. Les internistes généralistes prodiguent des soins complets à des patients adultes dans une perspective globale par opposition à la démarche diagnostique et thérapeutique axée sur un organe ou une maladie. Ils sont en mesure de maintenir la stabilité des patients présentant des troubles multiples à long terme ou durant une période de stress physiologique, telles la grossesse ou la période périopératoire.

Les internistes généralistes se portent à la défense de leurs patients et de tous les patients au sein des systèmes complexes de prestation de soins de santé, dans l’optique d’optimiser les soins, plutôt que de les maximiser, et de prévenir d’autres maladies. Ils savent que l’exercice de la médecine est intimement lié à l’art et à la science de la prestation de soins de santé et que, en raison de leur rôle central, ils occupent une place distinctive dans l'amélioration de la qualité des services de santé, la promotion de la sécurité des patients et l’innovation dans les systèmes de santé.

Pour paraphraser le « test du canard » (si ça ressemble à un canard, si ça nage comme un canard et si ça cancane comme un canard, c’est qu’il s’agit sans doute d’un canard), si un interniste se considère comme un interniste généraliste, exerce sa profession comme un interniste généraliste, soigne ses patients comme un interniste généraliste et s’exprime et communique comme un interniste généraliste, c’est qu’il est fort probablement un interniste généraliste. La SCMI fait valoir l’interniste généraliste, sa place et son importance, depuis bien longtemps, en l’absence de reconnaissance de la surspécialité et de certificat, et elle continuera de représenter tous les internistes généralistes du Canada.

Pour poursuivre la discussion, adressez un mot à la revue, allez-y de vos observations sur le nouveau blogue csim.ca ou communiquez avec moi à Benjamin.Chen@generalinternist.ca.
Osler Award Winners 2013/
Lauréats des Prix Osler 2013

Dr. Sharon Card is currently chair of the GIM Subspecialty Committee of the Royal College of Physicians and Surgeons. Dr. Card trained at Queen’s University, in Kingston, Ontario, and completed her fellowship training at Dalhousie University, in Halifax, Nova Scotia. She was head of the Division of Internal Medicine at the University of Saskatchewan, Saskatoon, from 2003 to 2007.

Dr. Card has been on numerous committees associated with the University of Saskatchewan for the past 17 years. She has received many awards and honours in recognition of her abilities as a teacher, clinician, and educator, but it is her commitment to her profession that most people recognize her for. She has demonstrated exceptional leadership skill in starting the GIM Program Director’s Committee in 2001, and tireless efforts in consensus building to bring GIM specialty status within the Royal College.

Dr. Card has been a remarkable supporter of CSIM, and a regular examiner for the Royal College. She is married, with one son.

Dr. James Kitchens is currently a general internist at St. Michael’s Hospital, University of Toronto, in Toronto, Ontario. Dr. Kitchens trained in North Carolina and took his IM fellowship at McGill University in Montreal. He moved to St. Michael’s Hospital in 1993, and was head of the Division of General Internal Medicine for the next 12 years. Dr. Kitchens spearheaded numerous educational innovations, including the teaching of evidence-based medicine, the development of ambulatory clinics, and the creation of a highly regarded annual retreat at St. Michael’s Hospital. Students regard him as an excellent clinician and teacher, known for his wisdom, patience, and encouragement. He has mentored several nationally acclaimed researchers and teachers, and helped build a faculty of clinician scientists at U of T.

Dr. Kitchens has an interest in the humanities. He has supported and promoted the “Reflections in Medicine” series, which addresses difficult topics in medicine using poetry and prose. He is married with two children, and enjoys photography, travel, and hiking.

La Dr Sharon Card préside le Comité de la surspécialité de la médecine interne générale du Collège royal des médecins et chirurgiens du Canada.

Diplômée en médecine de l’Université Queen’s à Kingston en Ontario, elle poursuit des études postdoctorales à l’Université Dalhousie à Halifax en Nouvelle-Écosse. De 2003 à 2007, elle dirige la division de médecine interne à l’Université de la Saskatchewan à Saskatoon.

Dans les 17 dernières années, elle siège à de nombreux comités de l’Université de la Saskatchewan. Plusieurs prix et distinctions soulignent ses talents dans l’enseignement, la pratique clinique, l’éducation, mais elle est renommée surtout pour son excellence et sa détermination dans l’exercice et l’avancement de sa profession. La mise sur pied du comité des directeurs de programme de médecine interne générale en 2001 illustre son art consummé de la direction et sa capacité à susciter le consensus tout au long de la démarche de reconnaissance de la médecine interne générale en tant que surspécialité.

La Société canadienne de médecine interne peut compter sur son appui indéfectible, et le Collège royal fait fréquemment appel à elle à titre d’examinatrice. Madame Card est mariée et elle a un fils.

Le Dr James Kitchens exerce la médecine interne générale à l’Hôpital St. Michael affilié à l’Université de Toronto.

Diplômé en médecine de la Caroline du Nord, il effectue sa résidence en médecine interne à l’Université McGill. Il entre en fonction à l’Hôpital St. Michael en 1993 et y dirige le service de médecine interne générale durant 12 ans. Il innove sur plusieurs plans dans le domaine de l’éducation, notamment par l’enseignement de la médecine factuelle, par la mise sur pied de cliniques ambulatoires et par la création de la retraite annuelle de l’hôpital qui a acquis ses lettres de noblesse. Les étudiants voient en lui un clinicien et un enseignant hors pair, réputé pour sa sagesse, sa patience et son soutien. Il s’est fait le mentor de nombreux chercheurs et enseignants dont la renommée s’étend d’un bout à l’autre du pays et il a contribué à la formation d’un solide corps professoral de cliniciens chercheurs à l’Université de Toronto.

Les lettres et les sciences humaines le captivent depuis fort longtemps. Il contribue à la série Reflections in Medicine qui aborde des sujets délicats en médecine par la prose et la poésie. Marié, il a deux enfants; il s’adonne à la photographie et à la randonnée pédestre et il aime voyager.
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Restricting Marketing of Unhealthy Foods: Should General Internists Engage?

Norm Campbell MD, Tara Duhaney MHSc

About the Authors
Norm Campbell (right) is a professor of medicine, community health sciences and physiology and pharmacology at the University of Calgary and is a member of the Libin Cardiovascular Institute, in Calgary, Alberta. Tara Duhaney is a policy director at the Libin Cardiovascular Institute. Correspondence may be directed to ncampbel@ucalgary.ca.

Unhealthy diet is the leading risk for death, years of life lost, and disability, causing an estimated 65,722 deaths and 864,032 life years lost in Canada in 2010. Although the causes of unhealthy diet are complex, unhealthy eating habits start early in life, and unhealthy food and beverage marketing to children is consistently associated with unhealthy dietary behaviours and childhood obesity. Although there have been recommendations from the World Health Organization (WHO) and the United Nations urging countries to restrict such marketing to children as a population strategy to improve diet, the food industry continues to direct millions of marketing dollars to increase the sales and consumption of the very foods that contribute to disease burden. While many countries have heeded the WHO recommendations, in Canada, outside of Quebec, the food industry largely self-regulates its marketing of unhealthy food to children, with no government monitoring or oversight. The result is that Canadian children are extensively exposed to marketing of unhealthy food products that would not be allowed in several other countries.

