# Finding answers quickly through *Clinical Q&A*

This column was written by Dr. John Hoey, CMAJ's editor-in-chief.

Clinical Q&A has been one of the most popular features of *CMA Online* since it was launched 18 months ago. The feature, which now has about 200 participants from around the world, serves as an electronic form of corridor consultation.

Electronic discussion groups like it have not indicated that they are going to transform medicine, but they do point to 2 of the Internet's biggest benefits: ease of use and speed. The discussion outlined in the following paragraphs took place over a 12-hour period in December, and brought together 3 physicians from 3 provinces. The first message was posted by Dr. Lorne Brandt of Brandon, Man., on Dec. 12 at 12:07 am; Dr. Joel Lexchin of Toronto made the first reply 10 hours later, and the final response, from Dr. David Maxwell of Halifax, was posted at 12:37 pm. The speed with which this exchange of ideas took place offers a lesson.
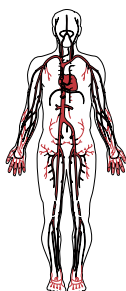
Dear colleagues (1):

Is there a cardiologist lurking among us? I recently started a young lad on imipramine for attention-deficit hyperactivity disorder [ADHD], with good results. However, the baseline ECG reported a shortened P-R interval [and] absent delta wave, consistent with Lown-Ganong-Levine [LGL] syndrome.

What is this? Do I need to worry about the conduction effects of arrhythmogenic potential of the tricyclic with this? The mother did say today that she wondered if the boy had experienced palpitations, as he sometimes clutched his chest and referred to his heart.

Dear colleagues (2):

I don't know what Lown-Ganong-Levine syndrome is, but there have been cases reported of sudden death, presumably from cardiac toxicity in children using desipramine. See *Med Lett Drugs Ther* 1994;36:109-10.

Dear colleagues (3):

I'm surprised nobody has jumped in with an explanation of LGL. Basically it looks like Wolff-Parkinson-White [WPW] syndrome, minus the delta wave (i.e. normal looking QRS, but short P-R interval). It behaves the same, too, and leads to episodes of supraventricular tachycardia [SVT]. Unlike WPW, which is usually due to an accessory pathway conducting around the AV node, LGL is more likely caused by an abnormally rapid conduction through the AV node. (Perhaps because it is congenitally small, allowing the impulse to get through it faster? Or because of intranodal conducting fibres that "short-circuit" the delay in the AV node? This is not well understood.) The episodes of SVT seem to be associated with AV nodal re-entry in most cases.

What risks do tricyclic antidepressants [TCAs] pose in this case? Given the poorly understood mechanisms underlying this syndrome, it is probably impossible to predict with accuracy, but I think it is at least possible that TCAs might increase the risk of episodes of SVT. However, SVT in a kid is likely to be very benign.

If he does have an episode you will have your answer, and you aren't going to do him any real harm. I think you should discuss it explicitly with both patient and mother, though. Essentially you are weighing the small risk of provoking a benign episode of SVT against the presumably greater benefit of treating his ADHD.

A lesson learned

There you have it — one exchange from among the hundreds that have appeared on *CMA Online*'s *Clinical Q&A* since its launch. We are pleased that this service is growing in popularity, and think the reasons are obvious: it offers nationwide corridor consultations, 24 hours a day.

Any practising physician is welcome to join the discussion. We require a written request on professional letterhead. It should be sent to Dawna Ramsay, Publications Department, CMA, PO Box 8650, Ottawa ON K1G 0G8; (fax) 613 523-0937. The physician's e-mail address and CMA membership number or licensure number must be included. American physicians may use their Drug Enforcement Agency number.