A PERSONAL VIEW

The difference between a clinical technician and clinical practitioner is in the scope of practice: the need for a bioscience understanding in paramedicine

Joseph A. Rathner1,2 and Christine Kettle1

1School of Pharmacy and Biomedical Sciences, La Trobe Institute for Molecular Sciences, La Trobe University, Bendigo, Victoria, Australia; and 2Department of Physiology, School of Biomedical Sciences, The University of Melbourne, Parkville, Victoria, Australia

Submitted 14 August 2019; accepted in final form 3 October 2019

INTRODUCTION

Often times students studying allied health degrees dismiss the need for detailed bioscience understanding in their clinical degree programs. This is often supported by their clinically oriented, discipline-specific instructors (15, 20). The perennial complaint from this group of students is that they feel that they are taught too much content that is not relevant to their clinical practice (“Only teach us what we need to know!”). Aside from the lack of intellectual curiosity from these university students, it is our view that their approach to the biosciences is detrimental to their clinical practice.

Medicine has a long history of teaching the discipline as a clinical science: that is, in medical education, high importance is placed on a deep understanding of the biology of the human body. As one discussion about the role of bioscience in the medical curriculum has noted:

Fundamental knowledge of the human body is necessary (1) for clinical application and (2) for developing effective thinking skills necessary for successful clinical reasoning and decision making... (18)

The authors further observe that there is a disparity between a technician and a clinician. The technician is trained to follow protocols and routine processes. Whereas the clinician, due to a fundamental knowledge of the human body and the ability to reason and assess evidence, can modify protocols to effectively respond to new and changing conditions (18).

McColl and colleagues (24) have cited empirical data that medical doctors trained as clinical scientists are better able to diagnose unexpected and unusual disease presentations compared with health care professionals trained to follow clinical practice guidelines. Furthermore, trainee medical doctors make better diagnoses when they have deeper understanding of bioscience than trainees who are taught to diagnose disease based on statistical models of disease (25, 37, 38).

Students studying for Paramedic Practice degrees at our university study principles in physiology and introduction to anatomy in the first two semesters of their degree. These students then build on their foundation in human bioscience education in the second semester of the second year by studying Foundations in Pharmacology subject. The instructor teaching pharmacology also teaches the introductory human biosciences subjects in the first year and so has a very clear idea of the expected levels of knowledge of physiology and anatomy that these students have before studying pharmacology.

The paramedic students enter directly into their paramedic degree from high school, and have a B to B+ average in their final year of school. Although their human bioscience education precedes their clinical management subjects, the students have had discipline-specific mentoring lead by paramedic instructors. They are a small cohort of students (~40 students).

Ambulance Victoria publishes Clinical Practice Guidelines for Ambulance and MICA Paramedics [CPG; see https://www.ambulance.vic.gov.au/paramedics/clinical-practice-guidelines/ (1)], which clearly sets out the protocols for the paramedic management of a whole range of clinical scenarios that the
paramedic is likely to encounter. It also lists a formula of
drugs that paramedics are licensed to administer, which in-
cludes explicit descriptions of how the drugs are packaged,
what they are used for, and a list of indications, contraindic-
tions, and precautions. The clinical management guidelines
can be described as a decision tree flow chart for describing
the protocols and procedures that paramedics should follow when
on call to an emergency. The students are encouraged to view
the CPG as their go-to guide for clinical patient management.

The pharmacology subject is taught by specialist bioscience
instructors (physiologists). The subject is an “off-the-shelf”
generic subject taught to allied health students, intended to
provide a foundation for understanding drugs and drug inter-
actions to students who may be prescribing medicines to
patients, or even recommending over-the-counter medicines
(e.g., analgesics) as part of their regular practice. In lectures,
students were taught concepts related to pharmacokinetics,
pharmacodynamics, adverse drug reactions, drug interactions,
and legislative regimes for drug prescription authorization.
There was also a system-by-system review of common medi-
cines and their mechanism of action for achieving therapeutic
effect.

