Recent hemodynamic studies in coronary patients indicate increased cardiac and stroke index without changes in mean arterial or pulmonary wedge pressures, which is consistent with the effect of food on STI in our study. Previous studies of normal conscious dogs were also in agreement, indicating an enhanced inotropic state during the postprandial period. These findings could have had a bearing on the altered thallium myocardial kinetics reported by Wilson and colleagues, perhaps through alterations in flow, and as the authors suggest, myocardial metabolism.

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REFERENCES

To the Editor:

Dr. Spodick's comments concerning the effect of eating on cardiac hemodynamics are greatly appreciated. With specific reference to our study, they point out the possibility that the increased clearance of thallium-201 from the myocardium may have been due in part to a decrease in coronary resistance and, therefore, an increase in coronary blood flow (since arterial pressure also increased), as observed by Vatner et al. However, previous studies by us failed to show an increase in coronary blood flow during intravenous infusion of glucose-insulin-potassium solution, even though thallium-201 myocardial clearance increased. We cannot exclude an increase in coronary blood flow in the patients who ate, but suspect that an independent effect of insulin on the myocardial handling of $^{201}$TI is also involved. Further studies are needed to elucidate the relative importance of flow and metabolism in $^{201}$TI kinetics.

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Effects of Beta-Adrenergic Agents on Hypokalemia

To the Editor:

We read with interest the paper of Rohr et al entitled, "Efficacy of parenteral albuterol in the treatment of asthma" (Chest 1986; 89:348). The authors discuss different metabolic effects of adrenergic agents parenterally administered. They note that these drugs could produce hypokalemia, potentially dangerous especially in patients with previous borderline or low serum K+ levels. This effect was previously described with different β-adrenergic agents administered either by inhalation or per os. In this context, it is interesting to note our findings in a group of 12 patients with COPD to whom we administered a β2-adrenergic agent (pirbuterol, 15 mg p.o.). None was on steroid or digitalis therapy. Aminophylline and diuretic therapies were withdrawn 24 hr before study. In these patients, we measured plasma K+ by means of a K+ sodium-potassium analyzer and arterial blood samples for gas analysis obtained through an indwelling catheter placed in the humeral artery. The samples were obtained before and at 60, 120 and 150 min after pirbuterol administration. Control plasma K+ (±SEM) was 4.25±0.14 and 3.76±0.14 at 60 and 120 min after pirbuterol administration, respectively. These values were significantly different as compared with control values (p<0.01). Maximal decrease of plasma K+ (∆max K+) was 0.58±0.10, range 0.10-1.20 mEq/L, and occurred in nine of the patients at 60 or 120 min. No significant changes were found in PCO2 or pH control values when compared with those at the moment of ∆max K+ (PCO2 47.8±2.4 vs 49.1±2.3 mm Hg; pH 7.39±0.01 vs 7.36±0.01). A significant correlation was found between control plasma K+ values vs ∆max K+ after pirbuterol administration (Fig 1).

That signifies that the patients with higher control plasma K+ levels would respond strongly to β2-adrenergic stimulation, while in those who have lower plasma K+ levels this response is less evident.

As far as we know, this response has not been previously described. We believe that this finding has clinical implications suggesting that previously hypokalemic patients respond to β2-adrenergic stimulation with a lesser decrease in plasma K+ levels. The mechanisms involved could be attributed to the decrease in the activity of the Na+-K+ pump ATPase activated and the modification of the cellular membrane permeability to ions as the concentration of extracellular K+ diminishes.xv

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When data suggest...