ASSESSMENT MODELS FOR MEDICAL SCHOOL ADMISSION

To the Editor:

Following the publication in August 2011 of our article "The effect of integration of non-cognitive parameters on medical students' characteristics and their intended career choices" [1], the following facts have been brought to my attention:

An assessment model for medical school admission, which focuses on candidates' personal and interpersonal qualities, was developed by the National Institute for Testing and Evaluation (NITE) and the Israel Center for Medical Simulations in 2003. The model, named "MOR" (the Hebrew acronym for "selection to medicine") involves multiple assessment tools, which include: individual behavioral simulations, group tasks, interviews and questionnaires. MOR was implemented at Tel Aviv University's Sackler Faculty of Medicine in 2004.

In 2006 the Hebrew University-Hadassah Medical School implemented the change in their admission system as described in my article. This admission system was based on the MOR model and NITE was (and still is) responsible for its development, scoring and reporting. Its implementation was supported by both the Hebrew University and the National Institute for Testing and Evaluation. An article describing MOR and its validation was published in 2008 by Ziv et al. [2]. MOR was found to be reliable and resulted in a 20% change in the cohort of students accepted.

I thank Dr. Naomi Gafni from the National Institute for Testing and Evaluation for enlightening me on this issue, thus enabling me to correct the omission in my article of the institute's contribution to the change I described.

N. Halpern MD
Sharett Institute of Oncology, Hadassah University Medical Center, Jerusalem, Israel
[Naamah2@hadassah.org.il]

References

1. Halpern N, Bentov-Gofrit D, Matot I, Abramowitz MZ. The effect of integration of non-cognitive parameters on medical students' characteristics and their intended career choices. *IMAJ Isr Med Assoc J* 2011; 13 (8): 488-93.
1. Ziv AM, Rubin OR, Moshinsky AV, et al. MOR: a simulation-based assessment center for evaluating the personal and interpersonal attributes of medical school candidates. *Med Educ* 2008; 42: 9919.

OCCAM'S RAZOR AND ISCHEMIC HEPATITIS

TO THE EDITOR:

We read with great interest the paper by Gluck et al. [1] in a recent issue of *IMAJ*. The authors retrospectively analyzed the cases of sharp rise of liver transaminases in their hospital, and added the cases published in the literature. As a result of their analysis, they questioned the very existence of acute hepatotoxicity of intravenous amiodarone. They suggested that the true diagnosis in these cases was ischemic hepatitis and therefore withholding intravenous amiodarone in critically ill patients with dangerous arrhythmia, in fear of acute hepatic toxicity, is not justified.

Several years ago we were invited to review and write an editorial comment on a paper reporting three cases of acute liver toxicity of intravenous amiodarone [2]. These three cases were included in the above paper published in your journal [1]. Analyzing this article we reached the same conclusion as Gluck et al. – namely, there was an alternative explanation for the clinical presentation in all three cases. The other diagnosis could be ischemic hepatitis. We entitled our editorial "Intravenous amiodarone: offender or bystander?" [3]

It is known that ischemic hepatitis, or shock liver, is typically caused by an acute event like shock or any hypotensive episode, especially in patients with preexisting hepatic compromise. One paper not cited by Gluck et al. but worth mentioning is a study where 142 patients had their hepatic blood flow measured. It appeared that the most common scenario of ischemic hepatitis with documented decrease in liver blood flow is chronic congestive heart failure with an acute precipitating event, e.g., arrhythmia [4]. Needless to say, acute arrhythmia in the setting of heart failure is typically treated with amiodarone because almost all other antiarrhythmics (except dofetilide) are contraindicated in this setting. The coincidence is therefore very likely. Interestingly, only 41% of the 142 patients had documented systemic blood pressure below 90 mmHg. The authors concluded that in chronic liver hypoxia resulting from venous congestion, even brief or unrecognized fall in blood pressure can cause ischemic hepatitis.

Besides, it is difficult to understand why amiodarone given orally results in completely different liver toxicity than the same medication given intravenously.