Notwithstanding the fact that the pharmacology subject was
designed specifically for allied health students from a wide
variety of disciplines, a consistent complaint from the para-
medicine students about the subject, across a number of years
and instructors, was the lack of relevance of the material for
their specific discipline. Examples of comments from the
institutional student feedback of subject include:

- “I found the subject to be mostly irrelevant to paramedicine,
sure it is insightful to know the workings of what happens in
the process of a drug from administration to excretion, it is
however not essential to know it in the depth that was
delivered and expected of by students within the subject.”
- “For paramedic students much of the information will be
irrelevant and it is hard to pay attention when this is known.”
- “Need to be able to better relate to paramedics. Not really
relevant.”
- “This subject should be more specific to paramedics. e.g.,
the allocated drugs for the oral presentation should be
paramedic drugs rather than random drugs, makes it hard to
apply to paramedicine.”
- “Lectures need to more specific to paramedicine.”
- “Needs to relate more to paramedics.”

The Clinical Scenario

As part of their assessment, the students work on a 4-wk
enquiry-based learning clinical scenario. The scenario (ori-
ginally written by a paramedic instructor) describes a 68-yr-old
woman who reports symptoms consistent with unstable angina,
including crushing chest pains (10/10 on analog scale) that
began during moderate-intensity activity (watering the garden)
but continued while resting and radiated through the arm and
jaw. The patient also reported nausea and was taking a teta-
cycline antibiotic to treat a urinary tract infection.

The students are asked a series of questions that are intended
to guide them through the case management. The students are
explicitly instructed that, while they will utilize their CPG, they
are expected to do deeper research, drawing on course content
and literature searches to determine both the correct course of
action and justification for those actions.

The key to understanding the rationale for clinical treatment
of acute coronary syndrome is recognizing that treatments are
aimed at both increasing perfusion of blood to coronary muscle
and decreasing oxygen demand to the heart, by decreasing
cardiac work [14 (see p. 516), 16, 29, 33, 35]. It is important
to address the pain associated with acute coronary syndrome,
since the pain increases anxiety, which activates the sympa-
thetic nervous system, increasing cardiac work and exacerbat-
ing the ischemia [Porth (35), see p. 457], thus alleviating the
pain is a priority in patient care.

This essay will present the clinical scenario and describe the
responses that were provided by the novice paramedic stu-
dents, following the CPG. Later, how the students might have
approached the scenario with a clear understanding of the
underlying physiology and pharmacology will be discussed.
What is evident is that the students, following the CPG alone,
make serious errors in judgment, which would be avoided if
they had employed effective thinking skills and a clear under-
standing of the underlying biology. The purpose here is to
demonstrate how strict adherence to the CPG, without resort-
ing to clinical reasoning skills or underlying bioscience under-
standing, can and will lead to fatal error. Or, to put it in other
words, the purpose of this essay is to demonstrate how, for
even relatively straightforward clinical scenarios, a deep un-
derstanding of the underlying human physiology and pharma-
cology can inform the clinical decision making of a beginning
clinical practitioner.

Treating Acute Coronary Syndrome: A Practice Guide
Approach

The CPG for acute coronary syndrome (4) indicates that
paramedics should immediately provide pain relief for the
ischemic pain, initially via the administration of glycerol trini-
trate (GTN), initiate anti-thrombotic therapy, and, if required,
provide additional pain relief via the appropriate guidelines (6).
Students following the CPG would thus initiate treatment by
administering GTN, which has hypotensive effects. The para-
medic trainee would then administer aspirin as an anti-coagu-
ant and, finally, if pain persists, morphine (or another opioid
analgesic, fentanyl) for pain management.

Throughout the enquiry, the students are asked to provide
“rationale” for their treatment choices. Students correctly iden-
tify that GTN will vasodilate blood vessels throughout the
body. They reason that this vasodilatation will allow coronary
vessels to dilate, facilitating reperfusion of the coronary mus-
cles, thus reversing the ischemia.

Students are then asked to identify their pain management
options. Paramedics in Victoria (Australia) have three analge-
sic options for pain scores above 2/10: methoxyflurane, mor-
phine, and fentanyl (6). Morphine is administered intrave-
nously, which paramedics are likely to cannulate as part of
their standard clinical approach (11), whereas fentanyl (intra-
nasal) (8) and methoxyflurane (inhalation) (10) are adminis-
tered through non-intravenous routes. The CPG identifies me-
thoxyflurane as being contraindicated when tetracycline anti-
biotics are being used (10), but makes no mention that
methoxyflurane should also be avoided for patients with car-
diac instability with pain due to acute coronary syndrome [13 (see p. 38), 19, 28].

