

unless the patient was in that ward before admission to the ICU and will be returned to the care of a team that knows him or her.

We agree with Karakitsos and Karabinis that having information on families' perception of the brochure, in general and for specific subgroups, would be of interest. In our study, the controlled design of the intervention did not permit the interviewer to ask study participants whether they read the brochure and how they perceived the information in it. It would be of value to understand through interviews the specific ways in which the communication strategy and the brochure helped the family.

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The Incidentally Discovered Adrenal Mass

TO THE EDITOR: The Clinical Practice article by Young (Feb. 8 issue),¹ on the incidentally discovered adrenal mass, provides a personal view of how to approach this clinical problem, but it does not mention relevant guidelines from a recent international symposium.^{2,3} An important recommendation endorsed at the meeting was that initial testing for pheochromocytoma include the measurement of fractionated metanephrines in plasma, urine, or both. This recommendation recognizes that a missed diagnosis (due to inadequate sensitivity) can have catastrophic consequences for the patient. Young emphasizes specificity and in this respect suggests that urinary measurement is preferable to plasma measurement. However, Lenders et al.⁴ reported a specificity of 69% for urinary fractionated metanephrines and 89% for plasma metanephrines. Apart from that report and another by Unger et al.,⁵ which also showed better diagnostic accuracy with the use of the plasma test than the urine test, there are no reliable head-to-head comparisons of these two tests of fractionated metanephrines. Furthermore, urinary dopamine is derived mainly from renal extraction and decarboxylation of circulating levodopa and is not a reliable marker for dopamine-producing tumors.²

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TO THE EDITOR: Primary aldosteronism due to an aldosterone-producing adenoma and resulting in surgically reversible hypertension is extremely rare. Defined by a ratio of the plasma aldosterone concentration to plasma renin activity of more than 20, primary aldosteronism in the absence of an aldosterone-producing tumor is considered to be idiopathic. Even during treatment with diuretics, the pattern of extreme suppression of plasma renin activity to undetectable levels (i.e., <0.1 ng per milliliter per hour) in the presence of a plasma aldosterone concentration of 10 to 20 ng per deciliter (i.e., a ratio of 100 to 200) is not unusual in black persons with hypertension, and in our experience, this pattern appears to be more prevalent among blacks from the Caribbean region

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.

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Urine Fluorescence in Ethylene Glycol Poisoning

TO THE EDITOR: With regard to the Image in Clinical Medicine by McStay and Gordon (Feb. 8 issue),¹ the presence of urine fluorescence can be short lived, less than 4 hours from the time of ingestion.² 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.^{3,4} 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.⁵ 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.

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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.¹ The emission spectrum of fluorescein peaks at approximately 540 nm (green), and it is virtually absent below 500 nm (blue).² 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.³ 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.

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