Should Canadian internists accept that children’s dietary health is being hijacked by an industry that generates profits by selling the very foods that are making our children and future adults unhealthy? Saying enough is enough, the Canadian Society of Internal Medicine and 23 other health and scientific organizations in Canada have called for policies and processes to stop all marketing of unhealthy foods and beverages to children. Individual internists can advocate through local, provincial, and national organizations, and in our communications with provincial and federal politicians. The role of unhealthy foods as the major cause of death and disability needs to be part of undergraduate and continuing education, and other educational opportunities focused on health promotion and disease prevention.

References
As a committed member of the healthcare community, AstraZeneca Canada proudly supports the Canadian Society of Internal Medicine in their efforts to improve the health of Canadian patients across the country.
Medical Problems in Pregnant Women
2012 ACCP Guideline Regarding the Pregnant Patient: A Case-Based Discussion
Michèle Mahone MSc MD, Nadine Sauvé MD

About the Authors
Michèle Mahone (right) is with the Centre Hospitalier Universitaire de l’Université de Montréal (CHUM), in Montreal, Quebec. Nadine Sauvé is with the Centre Hospitalier Universitaire de l’Université de Sherbrooke (CHUS), in Sherbrooke, Quebec. Correspondence may be directed to michele.mahone@umontreal.ca.

Summary
The American College of Chest Physicians (ACCP) published its latest (9th edition) guidelines in February 2012. This document is a valuable reference for all clinicians. In the current article, through the analysis of three clinical cases, the authors review, describe, and analyze the most significant new information from the chapter “VTE, Thrombophilia, Antithrombotic Therapy, and Pregnancy”: (1) strategies for clinicians facing the prescription of a new drug during pregnancy with little available data; (2) recommendations about indications of thromboprophylaxis for asymptomatic thrombophilias; and (3) thrombophilia screening and secondary prevention for placental complications.

Résumé
La 9e édition des lignes directrices du American College of Chest Physicians (ACCP) (Collège américain de médecine thoracique), publiée en février 2012, est un document de référence précieux pour tous les cliniciens. À partir de trois cas cliniques, les auteurs de l’article étudient, décrivent et analysent les nouvelles données du chapitre intitulé « VTE, Thrombophilia, Antithrombotic Therapy, and Pregnancy » (MTEV, thrombophilie, traitements antithrombotiques et grossesse) : 1) stratégies à l’intention des cliniciens qui envisagent de prescrire pendant une grossesse un nouveau médicament malgré le manque de données; 2) recommandations relatives aux indications de thromboprophylaxie pour les cas de thrombophilie asymptomatique; 3) dépistage de la thrombophilie et prévention secondaire des complications placentaires.

Physicians treating pregnant women with venous thromboembolism (VTE), thrombophilia, or placental complications must make clinical decisions despite a paucity of high-quality study data. They often rely on the information published by the American College of Chest Physicians (ACCP). The newest recommendations were published in February 2012: VTE, Thrombophilia, Antithrombotic Therapy, and Pregnancy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed. American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. The methodology of the ACCP 9th guideline uses structured clinical questions, and is updated to January 2010.1

We review and discuss these recommendations as they relate to three complex cases: a pregnant patient taking new antithrombotic oral agents, the management of asymptomatic thrombophilia, and the use of secondary prophylaxis for obstetrical complications.

Case 1: Exposure to New Antithrombotic Oral Agents during Pregnancy
A 32-year-old woman, G1, who is 6 weeks pregnant, is referred in an anxious state. She had a left deep vein thrombosis 4 weeks prior and is currently on rivaroxaban. What should we tell her, and what are the maternal and fetal risks related to that medication?
Organogenesis occurs from the 4th to 12th week of gestation. Although the neural tube develops early, the central nervous system will mature throughout pregnancy. When evaluating the risk of malformation with exposure to medications, clinicians must take into account the following: (1) that the baseline risk for major malformations is 2–3% in normal pregnancy; (2) that well-known teratogens are rare and cause malformations in fewer than 25% of exposed embryos; (3) the effect of all other medications, illicit drugs, alcohol, and smoking; and (4) the presence of confounding variables such as obesity, epilepsy, and congenital cardiac disease. 1–3

In our opinion, clinical decision making based solely on the US Food and Drug Administration (FDA) classification for medication risk in pregnancy should be discouraged. This classification can be misleading, does not always take into account new data on old drugs, and can give reassuring labels to new drugs based on animal data (and despite little human data). 1,2,4 Table 1 includes alternative tools to determine the safety of drugs in pregnancy and lactation.

### New Oral Anticoagulant Use during Pregnancy

Rivaroxaban is an anti-Xa inhibitor. There are no human safety data for its use during pregnancy. In animal models, it has been demonstrated to cross the placenta at about 20% of maternal serum concentration and is excreted in breast milk (at about 2%). In animals, it is associated with fetal toxicity, an increased prevalence of congenital malformations, and placental anomalies. 5,6

**ACCP Recommendation**

Pregnant and breastfeeding women should avoid “the use of oral direct thrombin (e.g., dabigatran) and anti-Xa (e.g., rivaroxaban, apixaban) inhibitors (Grade 1C).” 91

### Discussion

We agree with the recommendation that new oral antithrombotic agents should not be prescribed to women of child-bearing age due to the lack of data, 5,6 particularly because other therapeutic options exist. 7 However, fewer than 50% of pregnancies are planned, and therefore the risk of exposure to these new drugs is real. 1,2 If anticoagulant drugs are absolutely necessary, then contraception and adequate counselling are mandatory. If a patient is subsequently found to be pregnant, she should continue her antithrombotic drug temporarily and consult her physician immediately to change to a safer therapy. The risk of recurrence of a thrombotic event might be worse than the theoretical teratogenic risk.

### Case Conclusion

A frank discussion concerning the absence of data in human pregnancy is essential. However, since the medication was stopped very early in pregnancy, and since true teratogens are rare, the overall risk seems low. While she is considering her options, the rivaroxaban should be stopped immediately, and low molecular weight heparin (LMWH) started at therapeutic dose. Side effects of LMWH should be discussed with the patient. The risk of bleeding is <1%. 8 Risk of osteoporosis at therapeutic dose is probably lower than with unfractionated heparin (UFH), 8,9 and adverse skin reactions are generally benign. 10,11 The risk of developing heparin-induced thrombocytopenia is low for obstetrics patients (<0.1%); therefore, platelet monitoring is not recommended. 10,12

### Case 2: Asymptomatic Thrombophilia in Pregnancy

A 28-year-old woman, G1 and 6 weeks pregnant, is seen in your office. She has a family history of VTE and is found to have a deficiency in antithrombin (AT). She has no personal history of thrombosis. What is her risk of thrombosis, and should she receive prophylaxis during the pregnancy and/or postpartum?