There is no clear guideline within the CPG whether morphine or fentanyl should be used to provide additional pain relief (in addition to that likely provided by relieving the ischemia). The Australian National Health and Medical Research Council recommends the use of morphine in pain management of acute coronary syndrome (17).

Within the scenario, after the students have administered the analgesic, presumptively the opioid narcotic, the patient becomes bradycardic and hypotensive. In addition to the altered cardiovascular parameters, the patient shows evidence of altered mental state (Glasgow coma score falls from 15/15 to 10/15) (3), and (the students are told) respiratory rate falls to 12 breaths/min. The students are asked what caused the altered clinical condition, how would they respond, and what drug within their scope of practice would they administer?

Students correctly identified that hypotension, bradycardia, altered mental state, and respiratory depression are all known side effects of opioid overdose (7, 8, 11) and opted to treat the perceived overdose with naloxone, as per the CPG (7).

The problem with the students’ approach to this problem is that they neglected several important issues and questions, which require a more insightful understanding of the pharmacological and physiological mechanisms:

1) Known and predictable side effects of GTN include orthostatic hypotension and bradycardia (9, 14).

2) Twelve breaths per minute is within the normal respiratory rate for an adult [2, 23 (see p. 834)].

3) Treatment with naloxone will deprive them of any alternatives for analgesic care. The flow on effects of naloxone treatment is likely to be return of pain and increased anxiety, leading to increased cardiac stress.

TREATING ACUTE CORONARY SYNDROME: A CLINICAL SCIENTIST APPROACH

The cause of acute coronary syndrome is myocardial infarction, which is deprivation of oxygen to the cardiac muscle. In effect, occlusion of coronary blood vessels prevents oxygen delivery to the heart muscle, causing pain. The correct initial course of treatment is, as indicated in the CPG, GTN, aspirin, and, if indicated, morphine. What our students largely do not deduce, however, is that GTN will relax vascular smooth muscle throughout the body, causing both resistance and capacitance vessels to dilate (33). Reducing total peripheral resistance will predictably decrease mean arterial pressure (hypotensive action), but it will also lead to a decrease in venous return, leading to a decrease in end-diastolic volume and preload. Paying attention to the basic bioscience behind the use of GTN, the students should have recognized that decreased end-diastolic volume decreases preload, which will decrease cardiac inotropy, leading to reduced stroke volume and decreased cardiac output [Marieb and Hoehn (22), see Fig. 18.22, p. 682]. The combined effect of this is to decrease cardiac work and reduce oxygen demand on the heart (29, 33, 34). Thus GTN has the twin effect of facilitating reperfusion of cardiac muscle and also decreasing the oxygen demand to cardiac muscle, both of which will reduce the ischemia.

It is expected that treating the patient with GTN will alleviate pain. However, if pain persists, it is important to treat the pain, not insignificantly due to the sympathoexcitatory effects that the pain will have on heart rate and strength of contraction. That is, pain relief has two benefits in this scenario, both of which are clinically important: 1) reduce pain, and 2) reduce cardiac oxygen demand, by decreasing cardiac drive.

After morphine (or opiate) administration, the patient experiences bradycardia and hypotension. At this time, it is important to note that the patient has been administered two drugs that each induces hypotension, through separate mechanisms. This, coupled with the fact that the reported respiratory rate is within normal range, indicating that there is no respiratory suppression, suggests that the altered mental state that our patient is experiencing is most likely due to reduced cerebral blood flow, leading to syncope [see Porth (34), p. 437] and not opiate overdose.

