

and recent immigrants from Western Africa than among other black patients. Such patients tend to have a “resistant” hypertension, with or without a susceptibility to hypokalemia, and typically have a good response to the addition of spironolactone or amiloride.

In clinics serving such populations, following Young’s recommendations, especially in the case of older patients who have an increased coinci-

dence of silent adrenal nodules, could lead to unnecessary invasive diagnostic procedures such as adrenal venous sampling. Therefore, clinical considerations, not just hormonal data, should guide the choice of further interventions.

Irene Gavras, M.D.

Boston University School of Medicine  
Boston, MA 02118  
igavras@bu.edu

## Urine Fluorescence in Ethylene Glycol Poisoning

**TO THE EDITOR:** With regard to the Image in Clinical Medicine by McStay and Gordon (Feb. 8 issue),<sup>1</sup> the presence of urine fluorescence can be short lived, less than 4 hours from the time of ingestion.<sup>2</sup> This brief duration poses the potential for false negative results. Not all brands of antifreeze contain fluorescein as a colorant for the detection of radiator leaks. Other researchers have reported that urine specimens from children may fluoresce without an exposure to antifreeze.<sup>3,4</sup> A database lists 148 substances, including a number of drugs, food products, toxins, and endogenous compounds, that can contribute to urine fluorescence and the potential for false positive results.<sup>5</sup> Urine fluorescence as an adjunctive tool in the evaluation of ethylene glycol ingestion may be helpful, but physicians should be aware of the considerable limitations of this test, including both false negative and false positive results.

Mark L. Winter, Ph.D.

Wayne R. Snodgrass, M.D., Ph.D.

University of Texas Medical Branch  
Galveston, TX 77555-1175  
mwinter@utmb.edu

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4. Parsa T, Cunningham SJ, Wall SP, Almo SC, Crain EF. The usefulness of urine fluorescence for suspected antifreeze ingestion in children. *Am J Emerg Med* 2005;23:787-92. [Erratum, *Am J Emerg Med* 2006;24:396.]
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**TO THE EDITOR:** McStay and Gordon report on a patient with antifreeze (presumably ethylene glycol) intoxication. The urine of their patient showed blue fluorescence under ultraviolet excitation. Fluorescein is a chromophore with a high quantum yield of fluorescence. Therefore, assessment of urine fluorescence was suggested in cases of suspected ethylene glycol intoxication.<sup>1</sup> The emission spectrum of fluorescein peaks at approximately 540 nm (green), and it is virtually absent below 500 nm (blue).<sup>2</sup> Thus, the deep-blue urine fluorescence described by McStay and Gordon presumably originated from a fluorophore other than fluorescein. A number of unknown fluorophores may be assumed to be the source of the fluorescence observed. For instance, a strong blue fluorescence was observed after excitation with ultraviolet light for certain ion–chelate complexes.<sup>3</sup> Only a strong green fluorescence points to fluorescein ingested with antifreeze. In a case of unusual fluorescence, as shown here, one should consider substances other than ethylene glycol as the source of intoxication.

Thomas Theelen, M.D., Ph.D.

Radboud University Nijmegen Medical Center  
6500 HB Nijmegen, the Netherlands

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3. Bekiari V, Lianos P. Strongly luminescent poly(ethylene glycol)-2,2-bipyridine lanthanide ion complexes. *Adv Mater* 1998;10:1455-8.

**THE AUTHORS REPLY:** We agree with Winter and Snodgrass and strongly caution clinicians against

making critical management decisions solely on the basis of urine fluorescence. This recommendation is especially pertinent in the pediatric population, given the extremely high rate of fluorescence in patients who have not been poisoned.<sup>1,2</sup> Difficulties with positive and negative controls as well as interobserver variability are also important considerations. We acknowledge that the result of our own bedside test could represent a false positive finding from one or more fluorophores, as Theelen points out. In a report by Parsa et al., images of fluorescent urine samples from children who have not been poisoned retain their blue hue despite elevated levels of sodium fluorescein.<sup>2</sup>

Given the lack of rapidly available testing for ethylene glycol in many centers, we still believe

that urine fluorescence may have some use as an adjuvant test in adults. Above all, we strongly urge clinicians with questions regarding testing and management of both suspected and confirmed poisonings to consult with their local poison-control center.

Christopher M. McStay, M.D.

Peter E. Gordon, M.D.

New York University  
New York, NY 10016  
mcstac01@med.nyu.edu

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## Superior Athletic Performance Two Decades after Cardiac Transplantation

**TO THE EDITOR:** Cardiac-transplant recipients have a severely reduced exercise capacity (defined as the maximum volume of oxygen consumed [ $\text{VO}_2$ ]), in part because of chronotropic incompetence associated with cardiac denervation.<sup>1</sup> The upper limits of athletic performance and exercise capacity in the long term after cardiac transplantation are not clear. We report superior exercise capacity and athletic performance in a man two decades after he underwent cardiac transplantation.

The patient underwent cardiac transplantation in August 1986, at 26 years of age. Other than a history of post-transplantation hypertension that was easily controlled with the use of calcium-channel blockers, he has had an uneventful course since the surgery, with no biopsy-proven or clinical evidence of rejection. He has been physically active, performing aerobic exercise 2 to 3 days per week. In October 2004, at 45 years of age, he participated in a 12-week exercise study at our center. His peak  $\text{VO}_2$  after training (59 ml per kilogram of body weight per minute) was similar to the peak  $\text{VO}_2$  achieved by age-matched, endurance-trained male athletes.<sup>2</sup> He had a rapid increase in heart rate from rest to peak exercise (change in heart rate, 81 beats per minute), and the rate returned toward baseline during recov-

ery. His peak heart rate during exercise (177 beats per minute) was similar to the rate among age-matched healthy persons.<sup>3</sup> Eighteen years after the transplantation, he completed a half-ironman triathlon in 6 hours and 28.7 minutes (finishing as the 454th of 558 competitors). A year later, he completed the same triathlon in 6 hours and 15.9 minutes (416th of 611 competitors). He also completed an Olympic distance triathlon in 3 hours and 2.1 minutes (75th of 126 competitors).

Cardiac-transplant recipients typically attain their highest peak  $\text{VO}_2$  within the first year after surgery.<sup>4</sup> In contrast, our patient's experience illustrates that long-term aerobic training may result in a dramatic improvement in exercise capacity. One contributing factor is that his heart-rate response to stress was similar to that in cardiac-transplant recipients who have functional evidence of cardiac reinnervation.<sup>5</sup> This patient's history shows that superior athletic performance can be achieved two decades after cardiac transplantation.

Mark Haykowsky, Ph.D.

Wayne Tymchak, M.D.

University of Alberta  
Edmonton, AB T6G 2B7, Canada  
mark.haykowsky@ualberta.ca