**Background: Risk of VTE during Pregnancy in Women with Thrombophilia**

Familial history of VTE is associated with two- to fourfold increased risk of VTE compared with the general population. 13

---

**Table 1. Further Resources**

<table>
<thead>
<tr>
<th>PubMed search</th>
</tr>
</thead>
<tbody>
<tr>
<td>Briggs et al. (2008) Drugs in Pregnancy and Lactation. 42</td>
</tr>
<tr>
<td>Motherisk (Toronto) <a href="http://www.motherisk.org">www.motherisk.org</a></td>
</tr>
<tr>
<td>Toxnet.</td>
</tr>
</tbody>
</table>

**Table 2. Risk of Thromboembolic Disease in Asymptomatic Thrombophilia**

<table>
<thead>
<tr>
<th>Thrombophilia</th>
<th>Estimated Absolute Risk (%) in Antepartum and Postpartum (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antithrombin deficiency</td>
<td>3.0 (0.08–15.8)</td>
</tr>
<tr>
<td>Protein C deficiency</td>
<td>1.7 (0.4–8.9)</td>
</tr>
<tr>
<td>Protein S deficiency</td>
<td>6.6 (2.2–14.7)</td>
</tr>
<tr>
<td>Factor V Leiden heterozygous</td>
<td>3.1 (2.1–4.6)</td>
</tr>
<tr>
<td>Prothrombin heterozygous</td>
<td>2.6 (0.9–5.6)</td>
</tr>
<tr>
<td>Leiden homozygous</td>
<td>14.0 (6.3–25.8)</td>
</tr>
</tbody>
</table>

Adapted from Bates et al. 7
Table 2 summarizes the estimates of absolute risk of antepartum and postpartum VTE in women with thrombophilia and a family history.\textsuperscript{7,14,15} The presence of factor V Leiden homozygous state is associated with the highest risk. ACCP recommendations are outlined in Table 3.

The family history and the presence of high-risk thrombophilias (factor V Leiden homozygous and prothrombin variant homozygous) warrant prophylaxis during pregnancy (grade 2B).\textsuperscript{7}

For low-risk thrombophilias without family history, antepartum and postpartum clinical surveillance is suggested (grade 2C).\textsuperscript{7}

**Discussion**

In addition to the ACCP recommendations, individual VTE risk should be taken into account when evaluating prophylaxis for these women. For example, are there other VTE risk factors such as multiple thrombophilias, obesity, immobility, varicosities, advanced maternal age, or smoking?\textsuperscript{21,4,16–23} While women with a double-heterozygous mutation are not specifically addressed in the guidelines, these patients are often treated during pregnancy (grade 2B).\textsuperscript{7}

As antithrombin (AT) deficiency is exceedingly rare, the impact of asymptomatic carrier state in pregnancy has not been well studied. Earlier studies of AT deficiency in pregnant women suggested an incidence of up to 35% VTE in pregnancy and postpartum.\textsuperscript{25,26} However, these older studies had methodological flaws: a majority of events were not confirmed by objective testing, and inclusion of superficial phlebitis likely inflated the incidence. The study populations also included symptomatic women and compared pregnant women with non-pregnant women.\textsuperscript{25,26} A recent systematic review of asymptomatic women with AT and pregnancy (irrespective of family history) was consistent with the recommendations in the 9th ACCP guideline. In this review, 54 women with 124 pregnancies were identified with an estimated absolute-risk of VTE of 1.63% (95% CI 0.29–9.00) in cohort studies and 0.67% (95% CI 0.16–2.80) in case-control studies.\textsuperscript{27}

However, several factors mean the ACCP recommendations may be inadequate. These include the severity and atypical thrombotic sites that can occur with AT deficiency; the small number of patients and pregnancies in the literature; the large confidence intervals and the low quality of the studies. Therefore, we would recommend prophylaxis.

**Case Conclusion**

The ACCP states that since our patient is asymptomatic for thromboembolic disease, has a family history of VTE, and does not have a high risk thrombophilia, she should not receive thromboprophylaxis in antepartum. After discussing with our patient, we would recommend prophylaxis antepartum and postpartum, since the risk of bleeding is low and our confidence in the strength of the evidence is limited.

**Case 3: Thrombophilia and Placental Complications**

A 29-year-old woman, G2P1, is 6 weeks pregnant. Her previous pregnancy (2 years earlier) was complicated by severe pre-eclampsia with intrauterine growth restriction (IUGR) at 30 weeks. Her obstetrician is asking you if she should be screened for thrombophilia and if she should have a secondary prophylaxis to prevent recurrence.

**Thrombophilia: Background**

The evidence associating thrombophilia with placental complications (recurrent first trimester losses [more than three episodes], late fetal demise, pre-eclampsia, placental abruption, and IUGR) is of low level and therefore controversial. Although initial case-control studies showed an association,\textsuperscript{14} recent prospective cohort studies\textsuperscript{28} did not (Table 4).

**ACCP Recommendations**

For women with recurrent early pregnancy loss (three or more
consecutive miscarriages before 10 weeks of pregnancy), screening for antiphospholipid antibodies (APLA) is recommended (grade 1B). For all other pregnancy complications, screening for inherited thrombophilia is not suggested (grade 2C).7

### Discussion

There is no ACCP recommendation either for or against APLA screening in late pregnancy loss, pre-eclampsia, or IUGR (with concomitant APLA).29–31 This may be due to insufficient proof that any current therapy changes outcome. Although controversial, our practice is to screen these women for APLA and then, if present, recommend antepartum and postpartum prophylaxis with LMWH.

The ACCP guidelines do not recommend screening for inherited thrombophilias in women with obstetrical complications. This is because there is only a weak association at best, and LMWH efficiency does not seem to be correlated with thrombophilia status (Table 5).

### Prevention of Placental Complications: Background

Aspirin (acetylsalicylic acid, or ASA) has been shown to reduce the risk of developing pre-eclampsia by 17% overall,32 and by 53% if started before 16 weeks.33 For women at high risk, the number needed to treat (NNT) is <28.33 High risk is defined by previous pre-eclampsia, diabetes mellitus type 1 or 2, chronic renal disease, chronic hypertension, autoimmune disease, a body mass index (BMI) ≥30, twin gestation, or a family history of pre-eclampsia.32

LMWH use has generated a lot of interest and research in the past 10 years. Accordingly, we now have good data showing no benefit for recurrent early pregnancy losses with LMWH (outside of APLA).34,35 Other placental complications have often been studied together due to their common pathophysiology (see Table 5).

### Table 4. Association of Thrombophilia and Obstetrical Complications

<table>
<thead>
<tr>
<th>Thrombophilia</th>
<th>Recurrent Loss T1</th>
<th>Late Loss</th>
<th>Pre-eclampsia</th>
<th>Abruptio</th>
<th>IUGR</th>
</tr>
</thead>
<tbody>
<tr>
<td>FVL heterozygous28</td>
<td>⊕−/−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>Prothrombin gene mutation28</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>−</td>
</tr>
<tr>
<td>AT deficiency14</td>
<td>No data</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>No data</td>
</tr>
<tr>
<td>Protein C deficiency14</td>
<td>No data</td>
<td>−</td>
<td>−</td>
<td>−</td>
<td>No data</td>
</tr>
<tr>
<td>Protein S deficiency14</td>
<td>No data</td>
<td>+</td>
<td>−</td>
<td>−</td>
<td>No data</td>
</tr>
<tr>
<td>APLA29–31</td>
<td>+</td>
<td>+/−</td>
<td>+</td>
<td>+/−</td>
<td>+/−</td>
</tr>
</tbody>
</table>

APLA = antiphospholipid antibodies; AT = antithrombin; FVL = factor V Leiden; IUGR = intrauterine growth restriction; + = positive association; +/− = weak association; − = no association.

