astragalus, so prominent in later carnosaurs, can be seen (Fig. 1)[1]. This also implies that the AM joint could have evolved from a crocodiloid joint.

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Reduction in plasma calcium during exercise in man

RUBEN and Bennett[1] reported increased plasma calcium concentration [Ca_p] following exercise in various species. They suggested that increased [Ca_p] is due to bone resorption, implying an increase in total plasma calcium content (Ca_t). However, the rise in [Ca_p] following exercise might merely result from a reduction in plasma volume (PV)[2] with or without change in Ca_t.

We have now investigated the responses of PV, [Ca_p] and Ca_t in 13 men (22 ± 1 yr, 73.9 ± 2.1 kg and mean maximal oxygen uptake of 56.4 ± 2.2 ml kg^{-1} min^{-1}) who worked on a cycle ergometer at one of three randomly ordered work intensities for 6 min on three different days (Table 1). PV was measured with Evans blue dye[2], and change in PV was calculated from the haematocrit[3]. [Ca_p] was analysed by atomic absorption spectrophometry. Ca_t was calculated from [Ca_p] and per cent change in PV[4].

These results, from men, show that during exercise, [Ca_p] increased to a constant level while PV and Ca_t decreased progressively with increasing exercise level (Table 1). The rate of decrease in PV was greater than that for Ca_t (Fig. 1). This suggests that the increase in [Ca_p] during exercise can be attributed to a more rapid loss of fluid from the vascular space and does not necessarily require a significant net influx of calcium from extravascular sources. Thus, the conclusion of Ruben and Bennett[1] should be evaluated cautiously until measurements of vascular fluid and calcium shifts are available.

Table 1 Effect of light, moderate and heavy muscular activity on PV, [Ca_p] and (Ca_t)

Exercise level (W)	PV (ml)	[Ca_p] (mmol l^{-1})	Ca_t (mg)
Rest	3,504 ± 83	2.47 ± 0.03	347 ± 9
Light (100)	3,388 ± 91*	2.53 ± 0.03*	344 ± 9
Moderate (175)	3,208 ± 94*	2.55 ± 0.03*	324 ± 10*
Heavy (225)	3,072 ± 83*	2.57 ± 0.03*	317 ± 9*

Values are mean ±s.e.
* $P < 0.05$ compared with corresponding rest value.

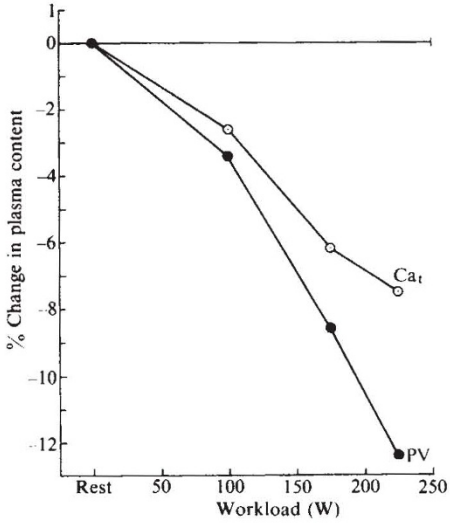

Fig. 1 Proportional changes in PV and Ca_t with graded work intensities of 100, 175 and 225 W. Plotted points are mean values of the percentage change calculated from pre- and post-exercise haematocrit and plasma calcium concentrations[4].

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RUBEN AND BENNETT REPLY—The values cited above by Convertino *et al.* for blood calcium levels during human exercise are not directly relevant to our previous study[1]. We hypothesize that the mechanism resulting in post-exercise hypercalcaemia involves dissolution of a fraction of the crystalline calcium hydroxyapatite compartment of the skeleton. This dissolution is a result of systematic lactic acid accumulation and *p*H depression associated with maximal levels of exercise in vertebrates. The plasma volume and calcium concentrations cited by Convertino *et al.* associated with 'heavy' muscular activity (225 W) represent submaximal exercise levels (about 60–65% of capacity) for humans. Thus, it is unlikely that exercise-related hypercalcaemia of the sort we described occurred in their experimental subjects. Significantly, Greenleaf *et al.*[2] have previously described a dramatic rise in the concentration of calcium ions in plasma immediately following maximal exercise, whereas there is a slight decrease in plasma calcium ion concentration at 43% and 62% work levels (Table 1).

Table 1 Effect of submaximal and maximal work on plasma volume and calcium ion concentration in humans (from Fig. 5 in ref. 2)

% Work load	% Net change in plasma volume	Ca^2 concentration (mmol l^{-1})
Rest	—	1.10
43	−8	1.03*
62	−14	1.02*
100	−16	1.20*

* After 10 min exercise.

Additionally, haemoconcentration cannot possibly account for the exercise-related hypercalcaemia measured in other species in our study. For haemoconcentration to account for the degree of post-exercise hypercalcaemia present in all non-mammalian osseous species, plasma volume reduction of at least 30% (for fish with acellular bone) and 50% (for fish with cellular bone and reptiles) would have to have occurred. Acute plasma volume losses approaching 20–25% are generally associated with hypovolemic shock. There were no indications that any of our experimental animals experienced such a condition. Moreover, the lack of exercise-related hypercalcaemia in non-osseous species investigated (lamprey, shark) seems particularly noteworthy in this respect.

While haemoconcentration may well be a contributing factor to some of the exercise-related hypercalcaemia we describe, it seems unlikely to be a major factor accounting for the phenomena discussed in our paper.

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