The clinical observation that diabetic patients who exercise require less insulin therapy than those who do not, led to interest in the behaviour of circulating insulin concentrations during exercise. An increase in insulin concentration should result in increased glucose transport, and thus a decrease in blood glucose concentration. Studies of diabetic patients have shown (Sanders et al. 1964) that blood glucose concentration does, in fact, decrease as a result of exercise. However, since the same effect can be elicited in pancreatectomized animals (Ingle et al. 1950, Goldstein et al. 1953, Dulin and Clark 1961), it seems impossible that an increase in plasma insulin concentrations could be the only cause of the increased glucose transport. Thus a decrease in plasma insulin concentrations was also a possibility, and because of the necessity of preserving the blood and liver glucose for utilization by the central nervous system, this latter possibility seemed logical. A few studies of the effect of various types of exercise on plasma immunoreactive insulin (IRI) concentrations have demonstrated a decrease (Cochrane et al. 1966, Rasio et al. 1966, Hunter and Sukkar 1968). A study of insulin secretion in both normal and diabetic patients showed no increase as a result of exercise (Nikkilä et al. 1969). Studies of exercising animals have shown that insulin concentrations could be decreased during exercise even when glucose was infused (Issekutz et al. 1967, Wright and Malaisse 1968).

As part of two rather comprehensive metabolic studies at this laboratory (in part in collaboration with University Hospital of Oslo), systematic investigations of changes in plasma immunoreactive insulin (IRI) levels with exercise have been carried out. In one of these investigations, healthy young men in the post-absorptive state exercised at work loads of from 20% to 90% of their previously
measured maximal aerobic power (maximal oxygen uptake, \( \text{max } \dot{V}_{O_2} \)) (Pruett 1970a,b). In one part of this investigation the subjects lived on one of three carefully measured diets in which the fat and carbohydrate (but not the protein) content were varied. They exercised alternately on the bicycle ergometer and the treadmill in 45 minute work bouts interspaced with 15 minute rest intervals for six hours or to exhaustion, whichever came first, at work loads of 20%, 50% and 70% \( \text{max } \dot{V}_{O_2} \). In another part of the investigation, the subjects lived on their own normal diet. Some exercised on the bicycle ergometer continuously, without pause, until exhausted at 70% \( \text{max } \dot{V}_{O_2} \). Others exercised to exhaustion twice, with a 15 minute pause in between, at 85–90% \( \text{max } \dot{V}_{O_2} \). A separate investigation was a study of pre-pubertal boys, exercising without pause on the treadmill for one hour at 70% \( \text{max } \dot{V}_{O_2} \) (Oseid and Hermansen 1970). In all investigations blood glucose and plasma insulin levels before, during and after exercise were followed.

The purposes of these investigations were to ascertain:
1) whether or not plasma IRI concentrations did, in fact, decrease as a result of exercise; 2) whether or not a change in insulin concentration was dependent upon the duration and intensity, or the type of exercise; 3) whether or not diet had an appreciable effect upon a change in plasma IRI concentration with exercise; and 4) whether or not the normal interdependence of circulating insulin and glucose levels upon each other remained unaltered during exercise.

**EFFECT OF INTENSITY, DURATION AND TYPE OF EXERCISE**

Interrupted exercise (20% to 70% \( \text{max } \dot{V}_{O_2} \)) in young men (Pruett 1970a)

Plasma IRI concentrations fell consistently and significantly (Fig. 1) at work loads of approx 20%, 50% and 70% \( \text{max } \dot{V}_{O_2} \) when subjects exercised alternately on the bicycle ergometer and the motor driven treadmill for 45 minute work bouts interrupted by 15 minute rest periods for six hours or until exhaustion, whichever came first. There was a small, but significant fall in 16 of 17 subjects during six hours of exercise at 20% \( \text{max } \dot{V}_{O_2} \). In the seventeenth experiment, the subject exhibited a variable response with a net rise in plasma IRI after six hours. Individual variations from hour to hour were great at this work load. At work loads of approximately 50% and 70% \( \text{max } \dot{V}_{O_2} \) the decrease in plasma IRI concentration was significant (\( p < .001 \)) during the first 45 minute exercise period, and the IRI levels continued to decrease until, at exhaustion, they reached an average 60 ± 7% of the preexercise value (at 50% \( \text{max } \dot{V}_{O_2} \)) and 58 ± 6% of the preexercise value (at 70% \( \text{max } \dot{V}_{O_2} \)). Blood glucose concentrations during these same experiments decreased in approximately the same manner (Fig. 2). There was evidence at 50% \( \text{max } \dot{V}_{O_2} \) that plasma IRI concentrations showed a greater decrease (\( p < .05 \)) during the first hour of work than did blood glucose.