### Table 5. Secondary Prevention of Obstetrical Complications with LMWH

<table>
<thead>
<tr>
<th>Studies</th>
<th>#</th>
<th>Inclusion Criteria</th>
<th>Thrombophilia</th>
<th>Interventions</th>
<th>Outcomes</th>
<th>NNT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mello et al., 200536</td>
<td>80</td>
<td>PE and ACE GG genotype</td>
<td>No</td>
<td>Dalteparin 5,000 mg vs. open label</td>
<td>PE 7.3% vs. 28.2%</td>
<td>5</td>
</tr>
<tr>
<td>Rey et al., 200937</td>
<td>116</td>
<td>Severe PE &lt;35 wk, IUGR, abruptio, fetal death</td>
<td>No</td>
<td>Dalteparin 5,000 mg vs. open label (ASA in &gt;80%)</td>
<td>Severe PE, IUGR, abruptio, fetal death 5.5% vs. 23.6%</td>
<td>5</td>
</tr>
<tr>
<td>NOH-AP, 201038</td>
<td>160</td>
<td>Abruptio</td>
<td>No</td>
<td>Enoxaparin 40 mg vs. open label</td>
<td>PE, abruptio, IUGR, fetal death 12% vs. 31.3%</td>
<td>5</td>
</tr>
<tr>
<td>NOH-PE, 201139</td>
<td>224</td>
<td>Severe PE</td>
<td>No</td>
<td>Enoxaparin 40 mg vs. open label (ASA in 100%)</td>
<td>PE, abruptio, IUGR, fetal death 8.9% vs. 25%</td>
<td>7</td>
</tr>
<tr>
<td>FRUIT-RCT, 201240</td>
<td>139</td>
<td>Before 34 wk: severe PE or SGA</td>
<td>Yes</td>
<td>Dalteparin 5,000 mg + ASA 80 mg vs. ASA 80 mg</td>
<td>PE &lt;34 wk 0% vs. 8.7% Total PE 18.6% vs. 21.7%, NS</td>
<td>12</td>
</tr>
<tr>
<td>HAPPY, 201241</td>
<td>128</td>
<td>Fetal death, mild PE, severe PE, HELLP, IUGR, (70 vs. 72%)</td>
<td>No*</td>
<td>Nadroparin 3,800 IU vs. medical surveillance</td>
<td>PE, eclampsia, HELLP, IUGR, abruptio 21% vs. 18%, NS</td>
<td>32</td>
</tr>
</tbody>
</table>

ACE GG = angiotensin I-converting enzyme GG phenotype; ASA = acetylsalicylic acid; HELLP = hemolysis, elevated liver enzymes and low platelet; IUGR = intrauterine growth restriction; NNT = number needed to treat; NS = not significant; PE = pre-eclampsia; SGA = small-for-gestational age.

*Screening for thrombophilia was done after randomization (around 60% was associated with low protein S level).
**ACCP Recommendations**

Low-dose aspirin starting in the second trimester is recommended throughout the pregnancy for women at risk of pre-eclampsia (grade 1B).

Low-dose aspirin plus prophylactic/intermediate-dose UFH or LMWH is recommended for APLA syndrome based on recurrent early pregnancy losses (grade 1B). Without APLA or thrombophilia, the recommendation is against antithrombotic prophylaxis (grade 1B).

For women with inherited thrombophilia and a history of pregnancy complications, the suggestion is not to use antithrombotic prophylaxis (grade 2C).

**Discussion**

Since 2010, there have been four more randomized controlled trials (therefore not available when the ACCP guidelines were written). These trials examined secondary prevention of obstetrical complication with LMWH in women with or without thrombophilia (see Table 5). The data are exciting, with most studies showing a reduction of severe outcomes associated with use of LMWH.

**Case Conclusion**

In this case, we would screen the patient only for APLA. Low-dose ASA would be recommended. Prophylactic LMWH would also be suggested, based on preliminary but accumulating evidence showing possible associated benefits. The risks, side effects, and cost of medication need to be discussed.

**Conclusion**

Women of child-bearing age and receiving anticoagulation treatment should also receive adequate contraception. With no human data, and uncertainty about animal studies in pregnancy and lactation, we believe that the new oral direct thrombin and anti-Xa inhibitors should be avoided.

VTE prevention in asymptomatic women with thrombophilia is still controversial. Risk assessment should take into account patient preference and other risk factors. Family history is an important consideration. Although antithrombin deficiency does not seem to be associated with a higher rate of VTE in pregnancy, we believe that cautious thromboprophylaxis is still warranted, until stronger evidence argues otherwise.

We believe that there is no indication to screen for inherited thrombophilia in placental complications. ASA is the only recommended therapy for secondary prevention of pre-eclampsia. Although the role of LMWH treatment seems to be promising, no definite recommendation can yet be made.

**References**

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Acute Care SINS: Surgical Insights for the Non-surgeon
Chapter 5: Small Bowel SINS
Rachel G. Khadaroo MD PhD, Kamran Fathimani MD, Peter G. Brindley MD

About the Authors
Rachel Khadaroo and Peter Brindley are members of the Division of Critical Care Medicine, and Rachel Khadaroo and Kamran Fathimani (far right) are members of the Division of General Surgery, 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 5 of this series, the authors discuss the small bowel — its anatomy, its obstruction, and small bowel/mesenteric ischemia.

Anatomy
The small bowel is divided into duodenum (approximately 30 cm), jejunum (approximately 300 cm), and ileum (approximately 100 cm). The duodenum is divided into four parts: the first part or bulb; the second part or descending portion; the third part or transverse portion; and the fourth part or portion ascending to the ligament of Treitz. Duodenal arterial supply is primarily via the gastroduodenal artery. It courses posterior to the duodenal bulb. Therefore, an ulcer that penetrates the posterior wall of the first part of the duodenum can cause massive hemorrhage. This may necessitate surgery (see Chapter 4: Stomach SINS¹). When there is <200 cm of small bowel, there is risk of short bowel syndrome (not enough bowel to absorb the nutritional requirements). Therefore, care is taken to save an adequate length following a large or multiple bowel resections.

Small Bowel Obstruction
Etiology
By anatomy:
• Obstruction from extraluminal causes: adhesions, hernias, carcinomas, and abscesses
• Obstruction intrinsic to the bowel wall: primary tumours, hematoma, inflammation, Crohn’s disease
• Intraluminal obstruction: gallstones, enteroliths, foreign bodies, and bezoars

By incidence:
• Adhesions (≈50–75%)
• Hernias (≈10–25%)
• Crohn’s disease (increasing frequency, >5%)
• Neoplasm (<5%)

“Measure thrice, think twice, cut once.”
— Old adage from carpentry …
and just as relevant to surgery
Pathophysiology
- Early stages: increased intestinal motility; may present as diarrhea
- Later stages
  - Small intestine dilates, and motility decreases
  - Failure to pass flatus and feces
  - Extravascular fluid gain and intravascular fluid loss
- Even later stages
  - Vomiting (metabolic alkalosis, hypochloremia, and hypokalemia)
  - Hypotensive shock (dehydration, oliguria, ± hypotension)
  - Sepsis (due to translocation of gut bacteria)

Diagnosis
History and physical:
- Nausea and vomiting, abdominal distension
- Cramping abdominal pain
- Volume depletion: tachycardia, postural blood pressure changes, shock