According to the principle of Occam's razor, the simplest explanation is usually correct, and the entities should not be multiplied unnecessarily. If there are two diagnostic entities with a similar clinical scenario, laboratory manifestation, natural course, and pathology, they likely represent the same condition. Since one of them – ischemic hepatitis – is much more common, it is likely that other cases ascribed to liver toxicity of intravenous amiodarone represent, in fact, ischemic hepatitis.

Maya Guglin MD PhD
Director, Heart Failure, Dept. of Cardiology, University of South Florida, Tampa, FL, USA
[mguglin@gmail.com]

References

1. Gluck N, Fried M, Porat R. Acute amiodarone liver toxicity likely due to ischemic hepatitis. *IMAJ Isr Med Assoc J* 2011; 13 (12): 748-52.
1. Rätz Bravo AE, Drewe J, Schlienger RG, Krähenbühl S, Pargger H, Ummenhofer W. Hepatotoxicity during rapid intravenous loading with amiodarone: description of three cases and review of the literature. *Crit Care Med* 2005; 33: 128-34.
1. Guglin M. Intravenous amiodarone: offender or bystander? *Crit Care Med* 2005; 33: (1): 245-6.
1. Henrion J, Schapira M, Luwaert R, Colin L, Delannoy A, Heller FR. Hypoxic hepatitis: clinical and hemodynamic study in 142 consecutive cases. *Medicine* 2003; 82: 392-406.

OPTIMIZING THE UTILITY OF COMPUTED TOMOGRAPHY IN DIAGNOSING PULMONARY EPITHELIOID HEMANGIOENDOTHELIOMA

TO THE EDITOR:

We read with great interest the article by Rosengarten et al. [1], which described the clinical, radiologic and pathologic features of three cases of pulmonary epithelioid hemangioendothelioma (PEH). Computed tomography revealed a right upper lobe mass with calcifications and several smaller bilateral lesions, and a right pleural effusion in one patient, a large left hydropneumothorax with collapse of the left lower lobe in the second patient, and bilateral lung nodules and right pleural effusion in the third patient. The authors commented that the delayed diagnosis could be explained in part by the difficulty in diagnosing PEH. In the cases presented several diagnostic procedures were needed to establish this diagnosis, and the final diagnosis was made on open lung biopsy or transbronchial biopsy.

We would like to highlight the importance of CT as an auxiliary tool in cases of suspected PEH. The most characteristic CT manifestation of PEH is the presence of multiple bilateral, well- or ill-defined, non-calcified nodules measuring up to 2 cm in diameter [1–3]. This presentation is often mistaken for metastatic carcinoma, which is the initial radiologic interpretation in nearly all cases. However, the evolution of PEH is very different. Most described cases of PEH show little or no growth on serial examinations, and partial spontaneous regression has been described several years after detection. Dissemination of the tumor may lead to extensive lymphangitic spread with interlobular septal thickening, enlarged lymph nodes, and small pleural effusions [3]. Other less frequent findings are a solitary lung nodule or mass, a single cavitary nodule, and nodule calcification [2,4].

Although histologic calcification and ossification are common, radiologic calcification is infrequent [2,4]. In most cases, calcification has evolved over years. Rare cases have been described in which calcifications were present at the time of diagnosis [2]. In some cases, hepatic lesions may be associated, especially as masses or calcified small nodules [3]. Open lung biopsy with immunohistochemistry is usually required to establish the diagnosis of PEH. To conclude, PEH should be considered in the differential diagnosis of multiple or solitary nodular lesions, both calcified and non-calcified.

