the reactants are exposed in the large flask is not the principal cause of the phenomenon.

I established that the size of the reaction vessel only affected the last stage of the reaction. The test could be performed in a small glass centrifuge tube up to and including the penultimate step (addition of sulfamic acid). Transfer of the reaction mixture to a 1-L volumetric flask containing sodium hydroxide produced the same high absorbance with a 3000 mg/L solution of salicylate that had been observed when the entire reaction was carried out in a 1-L flask.

Thus it is possible that a surface effect of glass modulates the formation of the chromophores produced from nitrated salicylate and acetaminophen in alkaline solution.

Because both phenol and carboxyl groups will be ionized at the high pH of the final reaction mixture, nitrated acetaminophen will have one charged group whereas nitrated salicylate will presumably have two. A glass surface interaction involving these groups may explain their differing behavior in alkaline solution. Examination of the absorption spectra associated with the two colored products may yield further information, as they are known to differ (3).

I am not prepared to comment on the suitability of various types of laboratory reaction vessels. However, laboratories using the original Glynn–Kendall technique may be using large glass or plastic reaction tubes (glass extraction tubes are used in our laboratory) because these tubes better contain the vigorous effervescence that results when sulfamic acid is added during the procedure. A recent modification of the Glynn–Kendall method allows the whole reaction to be done rapidly in conventional small test tubes because the concentration of sodium nitrite in the reaction mixture is so low.

Clearly it is unwise to rely on a single arithmetical correction when the reaction conditions for salicylate interference in the colorimetric acetaminophen method are incompletely understood. I therefore support the recommendation of Reed et al. (1) and Rosenbaum et al. (7) that each laboratory should check the degree to which salicylate interferes in their own assay system.

References

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Increased Urinary Vanillylmandelic Acid Excretion in a Patient with Essential Hypertension and Hyperthyroidism

To the Editor:

Measurement of vanillylmandelic acid (VMA) in 24-h urine specimens is useful in the diagnosis of pheochromocytoma (1,2). A major problem is that patients with essential hypertension occasionally have borderline-increased values for urinary VMA (3), 7–10 mg/24 h (normal = 2–7 mg/24 h), especially after a stressful event such as a hypertensive or hypertensive episode within a day or two preceding specimen collection. Thus a value exceeding 10 mg/24 h almost always indicates a pheochromocytoma.

We report here a patient with essential hypertension and hyperthyroidism, whose urinary excretion rate was abnormally high. Because the clinical features of hyperthyroidism and pheochromocytoma are similar, pheochromocytoma may be wrongly diagnosed on the basis of the increased VMA excretion.

A 55-year-old woman complaining of headaches and sweating was found to have a pulse rate of 120/min and blood pressure of 190/110 mmHg. Results of physical examination were otherwise normal. Relevant laboratory data included values for serum electrolytes, urea nitrogen, creatinine, and glucose that were within normal limits. Urinary VMA on two occasions was 13 mg/24 h. The concentration of plasma catecholamines as measured by liquid chromatography was 600 ng/L (normal 250–620 ng/L). The serum thyroxin concentration was 13.4 mg/L (normal 50–120 mg/L), the free thyroxin index 6.1 (normal 2.2–4.7). Results of ultrasound examination and an abdominal computerized tomography scan were negative for pheochromocytoma.

The patient’s blood pressure declined to normal on treatment with antihypertensive drugs (thiazides and propranolol). Urinary VMA values eight and nine days after therapy was begun were 9 and 7 mg/24 h, respectively. Essential hypertension and hyperthyroidism were diagnosed.

Many of the effects induced by excessive quantities of thyroid hormones resemble those induced by epinephrine: tachycardia, increased cardiac output, and enhanced calorigenesis. The mechanisms responsible for the increased adrenergic activity are not known. Secretion rates and the concentrations in plasma of both epinephrine and norepinephrine are normal in hyperthyroidism (5,6). Urinary excretion of VMA has previously been reported to be either normal or decreased (7).

The reason for the increased urinary VMA seen in our patient is not clear, but the possibility of co-existing hyperthyroidism should be considered in a patient with essential hypertension who has an increased 24-h urinary excretion of VMA.

References

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