Radiological and laboratory examinations:
- Abdominal radiography
  - Still the best initial radiological test (Figures 1 and 2)
  - Look for air-fluid levels and small bowel dilation
- Abdominal/pelvic computed tomography (CT)
  - May help differentiate partial from complete obstruction
  - Assists with pinpointing location and cause
  - Useful to identify bowel strangulation
- Barium swallow
  - Can show the level and possible causes of small bowel obstruction (SBO)
  - Not commonly used nowadays
- Enteroclysis
  - Oral insertion of tube into duodenum
  - Instill air and barium directly into the small intestine
- Magnetic resonance imaging (MRI): no better than CT
- Laboratory tests: for assessing volume depletion or ischemia (acidosis, lactate)

Strangulating Obstruction
- Usually implies a closed-loop obstruction; vascular supply compromised – ischemia results
- Immediate surgery to prevent bowel necrosis

Treatment of SBO
Medical:
- Intravenous (IV) fluid replacement (“drip”): if making urine, you should add IV potassium
- Nasogastric tube (“suck”)
  - Reduces aspiration
  - Reduces further distension from swallowed air
Surgical Pearls: Summary of SBO

**Pearl 1: Classification of SBO Etiology**

**Common:**
- Adhesions
- Hernias

**Uncommon:**
- Crohn’s disease
- Small bowel neoplasm
- Meckel’s diverticulum
- Intussusception
- Gallstone ileus
- Congenital adhesions
- Appendicitis
- Cecal cancer
- Small bowel volvulus
- Foreign body

**Pearl 2: Be Alert to Common Causes of SBO**
- Adhesions: during history taking, ask about all previous abdominal surgeries
- Hernias: during physical examination, inspect for abdominal scars and palpate for hernias (both abdomen and groin)

**Pearl 3: Management of SBOs – Adhesions and Hernias**
- Adhesions
  - NPO (nothing by mouth), nasogastric tube, IV fluids, serial abdominal examination and radiography
  - Surgery if peritonitis or failure of medical management
- Hernias: surgical repair of abdominal wall or inguinal hernia

**Pearl 4: Management of SBOs – Uncommon Etiologies**
- SBO despite no previous abdominal surgeries, or abdominal wall hernias, usually means a pathological cause and, therefore, the need for surgery

- Broad-spectrum antibiotics not routinely recommended for uncomplicated SBO
- 60–85% recover from partial SBO with just “suck and drip” therapy
- Serial abdominal radiography

**Surgical:**
- Notify surgery if any of the following occur:
  - Ischemia, shock, increasing white blood cell (WBC) count
  - Complete SBO >24–72 hours
  - Peritonitis
  - Worsening results on serial abdominal radiographs
  - Increasing abdominal pain (with concerns of bowel ischemia)
  - Failure to resolve within 24 hours of medical management: “Never let the sun rise and set on a complete bowel obstruction”
- In short, keep in contact with your friendly surgeon

**Small Bowel Ischemia/Mesenteric Ischemia**
- Rare but life threatening
- All physicians need to know the basics
- Can occur gradually; better tolerated if collateral blood supply forms over time
- Can occur acutely; associated with high mortality

**Three Main Causes**
**Embolus:**
- Common cause of mesenteric ischemia
- Usually clot from cardiac source embolized to superior mesenteric artery (SMA)
- Occlusion usually distal to the SMA origin because the clot lodges in the smaller branches

**Thrombosis:**
- Less common
- Usually from plaque occlusion at the vessel’s origin
- Often less severe due to chronicity (i.e., collaterals exist)

**Acute non-occlusive mesenteric insufficiency (NOMI):**
- Associated with hemodynamic compromise (e.g., sepsis, heart failure)
- Global decrease in blood supply to the bowel

**Clinical Presentation**
- Pain out of proportion to physical examination!
- However, initial presentation may be only mild tenderness, with or without peritonitis
- Tenderness increases over time (therefore, serial examinations are very useful)
- May have blood in stool from sloughing mucosa

**Laboratory Investigations**
- Elevated WBC count
- Metabolic acidosis and lactate (serial electrolytes or arterial blood gases)

**Radiology**
- AXR and CT may show fluid-filled bowel-loops and bowel edema
- Arteriogram or contrast CT may show the arterial cut-off (Remember IV contrast put kidneys at risk)
- Gas in bowel wall (pneumatosis intestinalis) is a late finding: indicates bacterial spread into tissue
• Gas in portal system (portal venous gas) is a very bad sign: indicates bacterial spread into the blood supply

**Differential Diagnosis**
- Acute pancreatitis
- Perforated viscus
- Ruptured aneurysm
- Kidney stone

**General Treatment**
- Requires laparotomy for diagnosis (“peak and shriek”) and treatment (“damage control”)
- Extensive necrosis has poor prognosis
- Necrotic bowel is resected
- Typical to perform a “second-look laparotomy” in 24–48 hours to reassess the bowel

**Specific for Occlusive Mesenteric Disease (Embolus or Thrombosis)**
- Primarily surgical treatment with embolectomy or thrombectomy ± arterial reconstruction
- Alternative therapeutic options during angiography
  - Intra-arterial vasodilators or thrombolytic agents
  - Angioplasty
  - Placement of a vascular stent
  - Embolectomy
- Early discussion with a vascular surgeon on best approach

**Specific for Non-occlusive Mesenteric Insufficiency**
- Usually in patients that have had hemodynamic instability (e.g., patients in intensive care unit or cardiac patients)
- Difficult diagnosis if patient is sedated and intubated
- Signs and symptoms similar to occlusive mesenteric disease

**Treatment:**
- ABCs (airway, breathing, and circulation) and treat underlying cause
- Fluid resuscitate and limit vasoconstrictors
- Antibiotics to combat bacterial translocation
- Surgery for dead gut
- Generally poor prognosis

**Mesenteric Venous Occlusion**
- Associated with medical illnesses
  - Portal hypertension, post-operative state
  - Intra-abdominal inflammation (pancreatitis, inflammatory bowel disease)
  - Hypercoagulable states (neoplasm, protein C and S deficiencies)
- Less acute presentation
- Contrast CT for diagnosis
  - Shows thickened bowel wall
  - Slow passage of IV contrast into the portal system
  - Lack of opacification of the portal vein
- Arteriography may show venous congestion and poor filling of portal system

**Treatment:**
- Hemodynamic support
- Systemic anticoagulation
- Surgery for peritonitis and suspected necrotic bowel: venous thrombectomy is rarely performed (due to poor success rate)
- Good prognosis due to (1) collateral venous drainage and (2) mesenteric veins recanalization

**Chronic Mesenteric Insufficiency**
- Also known as intestinal angina
- Almost always in elderly patients with diffuse atherosclerosis
- Otherwise, suspect celiac artery compression by diaphragm’s median arcuate ligament
- Causes post-prandial abdominal pain (mesenteric supply-demand imbalance)

**Diagnosis:**
- Doppler sonography of proximal superior mesenteric artery and celiac artery
- Arteriography for definitive diagnosis: requires occlusion of at least two of the three major mesenteric arteries
- MRI useful in younger patient to show compression of the proximal celiac artery

**Treatment:**
- Angiographic: balloon angioplasty or stent placement
- Surgery: transaortic endarterectomy or bypass graft
  - From supraceliac artery to the celiac artery and superior mesenteric
  - Or retrograde bypass from infrarenal aorta or iliac artery

**Works Cited or Consulted**
Education Research Productivity of Academic Physicians

Liam Rourke PhD, Dale Storie MA MLIS

About the Authors
Liam Rourke is director of medical education research in the Department of Medicine, and associate professor in the Division of General Internal Medicine, University of Alberta, in Edmonton, Alberta. Dale Storie is a librarian at the University of Alberta. Correspondence may be directed to liam.rourke@gmail.com.