E. Marchiori MD PhD, B. Hochhegger MD PhD and G. Zanetti MD PhD
Department of Radiology, Rio de Janeiro Federal University, Rio de Janeiro, Brazil [edmarchiori@gmail.com]

References

1. Rosengarten D, Kramer MR, Amir G, Fuks L, Berkman N Pulmonary epithelioid hemangioendothelioma. *IMAJ Isr J Med Assoc J* 2011; 13: 676-9.
1. Marchiori E, Hochhegger B, Irion KL. Pulmonary epithelioid hemangioendothelioma presenting as multiple large calcified nodules. *Arch Bronconeumol* 2010; 46: 103-4.
1. Sakamoto N, Adachi S, Monzawa S, et al. High resolution CT findings of pulmonary epithelioid hemangioendothelioma: unusual manifestations in 2 cases. *J Thorac Imaging* 2005; 20: 236-8.
1. Jinghong X, Lirong C. Pulmonary epithelioid hemangioendothelioma accompanied by bilateral multiple calcified nodules in lung. *Diagn Pathol* 2011; 6: 21. doi:10.1186/1746-1596-6-21.

INCREASED ECHOGENICITY OF THE INTERHEMISPHERIC SULCUS: A POTENTIAL DIAGNOSTIC PITFALL ON CRANIAL ULTRASOUND OF THE NEWBORN

TO THE EDITOR:

We are writing in response to the original article published by Gover et al. [1]. In addition to its use as a screening tool in healthy newborns, cranial ultrasound is one of the most common diagnostic imaging studies performed on preterm infants hospitalized in the neonatal intensive care unit. The most common indication for this study is the evaluation of suspected intracranial hemorrhage. Preterm newborns are at risk for hypoxic-ischemic insults that can lead to hemorrhage, which usually originate in the subependymal germinal matrix – the site where neuronal proliferation occurs, as neuroblasts divide and migrate into the cerebral parenchyma [2]. Supplying this metabolically active area of differentiating cells is a primitive and fragile retelike capillary network that is susceptible to rupture and can easily bleed upon minor insult.

Severe germinal matrix hemorrhages can result in hemorrhage in the white matter of the brain parenchyma adjacent to the lateral ventricles, classified as grade IV (this classification system was initially developed for computed tomography and later adapted for ultrasound use) [2]. At times, however, a major hypoxic insult may result in a bleed more distal than the lateral ventricles, which manifests as an intracerebral hemorrhage.

On ultrasound, acute hemorrhage appears as a hyperechoic structure either within the brain parenchyma or within the ventricles. With further progression of the bleed, the clot liquefies and becomes progressively more hypoechoic, and ultimately anechoic.

The interhemispheric fissure is usually seen on brain sonography of preterm infants, even if the major sulci have not yet developed. The surface of the interhemispheric brain, in contrast to the convex surface of the brain, is convoluted with many ridges. These ridges extend deep into the hemispheres lateral to the fissure. The surface of these ridges appears hyperechoic.

Rarely on brain sonography of preterm infants is a hyperechoic focus seen between the ridges of the interhemispheric region that mimics intracerebral hemorrhage. This hyperechoic focus is a well-recognized artifact and was initially described by Bowerman in 1987 [3]. When a cerebral sulcus or edge of a gyrus is imaged along its long axis, it may

appear relatively hyperechoic with respect to adjacent parenchyma, mimicking a focal mass lesion. This "pseudolesion" is mostly seen on coronal images, when a normal sulcus is imaged tangentially as it courses around the gyrus. Rotation of the transducer to an orthogonal plane can confirm that the brain parenchyma is normal. Other features that suggest a "pseudolesion" are the continuity of the echogenic focus with normal sulci, and lack of a mass effect [4].

We present two patients [Figures 1 and 2] in whom a hyperechoic focus was seen in the interhemispheric fissure, but on follow-up brain sonography and in one case magnetic resonance imaging did not demonstrate the presence of hemorrhage whatsoever, and the infants did well on clinical follow-up. We present these cases to alert radiologists to this variant appearance of a normal structure that could create a diagnostic pitfall, erroneously attributing the hyperechoic lesion we describe in this letter as an intraparenchymal hemorrhage.