The immediate treatment should be to lay the patient down and elevate her feet. The rationale for this relates to the decreased venous return caused by the relaxation of the venous capacitance vessels. Elevating the feet will increase the venous return (27, 36), which will increase the end-diastolic volume, thus increasing preload, which will increase the stretch on the ventricular wall, which increases the force of contraction of the heart. This is a practical example of the Frank-Starling law of the heart [Marieb and Hoehn (22) see p. 682]. All of this will lead to increased cardiac output and higher mean arterial pressure. Unfortunately, this simple procedure is not an option for our novice students, as it is not indicated in their CPG and, therefore, was not considered by the paramedic student.

The choice to use naloxone with the presentation of these symptoms would seem to be counterproductive. Although the priority for the paramedic is to sustain life, the 68-yr-old woman in this case scenario does not seem to be at risk at this moment. Treating with naloxone would reverse the pain treatment, and the paramedic has no alternative pain treatments available: fentanyl also being an opioid would be ineffective after naloxone, and methoxyflurane is contraindicated, as explained above. The pain itself is likely to exacerbate the cardiac ischemia by increasing anxiety, leading to increased heart rate.

Our paramedic has three more treatment options: intravenous saline, atropine, and adrenaline. The intravenous saline would increase fluid volume in the venous system, again acting to increase venous return and increase cardiac output.

Atropine is recommended by the CPG for treating bradycardia associated with ischemic chest pain (5). The choice of atropine, a muscarinic antagonist, rather than epinephrine, an adrenergic agonist, is obvious to the clinical scientist. The parasympathetic nervous system acts as a break on heart rate, whereas the sympathetic nervous system acts to accelerate heart rate and to increase vasomotor constriction (22). The neurotransmitter released by parasympathetic postganglionic neurons is acetylcholine, acting on muscarinic receptors, whereas norepinephrine is released by sympathetic postganglionic neurons onto adrenergic receptors [Marieb and Hoehn (21), see Fig. 14.2, p. 526]. Crucially, the sympathetic nervous system innervates both the heart and blood vessels (Ref. 21, see Fig. 14.6, p. 532), whereas the parasympathetic nervous system innervates the heart muscle but not blood vessels (Ref. 21, see Fig. 14.4, p. 528). This selective innervation of the heart and not blood vessels means that the muscarinic antagonist will release the break on heart rate (allowing for increased heart
rate) without causing vasoconstriction, preserving the vasodilation induced by the GTN and facilitating adequate perfusion of blood to cardiac muscle.

Conclusion

The purpose of this essay was to demonstrate the distinction in clinical outcome when practitioners draw on and apply basic science knowledge, leading to good clinical outcomes, compared with the rigid application of clinical practice guidelines. The proper management of the patient required the clinical scientist to draw on his/her understanding of cardiovascular physiology, particularly the details of the Frank-Starling mechanism, and a detailed understanding of the organization of the autonomic nervous system to deliver appropriate care. In addition, the students were misled in the case history into believing that a respiratory rate of 12 breaths/min was hypoventilation (mistaking reduced ventilation for hypoventilation). Finally, the case study was intended to assess the students’ understanding of the pharmacology of their practice, considering drug indications, contraindications, and (significantly) mechanism of action. Not considering the drugs pharmacodynamics and simply following the CPG lead the novice clinician to make errors in judgment that have potentially serious adverse outcomes.

In the Australian context, the scope of Paramedic Practice has been outlined by Paramedics Australasia (32) and is broadly similar to the roles assigned to emergency medical technicians and paramedics in the United States (26). In the Australasian context, paramedicine roles are divided between the professional stream, technical stream, and ambulance communication stream (32). A first responder is the lowest level of training within the technical stream of Paramedic Practice, and a basic life support medic is the highest level of practice. Minimum educational standards for each of these roles is Certificate II and Certificate IV, both of which are pre-Bachelor’s qualification, according to the Australian Qualification Framework (12). In contrast, the minimum standard of education for a professional stream paramedic is a Bachelor’s degree in Paramedics (32) (level 7 within the Australian Qualifications Framework). The key distinction in scope of clinical practice between the technician and the practitioner, as described by Paramedics Australasia, is the requirement to operate within prescribed clinical guidelines. The paramedic professional is required to have a Bachelor’s degree and manages patient care to his/her best clinical judgment. The technician has a lower level (or pre-Bachelor’s) of education, and clinical practice is limited to CPG (32). Furthermore, the competency standard for Australian paramedics states, in addition to an understanding of human anatomy, physiology, and pathophysiology relevant to paramedic clinical practice, that paramedics need to “understand the clinical sciences underpinning paramedic practice, including physiological, pharmacological behavioural and functional” (31). To assess a student’s ability to meet this competency standard, there is a need to be able to reason a clinical intervention in an atypical or previously unexperienced situation based on available evidence and an understanding of the underlying biology. The student should not reject a simple procedure like elevating a patient’s feet simply because it is not in the CPG.