Summary
For several decades, organizations such as the Royal College of Physicians and Surgeons of Canada have encouraged academic physicians to engage in medical education research. The extent to which these efforts have been persuasive is unclear. This article discusses a study whose purpose was to describe changes in the educational research productivity within this group from 1997 to 2010. The authors found that there has been a substantial increase in the publishing reports of medical education research by Canadian academic physicians.

Résumé
Depuis plusieurs décennies, des organismes, dont le Collège royal des médecins et chirurgiens du Canada, encouragent les médecins enseignants à mener des activités de recherche en enseignement de la médecine. Il est difficile de déterminer dans quelle mesure ces efforts ont porté fruit. L'article porte sur une étude décrivant la participation de ces médecins à la recherche entre 1997 et 2010. Selon les auteurs, le nombre de publications par des médecins enseignants canadiens dans le domaine de la recherche en enseignement de la médecine a considérablement augmenté.

For several decades, groups such as the Royal College of Physicians and Surgeons of Canada (RCPSC), the Canadian Association of Medical Education (CAME), and the Association of American Medical Colleges (AAMC) have urged physicians with academic appointments to conduct education research.1–3 They argue that this activity can (1) provide the evidence on which to build an evidence-based educational practice, (2) satisfy institutional expectations for tenure-track faculty to engage in scholarly activity, (3) provide a structured activity through which clinical educators can reflect on their educational practice, and (4) contribute meaningfully to the broader educational discourse. The extent to which this campaign has swayed academic physicians is unclear. The purpose of this study is to describe educational research productivity among Canada’s academic physicians from 1997 to 2010.

Methods
We conducted a bibliometric analysis in which our main metric was the per capita rate of education research productivity of Canada’s academic physicians. One of the two terms in this metric is the number of Canadian academic physicians, which we defined as a member of RCPSC with a tenure-track appointment at one of Canada’s 17 faculties or schools of medicine. We obtained this information from a database maintained by the Association of Faculties of Medicine in Canada’s (AFMC) Office of Research Information Services (ORIS).4
The other term in the metric is the number of reports of medical education research, published in a peer-reviewed forum, whose authorship included a Canadian academic physician was 139 in 1997 and 591 in 2010. The numbers of Canadian academic physicians in those years were, respectively, 8,762 and 11,780. The per capita medical education research productivity of this group therefore rose from 16 per 1,000 in 1997 to 50 per 1,000 in 2010.

Discussion
In 1997, Canadian academic physicians were publishing articles on medical education research at the rate of one peer-reviewed publication for every 63 members. By 2010, the rate had risen to one publication for every 20 members. This increase is substantial.

There are few studies of education research productivity among academic physicians with which to compare our results. Similar types of data have been collected to evaluate programs that develop physicians’ ability to conduct education research, but the efforts are not systematic or rigorous.5–10 A possible comparison is with studies of academic physicians’ clinical research productivity. Several studies have been conducted across a variety of academic settings, and these report productivity values ranging from a high of nearly three studies per physician (who had graduated from a physician-scientist training program11) to a low of one publication for every three physicians (who had received a small, early-career research grant).12–17

The robustness of bibliometric analyses are limited by the quality of the information in the databases on which they draw.18 Our query of the SCOPUS database returned hundreds of articles that did not match our inclusion criteria because they were incorrectly indexed. The AFMC database, though accurately indexed, is built on categories whose broad inclusiveness responds to the needs of medical school administrators more than scientometricians. Among the difficulties stemming from the database’s structure was our inability to restrict our count to academic physicians in clinician-educator tracks.

Nevertheless, the data we have presented can be used for three evaluative projects. First, it is one measure of the impact of the efforts to interest academic physicians in education research. Second, it provides a normative context for assessing the research productivity of faculty members who wish to include education research in their annual review. And, similarly, it provides a context for evaluating programs aimed at developing faculty members’ ability to conduct education research.

References
12. Mahoney M, Verma P, Morantz S. Research productivity among recipients
Indications and clinical use
• Once-daily subcutaneous administration in the treatment of Type 1 or Type 2 diabetes in patients over 17 years of age who require basal (long-acting) insulin for the control of hyperglycemia
• Treatment of pediatric patients over 6 years of age with Type 1 diabetes mellitus who require basal (long-acting) insulin for the control of hyperglycemia

Most serious warnings and precautions
• Hypoglycemia is the most common adverse effect of insulin
  – Uncorrected hypo- or hyperglycemia can cause loss of consciousness, coma or death
  – Glucose monitoring is recommended
  – Changes to insulin should be made cautiously, under medical supervision

• Administration
  – Not intended for intravenous or intramuscular administration
  – Do not mix with any other insulin or dilute with any other solution
  – Do not use if not water-clear and colourless or if a deposit of solid particles has formed on the wall of the vial or cartridge

Other relevant warnings and precautions
• Risk of allergic reactions, injection site reactions, lipodystrophy, pruritus, rash and antibody formation
• Rate of absorption is dependent on blood supply, temperature and physical activity
• Hyperkalemia
• Sodium retention and edema

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Scurvy in the Context of End-Stage Liver Disease
Amiirah Aujnarain MSc, Michael Hackett CD CCPA, Kumanan Wilson MD MSc

About the Authors
Amiirah Aujnarain is a member of the Faculty of Medicine. Michael Hackett and Kumanan Wilson are members of the Division of General Internal Medicine, and Kumanan Wilson is also a member of the Department of Medicine, Ottawa Hospital Research Institute. All are at the University of Ottawa, in Ottawa, Ontario. Correspondence may be directed to kwilson@ohri.ca.

Summary
The authors present a case of scurvy in a 38-year-old woman with a history of alcohol abuse and end-stage liver disease. Their case demonstrates that nutritional deficiencies are a concern in patients affected with alcoholic liver disease. The history, symptoms, and treatment of scurvy are also discussed.

Résumé
Les auteurs analysent un cas de scorbut chez une femme de 28 ans présentant des antécédents d’alcoolisme et atteinte de maladie hépatique terminale. Ils démontrent que les carences nutritionnelles sont problématiques chez un patient atteint de maladie hépatique chronique. Ils décrivent également l’histoire, les symptômes et le traitement du scorbut.

Case
A 38-year-old Caucasian female presented with coffee-ground emesis, melena, lethargy, malaise, jaundice, and confusion. The patient had a history of excessive alcohol use, cirrhosis, alcoholic hepatitis, progressive abdominal pain, and increasing scleral icterus. She had also had a polyneuropathy since 2009, presumed to be secondary to excess alcohol intake. Past medical history included fibromyalgia and possible systemic lupus erythematosus (SLE).

On admission, her blood pressure was 96/60 mm Hg, heart rate 115 beats per minute, respiratory rate 18 breaths per minute, and oxygen saturation 98% on room air, and she was normothermic. She was jaundiced, with blood in her mouth. Her abdomen was distended with bulging flanks but with no palpable liver or spleen. She had pitting edema to her sacrum. The neurological examination was consistent with the known peripheral sensory motor neuropathy. There was no bleeding of her nails and no corkscrew hair.

