present here, the samples were at least 7 h apart but not more than 24 h apart, most differences being 12–17 h. LD-1 was determined by the Roche procedure, and LD was determined by using the pyruvate → lactate assay of Biomedix, at 30 °C. Calculations were based on using the highest value of LD-1 from each patient to discriminate between infarct and non-infarct.

In a population of 47 patients, 19 were considered not to have had infarcts (MI−) and 28 to have had infarcts (MI+). Selecting the best cutoff point for the ratio as 27.5%, there were six false positives and six false negatives (Figure 1).

This may be calculated to yield a predictability of 68.4% for no infarct or 78.5% for infarct. Although we concur that the proper use of LD-1 may facilitate the early diagnosis of a myocardial infarct, we have not found the LD-1/LD ratio to be nearly as predictive of myocardial infarct as have our colleagues. It is unfortunate, in both studies, that our samples are relatively small and biased, in that the patients were already selected for the CCU. A test of true predictability ought to be done on a totally random assortment of patients from whom, unfortunately, it would be much more difficult to collect data.

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Naproxen Interferes Positively with 5-Hydroxyindoleacetic Acid Assay

To the Editor:

Over the last 18 months we have found increased urinary excretion of 5-hydroxyindoleacetic acid (5HIAA) in four patients who were taking an anti-rheumatoid drug, naproxen. We use the Goldenberg method (1) for 5HIAA, and the values found in these patients were unequivocally high, ranging from three- to sevenfold the upper reference value. When naproxen was withdrawn, excretion of 5HIAA returned to normal within three days. To confirm interference with the 5HIAA assay, two of us took naproxen; values for apparent 5HIAA in urine increased by four- to fivefold within two days.

Investigation of the urine by chromatography indicated that the increase in apparent 5HIAA was due to interference by a metabolite of naproxen in the nitrosonaphthol color reaction for 5HIAA, rather than an effect of naproxen on serotonin metabolism. We believe that all methods in which this color reaction is used will be so affected by the naproxen metabolite, including the widely used Goldenberg modification (conversion of the violet chromophore to blue by mercaptoethanol), a modification that has been regarded as highly specific for 5HIAA.

The only reference to this drug effect on 5HIAA excretion appears to be in the Proceedings of the 1976 Scandinavian Rheumatology Congress (2). We believe that this effect deserves to be more widely known in order to prevent extensive investigations for carcinoid syndrome, as happened in the first of our patients.

References

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Lack of Influence of Some Antibiotics on Various Thyroid Hormone Assays

To the Editor:

Because sera submitted for laboratory tests may contain antibiotic agents, we have examined the effect of some of them on assays for thyroid hormones.

We obtained 20 mL of venous blood from each of eight healthy white male volunteers and prepared 1.9-mL serum aliquots from each sample. These men were 25 to 30 years old, and had no history of prior thyroid disease or ingestion of medication during the preceding four weeks.

Laboratory-grade antibiotic powders (USP) were dissolved in appropriate buffers (Table 1) and stored at −70 °C. Control tubes containing only buffer were treated in an identical manner.

We added 0.1 mL of antibiotic solution or buffer to each serum aliquot, and kept the mixtures at room temperature for 1 h. The final antibiotic concentrations are recorded in Table 1. Antibiotics were allocated such that each agent was added to serum from four subjects and serum from each subject was assessed in the presence of four different antibiotic agents and corresponding controls.

All solutions were encoded with random numbers before submission for assay.

The following assays were performed during a single day by one technician, according to methodology supplied by the manufacturers: thyroxin (T₄) radioimmunoassay (Nuclear Medical Laboratories, Dallas, TX 75247); thyrotropin (TSH) radioimmunoassay (Beckman Diagnostics, Fullerton, CA 92634); triiodothyronine (T₃) radioimmunoassay (Nuclear Medical Laboratories); and T₃ resin uptake (Nuclear Medical Laboratories).

The result of each serum assay was within 5% of that for its control. Furthermore, all hormone concentrations were within normal limits for our laboratory, despite possible dilutional or pH artifacts introduced by the addition of antibiotic solutions.

A recently published review (1) of drugs that may affect thyroid function tests mentions only one antibacterial agent, p-aminosalicylic acid, which may interfere with measurement of T₄, free T₄ index, and presumably TSH. Moulding and Fraser (2) have identified additional instances in which hypothyroidism resulted from ingestion of ethionamide, with suppression of T₄, by both column and radioimmunoassay.

Each of the above reports suggests that these two drugs directly (rather than artifactually) affect thyroid hormone assay. This may, in turn, reflect the action of either the native drug or its metabolites on thyroid physiology, hormonal protein binding, or hepatic metabolism (3). Thus, penicillin has been shown to decrease the concentration of protein-bound iodine and T₄ (Murphy-Pattie method) through competition for sites on thyroid-binding prealbumin (4).

To date, no antimicrobial agent has been reported to interfere with radioimmunoassay for T₃ or T₄, unless the thyroid gland itself was affected. The present investigation suggests that therapeutic concentrations of commonly used antibiotic agents do not interfere with such tests.

References
2. Moulding, R., and Fraser, R., Hypothy-