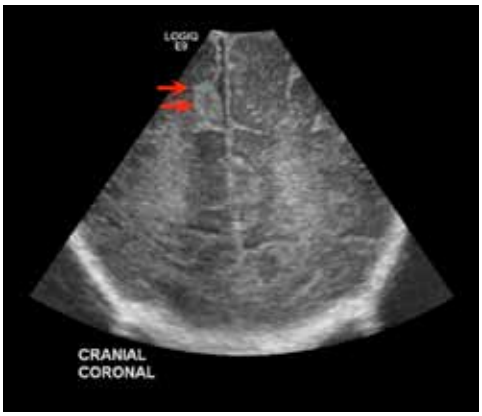

Figure 1. Cranial ultrasonography to assess for intracranial bleed in a 5 day old male with respiratory distress. The study was obtained with a 10 MHZ transducer and the coronal views demonstrate the hyperechoic focus between the ridges of the interhemispheric fissure. The interpreting radiologist mistakenly diagnosed this focus as an intraparenchymal hemorrhage. The focus was not seen on a follow-up cranial ultrasound. The patient underwent an MRI of the brain, which was also negative.

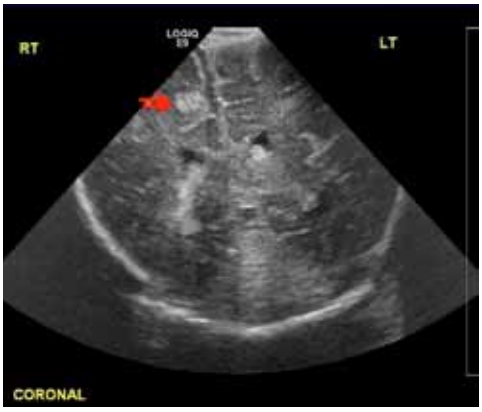

Figure 2. A 3 week old female with congenital diaphragmatic hernia. The patient was receiving ECMO (extracorporeal membrane oxygenation), and a routine cranial ultrasound was performed with a 10 MHZ transducer to assess for intracranial hemorrhage. Coronal and sagittal views of the brain did not show a hemorrhage, but a hyperechoic focus was seen between the ridges of the interhemispheric fissure. In a follow-up ultrasound this echogenic focus was not visualized at all.

A. Franco MD[1], K.N. Lewis MD[1], H. Maresky MD[2] and I. Shelef MD[2]

[1]Dept. of Radiology, Georgia Health Sciences University, Augusta, Georgia, USA
[2]Dept. of Diagnostic Imaging, Soroka University Medical Center, Beer Sheva, Israel [ilans@clalit.org.il]

References

1. Gover A, Bader D, Weinger-Abend M, et al. Head ultrasonograhy as a screening tool in apparently healthy asymptomatic term neonates. *IMAJ Isr Med Assoc J* 2011; 13 (1): 9-13.
2. Volpe JJ. Neurology of the Newborn. 4th edn. Philadelphia: WB Saunders, 2001: 428-93.
1. Bowerman RA. Tangential sulcal echoes: potential pitfall in the diagnosis of parenchymal lesions on cranial sonography. *J Ultrasound Med* 1987; 6: 685-9.
2. Siegel MJ. Chapter 3: Brain. In: Pediatric Sonography. St. Louis, MO: Lippincott Williams & Wilkins in conjunction with Washington University School of Medicine, 2010.

Capsule

Yeast prion protein responds to an environmental stressor

It is not clear if prion induction in yeast is truly linked to physiological roles. Suzuki et al. show that the yeast prion protein Mod5 (a transfer RNA isopentenyltransferase) responds to an environmental stressor by converting to an aggregated amyloid form, which leads to phenotypic changes in cell metabolism and drug resistance. Introduction of Mod5 amyloid into yeast resulted in the formation of a dominantly heritable prion state (*MOD*$^{+}$), in which Mod5 is aggregated. (*MOD*$^{+}$) yeast showed high ergosterol levels and acquired resistance to several antifungal agents. Selective pressure by antifungal drugs on non-prion (*mod*$^{-}$) yeast induced the (*MOD*$^{+}$) prion state, formation of amyloid, and increased cell survival.

Science 2012; 336: 355

Eitan Israeli

"Life is what happens when we are making other plans

Allen Saunders (1899-1986), American writer, journalist and cartoonist"