Initial blood work demonstrated a hemoglobin of 70 g/L, a white blood cell count of 17.2 × 10^9/L, and a platelet count of 177 × 10^9/L. Her electrolytes showed a sodium level of 119 mmol/L, potassium 5.1 mmol/L, chloride 89 mmol/L, and carbon dioxide 20 mmol/L. Creatinine and urea were 75 and 5.4 mmol/L, respectively. Her International Normalized Ratio (INR) on admission was 2.7 (normal 0.9–1.2). The total bilirubin level was 193 µmol/L (normal 3–17 µmol/L). A subsequent indirect bilirubin level was 74. Her level of alanine aminotransferase (ALT) was at 27 U/L (normal 17–63 U/L), aspartate aminotransferase (AST) 114 U/L (normal 15–37 U/L), γ-glutamyl transpeptidase (GGT) 34 U/L (normal 5–55 U/L), alkaline phosphatase (ALP) 175 U/L (normal 50–136 U/L), and lipase 56 U/L (normal 73–393 U/L).

The patient was given two units of packed red blood cells, pantoprazole and octreotide for her upper gastrointestinal (GI) bleed, and ceftriaxone for possible spontaneous bacterial peritonitis. She had no further GI bleeds and received lactulose (titrated to three bowel movements per day) for hepatic encephalopathy. She received furosemide for peripheral edema, which was later switched to a potassium-sparing diuretic, and pentoxifylline for hepatitis.

The patient had progressive non-resolving anemia despite no obvious ongoing GI source. She also had a progressive peripheral neuropathy. Increasing non-conjugated hyperbilirubinemia led to the consideration of autoimmune hemolysis, and rheumatology was consulted to determine if this and her arthralgias were from SLE. Rheumatology found her to be positive for anti–smooth muscle antibody and antinuclear antibody (homogeneous pattern; titre of 1:160). They concluded that there was an autoimmune component to her symptoms and started her on steroids, but with minor improvement only. Neurology concluded that she had a severe generalized polyneuropathy following electromyographic
studies that showed axonal loss and demyelination. She received low-dose hydromorphone for pain. Of note, the patient had continued gingival bleeding despite vitamin K administration and the normalization of her INR.

Many symptoms, such as the gingival bleeding, arthralgias, weakness, anemia, and jaundice, indicated the possibility of scurvy. Testing confirmed this suspicion, with vitamin C levels <5 µmol/L (normal >25 µmol/L). However, oral vitamin C intake did not resolve her symptoms. Due to the possibility of malabsorption secondary to gut edema, IV vitamin C was started. Within days, the patient’s muscle weakness improved, her hemoglobin stabilized, her arthralgias improved, and she had no further gum bleeding.

Discussion
Scurvy was first documented in 1500 BC; however, the etiology remained unclear until Sir James Lind conducted a study upon a ship where he treated affected soldiers and found the cure to be lemons and oranges. Scurvy is now known to result from a nutritional deficiency of vitamin C (ascorbic acid). In the developing world, the last significant outbreak was in 2002 in Afghanistan after a long drought. While infrequently diagnosed in the developed world, it is important to consider, especially for populations such as the elderly, alcohol abusers, drugs abusers, those who follow strict diets, those who have eating disorders, or those with malabsorption.

Early symptoms are nonspecific: fatigue, weakness, weight loss, myalgias, arthralgias, and irritability. This makes the diagnosis of scurvy challenging. More identifiable symptoms take 1–3 months to appear. Vitamin C deficiency impairs collagen synthesis. Specifically, it promotes molecular cross-linking, which gives collagen its elasticity. Deficiency results in bleeding gums, joint pain, and petechiae. Rarely, there are cardiac changes including ST elevation and atrioventricular blocks. Left untreated, vitamin C deficiency can be fatal. Humans are unable to synthesize vitamin C or store it. Exogenous sources include fruits and vegetables. Fresh foods are needed as ascorbic acid deteriorates through cooking or storage.

The effects of alcohol on ascorbic acid are poorly understood. Faizallah et al. conducted a small study in nine healthy male subjects. The study found that almost half of ascorbic acid was excreted within 4 hours of alcohol ingestion. Although this relationship requires further study, our case highlights scurry’s potential in alcohol abusers and the potential for late diagnosis due to non-specific signs and symptoms. Scurvy is important to recognize given its potentially fatal consequences, and the simplicity of treatment. Scurvy is cured with vitamin C administration. Currently, the daily recommended intake is 75 mg for women and 90 mg for men. For those with scurvy, 1–2 g of daily vitamin C is recommended for the first 3 days, then 500 mg daily for 7 more days. After that, 100 mg of vitamin C should be taken daily for 3 months. Fatigue, lethargy, pain, and confusion typically improve within 24 hours. Bruising, gingival bleeding, and weakness resolve within 2 weeks. Complete recovery should be expected within 3 months.

For our patient, vitamin C deficiency explains several, but not all, of her signs and symptoms. Vitamin C deficiency likely caused her bleeding gums and nails and contributed to arthralgias, all of which improved with treatment. Her anemia was likely multifactorial. However, a component may have been from scurvy-induced intravascular hemolysis (which also explains the unconjugated hyperbilirubinemia). Her peripheral neuropathy was likely unrelated, although vitamin C deficiency has previously been associated with femoral nerve neuropathy.

References
Commentary
Lessons from History: Still Relevant in the “Information Age”

Once you eliminate the impossible, whatever remains, no matter how improbable, must be the truth.
—Sherlock Holmes (by Sir Arthur Conan Doyle, 1859–1930)

In this edition of CJGIM, Aujnarain et al. present an interesting case and a fascinating disease. In so doing, they also provide several reminders for our specialty: (1) before you can treat a disease, you must think of it; (2) our work remains as much cerebral as procedural; and (3) when all else fails, perform (and then re-perform) a full history and physical examination. It is also a chance for those of us excited by history and discovery (the so-called “exploration of alleyways to see if they are blind”) to retell a story that is equal parts science, tradition, and innovation—much like medicine itself.

Most people know that scurvy refers to a deficiency of vitamin C that impairs collagen synthesis and results in weakness, bleeding, and unhealed wounds.1–5 Scurvy is fatal if untreated for months, but is otherwise easily prevented and cured. Therefore, it is now rare, and often not considered until multiple specialists have been engaged and many diagnoses ruled out. Accordingly, its historical importance may be tough to grasp. However, with the exception of famine, scurvy has caused more suffering than any other nutritional disease.1,2 Its infamy is further illustrated by the fact that it is not just a noun but also a pejorative adjective, meaning worthless, contemptible, or vile.

Derived from the Latin scorbutus, it was described by Hippocrates over 2,000 years ago.2–5 While the need for vitamin C seems elementary to modern readers, the journey to discovery represents centuries of theory versus counter-theory, and unmovable traditionalists versus scientific pioneers. It encompasses high seas adventure and travel to the most inhospitable lands of earth. An argument can also be made that the history of Canada (and the history of northern exploration) is actually the history of conquering scurvy.

Portuguese and Spanish sailors secured the southern routes to the “New World.” This left the French and British to battle for less tropical lands to the north.2 Samuel Champlain’s early voyages to Canada, the pilgrim’s first winter, and Franklin’s ill-fated journey to the Northwest Passage were almost certainly beset by scurvy.1–3 The Irish potato blight of 1845–1848 caused horrific starvation along with widespread scurvy.2 This catalyzed mass migration to North America, and Canada was a more affordable destination than America.2 This is one reason why over four million (or approximately 14%) of the Canadian population has Irish roots.2

Until the 19th century, scurvy killed more sailors than enemy action.1–3 The eventual adoption of antiscorbutics (and improved hygiene in general) changed this dramatically. James Cook (arguably the greatest explorer of all time) mapped Newfoundland and claimed for Britain the west coast of North America (along with the South Pacific and Antipodes). Cook relied upon (relatively ineffective) sauerkraut but coupled this with regular replenishment of fresh food. By the 1790s, and following regular citrus rations, the British navy could remain at sea longer than the French. This was crucial in the eventual victory over Napoleon Bonaparte and, therefore, the transfer of French North America to the British. This helps explain why this Canadian editorial is written in English rather than French. It is also why this English-Canadian author has to tolerate being called a “limey.”