A note of caution, the purpose here was not to suggest that paramedics are ill equipped to handle emergency medical care. Emphasis should be placed on the fact that the students here are novices. Working under an experienced clinician, and as they gain experience themselves, these students will come to rely more and more on their experience in clinical decision making; consequently, their speed of response and clinical decision making will improve, just as it does for a medical practitioner. This would be consistent with the “two worlds model” that describes the theory that medicine educators put forward to describe the development of clinical thinking as medical practitioners from novice to expert (30) and the importance of providing students with sufficient bioscience education to facilitate the transition to informed clinical practitioner.

DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

J.A.R. conceived and designed research; J.A.R. drafted manuscript; J.A.R. and C.K. edited and revised manuscript; J.A.R. and C.K. approved final version of manuscript.

REFERENCES

1. Ambulance Victoria. Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014.
2. Ambulance Victoria. CPG A0103 respiratory assessment. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 10.
3. Ambulance Victoria. CPG A0104 conscious state assessment. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Bernard S. Doncaster, VIC, Australia: Ambulance Victoria, 2019, p. 12–13.
4. Ambulance Victoria. CPG A0401 acute coronary syndrome. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 50–53.
5. Ambulance Victoria. CPG A0402 bradycardia. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 54–55.
6. Ambulance Victoria. CPG A0501 pain relief. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 76–79.
7. Ambulance Victoria. CPG A0707 overdose: opioids. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 108–109.
8. Ambulance Victoria. CPG D010 fentanyl. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 312–313.
9. Ambulance Victoria. CPG D013 glycerol trinitrate (GTN). In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 316–317.
10. Ambulance Victoria. CPG D017 methoxyflurane. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 326.
11. Ambulance Victoria. CPG D020 morphine. In: Ambulance Victoria Clinical Practice Guidelines for Ambulance and MICA Paramedics, edited by Dixon A. Doncaster, VIC, Australia: Ambulance Victoria, 2014, p. 332–333.
12. Australian Qualification Framework Council. Australian Qualification Framework, Canberra, ACT, Australia: Australian Government Department of Education and Training, 2013.