North American Natives knew to store berries and leaves. They also got vitamin C from a raw diet that included organ meat.2 Interestingly, during his 1903 and 1911 expeditions, the British explorer Robert Scott (“Scott of the Antarctic”) was meticulous about eating fresh meat while at base camp (seals and penguins … for those of an epicurean bent).4,5 Fresh meat contains some vitamin C, and his team remained healthy.4,5 However, on long-range expeditions, he carried dehydrated meat (which he boiled, thereby destroying its vitamin C). Predictably, Scott’s group developed scurvy within 3 months of the switch to pemmican.4 He was also foiled because the prescribed lime juice was boiled in copper vats, which denatured the vitamin C.4,5 In addition, Scott was a victim of alternative theories of nutrition. His chief physician believed (like many) that scurvy was caused by bacterial contamination rather than nutritional deficiency.4 Scott fallaciously believed he would avoid nutritional deficiency because of the care taken in the preparation of his pemmican.

Scurvy does not occur in most animals because they
synthesize vitamin C. Humans and primates lack the enzyme L-gulonolactone oxidase.\textsuperscript{2,3} In the 1400s, Vasco da Gama sought out Moorish traders for “their fine oranges.” In the 1500s, Jacques Cartier explored the St. Lawrence River, and mimicked Natives by making tea from the needles of the arbor vitae tree (white cedar). Sir Richard Hawkins suggested using citrus fruit to eradicate scurvy in the 1500s, as did Sir James Lancaster in the 1600s, and Johann Bachstrom in the 1700s.\textsuperscript{1–3} However, it is James Lind who is best known.\textsuperscript{1–3} This is because he performed what most believed to be the first randomized clinical trial.\textsuperscript{2}

In 1747, Lind divided 12 sick sailors into groups of two and prescribed one of six treatments: (1) alcoholic cider, (2) elixir vitriol, (3) vinegar, (4) sea water, (5) oranges and lemons, and (6) spices.\textsuperscript{2,3} The modern reader would not be surprised that group five showed the greatest improvement. However, Lind had no idea why oranges and lemons worked. He chose his treatments because they were common.\textsuperscript{2} Nor did he question the leading hypotheses, which included “putrefaction,” “blocked perspiration,” and “an excess of melancholic humor.”\textsuperscript{2,2} It took until the 20th century and required worldwide efforts to understand the role of vitamins, to isolate what became known as vitamin C, to determine its chemical structure, and to synthesize it commercially.\textsuperscript{2,3} Discovery requires time … often a lot of time. It also requires the best of the world to collaborate freely.

Modern-day scurvy has occurred in students (poor diet combined with excess alcohol), those living in isolation (no access to fresh food), those belonging to extreme diet groups (zero carbohydrates), and those beleaguered by alcohol or drugs. Ideas used to travel via the high seas, but now travel by the Internet superhighway. The web can help form virtual communities that challenge established knowledge, foster distrust of authority, and increase non-mainstream behaviour (including extreme diets). In short, vitamin deficiency can still occur in modern Canada just as it occurred in our exploratory past. We should remain alert to the possibility even in this so-called “information age.”

Peter G. Brindley MD
Associate Editor
Canadian Journal of General Internal Medicine

References
1. Bow \textsuperscript{ }n SR. Scurvy: how a surgeon, a mariner and a gentleman solved the greatest medical mystery of the age of sail. Toronto (ON): Thomas Allen Publishers; 2003.
Indications and clinical use: Victoza® is indicated for once-daily administration for the treatment of adults with type 2 diabetes to improve glycemic control in combination with:

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Not a substitute for insulin. Should not be used in type 1 diabetes. Patients >70 years may experience more gastrointestinal side effects. Victoza® is not indicated for use in pediatric patients.

Contraindications:

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Thyroid C-cell tumours: Liraglutide causes thyroid C-cell tumours in both genders of rats and mice. It is unknown whether Victoza® causes thyroid C-cell tumours, including medullary thyroid carcinoma (MTC), in humans. Patients should be counselled regarding the risk and symptoms of thyroid tumours.

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- Risk of cardiovascular effects (increased heart rate, PR interval prolongation)
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- Association with transient gastrointestinal adverse reactions
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*Adapted from Pratley RE et al, 2011. A 26-week extension of the original 26-week Pratley RE et al, 2010 trial (total 52 weeks); an active-comparator, parallel-group, open-label, multicentre trial randomized 665 patients with type 2 diabetes (1:1:1) to once-daily Victoza® 1.2 mg or 1.8 mg or once-daily sitagliptin (100 mg) in combination with metformin (a 1500 mg/day). The primary outcome measure was change in A1C from baseline to week 26. Values for A1C were Victoza® 1.2 mg (n=221): (Baseline: 8.4%, change was -1.3%); Victoza® 1.8 mg (n=225): (Baseline: 8.4%, change was -1.5%); sitagliptin 100 mg (n=219): (Baseline was 8.5%, change was 0.0%). Values for weight were Victoza® 1.2 mg (n=221): (Baseline was 93.7 kg, change was -2.8 kg); Victoza® 1.8 mg (n=225): (Baseline was 94.6 kg, change was -3.7 kg); sitagliptin 100 mg (n=219): (Baseline was 93.1 kg, change was -1.2 kg) (all in combination with MET).

References:

CSIM Continuing Professional Development Mission Statement

Our ultimate goal is to go beyond the simple transmission of information. Our goal is 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.

Mission de la SCMI sur le plan du développement professionnel continu

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Kelowna General Hospital (KGH) is seeking General Internists to join a new medical teaching service at one of BC’s most progressive hospitals, in partnership with the University of British Columbia’s Faculty of Medicine and its Southern Medical Program (SMP). Located in the heart of the Okanagan, in Southern British Columbia, KGH is a 350 bed state-of-the art tertiary centre offering a full range of clinical services and a campus that includes the recently opened brand-new Emergency Department, a new ambulatory care facility, and an innovative Heart and Surgical Centre, currently under construction.

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Not recommended in children.

b

SUPERIOR for the endpoint of major bleeding

PRIMARY SAFETY ENDPOINT (31% RRR, 2.13%/year vs. 3.09%/year, respectively; HR 0.69, 95% CI 0.60-0.80, p=0.0001)

C

SUPERIOR for the endpoint of intracranial hemorrhage vs. warfarin

COMPONENT OF PRIMARY SAFETY ENDPOINT (58% RRR, 0.33% vs. 0.80%/year, respectively; HR 0.42, 95% CI 0.30-0.58, p<0.001)

D

SUPERIOR reduction in all-cause mortality

SECONDARY ENDPOINT (11% RRR, 3.52%/year vs. 3.94%/year, respectively; HR 0.89, 95% CI 0.80-1.00, p=0.0465)

E

ALL OF THE ABOVE
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