Advances in Physiology Education • doi:10.1152/advan.00113.2019 • http://advan.physiology.org
13. Bryant B, Knights K (Editors). Pharmacotherapy: clinical use of drugs. In: Pharmacology for Health Professionals (4th ed.). Chatswood, NSW, Australia: Mosby Elsevier, 2015, p. 31–57.
14. Bryant B, Knights K (Editors). Drugs affecting vascular smooth muscle. In: Pharmacology for Health Professionals (4th ed.). Chatswood, NSW, Australia: Mosby Elsevier, 2015, p. 513–535.
15. Davies S, Murphy F, Jordan S. Bioscience in the pre-registration curriculum: finding the right teaching strategy. Nurse Educ Today 20: 123–135, 2000. doi:10.1054/nedt.1999.0375.
16. Dean CS. Non-ST-elevation myocardial infarction (Online). BMJ Best Practice. https://bestpractice.bmj.com/topics/en-us/151 [26 Sept 2019].
17. Doherty S, Acworth J, Taylor S, Bennett S. Emergency Care Acute Pain Management Manual. Canberra, Australia: National Health and Medical Research Council, Commonwealth Government of Australia, 2011.
18. Finnerty EP, Chauvin S, Bonaminio G, Andrews M, Carroll RG, Pangaro LN. Flexner revisited: the role and value of the basic sciences in medical education. Acad Med 85: 349–355, 2010. doi:10.1097/ACM.0b013e3181c88b09.
19. Grindlay J, Babil FE. Efficacy and safety of methoxyflurane analgesia in the emergency department and prehospital setting. Emerg Med Australas 21: 4–11, 2009. doi:10.1111/j.1742-6723.2009.01153.x.
20. Larcombe J, Dick J. Who is best qualified to teach bioscience to nurses? Nurs Stand 17: 38–44, 2003. doi:10.7748/nst.17.51.38.s51.
21. Marieb EN, Hoehn K. The autonomic nervous system. In: Human Anatomy and Physiology (9th ed.). Boston, MA: Pearson, 2013, p. 524–543.
22. Marieb EN, Hoehn K. The cardiovascular system; the heart. In: Human Anatomy and Physiology (9th ed.). Boston, MA: Pearson, 2013, p. 658–691.
23. Marieb EN, Hoehn K. The respiratory system. In: Human Anatomy and Physiology (9th ed.). Boston, MA: Pearson, 2013, p. 658–691.
24. McColl GJ, Bilszta J, Harrap S. The requirement for bioscience knowledge in medical education. Med J Aust 196: 409, 2012. doi:10.5694/mja11.10474.
25. Mylopoulos M, Woods N. Preparing medical students for future learning using basic science instruction. Med Educ 48: 667–673, 2014. doi:10.1111/medu.12426.
26. National Highway Traffic Safety Administration. National Emergency Medical Services Education Standards. Washington, DC: US Department of Transportation, National Highway Traffic Safety Administration, 2009.
27. Nixon JV, Murray RG, Leonard PD, Mitchell JH, Blomqvist CG. Effect of large variations in preload on left ventricular performance characteristics in normal subjects. Circulation 65: 698–703, 1982. doi:10.1161/01.CIR.65.4.698.
28. NPS Medicinewise. Methoxyflurane (Penthrox) for analgesia (doctor’s bag listing) (Online). https://www.nps.org.au/radar/articles/methoxyflurane-penthrox-for-analgesia-doctors-bag-listing [25 Oct 2019].
29. Overbaugh KJ. Acute coronary syndrome. Am J Nurs 109: 42–52, 2009. doi:10.1097/01.NAJ.0000351508.39509.e2.
30. Pangaro LN. The role and value of the basic sciences in medical education: the perspective of clinical education -students’ progress from understanding to action. J Int Assoc Med Sci Educ 20: 307–313, 2010.
31. Paramedics Australasia. Paramedic Professional Competency Standards (2.2th ed.), edited by Sassella G, Green R. Melbourne, VIC, Australia: The Council of Ambulance Authorities, 2013.
32. Paramedics Australasia. Paramedic Role Descriptions. Melbourne, VIC, Australia: Paramedics Australasia, 2016.
33. Parker JD, Parker JO. Nitrate therapy for stable angina pectoris. N Engl J Med 338: 520–531, 1998. doi:10.1056/NEJM199802193380807.
34. Porth CM (Editor). Disorders of blood flow and blood pressure. In: Essentials of Pathophysiology: Concepts of Altered Health States. Philadelphia, PA: Kluwer Health/Lippincott Williams & Wilkins, 2011, p. 405–446.
35. Porth CM (Editor). Disorders of cardiac function. In: Essentials of Pathophysiology: Concepts of Altered Health States. Philadelphia, PA: Walthers Kluwer Health/Lippincott Williams & Wilkins, 2011, p. 450–461.
36. van Lieshout JJ, Harms MPM, Pott F, Jenstrup M, Secher NH. Stroke volume of the heart and thoracic fluid content during head-up and head-down tilt in humans. Acta Anaesthesiol Scand 49: 1287–1292, 2005. doi:10.1111/j.1399-6576.2005.00841.x.
37. Woods NN, Brooks LR, Norman GR. The role of biomedical knowledge in diagnosis of difficult clinical cases. Adv Health Sci Educ Theory Pract 12: 417–426, 2007. doi:10.1007/s10459-006-9054-y.
38. Woods NN, Brooks LR, Norman GR. The value of basic science in clinical diagnosis: creating coherence among signs and symptoms. Med Educ 39: 107–112, 2005. doi:10.1111/j.1365-2929.2004.02036.x.