Fluid and electrolyte hormonal responses to exercise and acute plasma volume expansion

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Grant, S. M., H. J. Green, S. M. Phillips, D. L. Enns, and J. R. Sutton. Fluid and electrolyte hormonal responses to exercise and acute plasma volume expansion. J. Appl. Physiol. 81(6): 2386–2392, 1996.—To investigate the effect of acute graded increases in plasma volume (PV) on fluid and regulatory hormone levels, eight untrained men (peak aerobic power 45.2 ± 2.2 ml·kg⁻¹·min⁻¹) performed prolonged cycle exercise (46 ± 4% maximal aerobic power on three occasions, namely, with no PV expansion (Con) and after 14% (Low) and 21% (High) expansions, respectively. The exercise plasma levels of aldosterone (Aldo), arginine vasopressin (AVP), and atrial natriuretic peptide (ANP) were all altered by acute PV increases. A pronounced blunting (P < 0.05) of the Aldo response during exercise was observed, the magnitude of which was directly related to the amount of hypervolemia (Con < Low < High). At 120 min of exercise, Aldo concentrations were 660 ± 71, 490 ± 85, and 365 ± 78 pg/ml for Con, Low, and High conditions, respectively. In contrast, the lower AVP and the higher ANP observed during exercise appeared to be due to the effect of PV expansion on resting concentrations. Because osmolality did not vary among conditions, the results indicate that PV represents an important primary stimulus in the response of Aldo to exercise. The lower exercise blood concentrations of both epinephrine and norepinephrine observed with PV expansion would suggest that a lower sympathetic drive may be implicated at least in the lower Aldo responses.

Hypervolemia; exercise; fluid and electrolyte hormones

PROLONGED EXERCISE, particularly in the heat, provokes both fluid and electrolyte disturbances. In the vascular system, these disturbances are characterized by a loss of plasma volume (PV) and an increase in osmolality (28). This condition, often referred to as hyperosmotic hypervolemia, stimulates secretion of fluid and electrolyte regulatory hormones that are designed to minimize the potential imbalance that could result during prolonged exercise (35). The major regulatory hormones have been identified as arginine vasopressin (AVP), aldosterone (Aldo), and atrial natriuretic peptide (ANP) (7, 29, 35). Collectively, these hormones display a variety of functions, acting on the cardiovascular system, sweat glands, and the kidney to defend blood pressure and sodium loss (7, 29, 31).

With training, it has been demonstrated that when exercise exceeds a certain threshold intensity, the increase in, at least, plasma AVP and plasma Aldo is not as pronounced (5). The attenuation in the response of both of the hormones has, in large part, been attributed to an improved protection of both PV and osmolality (5). Curiously, however, when prolonged moderate exercise is performed, before and after training, the eight- to ninefold increase observed in the plasma levels of both of these hormones is not altered (4). These contradictory results raise the possibility that the training-induced effect on the hormonal responses is specific to the exercise protocol employed and dependent on specific adaptations induced by the training.

A better protection of PV and osmolality levels with training could occur either because of accomodations expressed during the exercise itself or because of persistent adaptations induced after the exercise that alter fluid and electrolyte status before the next period of exercise. One such adaptation that alters fluid status before the exercise is an increase in PV (3). The hypervolemia, the magnitude of which may exceed 20% depending on the training status of the participants, the training stimulus, and the environmental conditions (18, 33), has been repeatedly demonstrated to be a characteristic response to, at least, short-term training (3). The training-induced adaptation in PV also appears to be an isosmotic hypervolemia because no alteration in resting osmolality has been found (4, 5).

Because the increase in PV with training appears to convey a larger absolute PV during exercise and a more stable osmolality (3), the hormones associated with fluid and electrolyte homeostasis should be depressed. However, this hypothesis has been difficult to assess during exercise after training because of a variety of other adaptations that occur (15, 17). Maximal aerobic power (VO₂max), for example, appears to increase (4, 5), and when the exercise is adjusted to the increase in VO₂max and performed at the same relative percent before and after training, the effects on plasma Aldo and AVP disappear (5). Moreover, several other adaptations occur with training, including alterations in cardiovascular function, thermoregulation, and muscle metabolism, all of which may impact on the hormonal concentrations associated with fluid and electrolyte balance. These considerations bring into question the independent role of training-induced elevations on PV in modifying the hormonal response. One approach to examining the isolated effect of increases in PV is to induce an expansion through artificial means via high-molecular-weight compounds such as dextran (Macrodex) infused in physiological saline (21, 22) to produce an isosmotic hypervolemia. With this technique, expansion in excess of 20% can be realized without an impairment in VO₂max (21). In this study, we have used the dextran procedure to artificially expand PV to investigate the hypothesis.
that the plasma levels of Aldo, AVP, and ANP will all be altered as a consequence of the higher PV levels during exercise.

METHODS

Participants. Eight untrained but active and healthy men volunteered for the study. Their age, weight, peak aerobic power (VO$_{2\max}$), and maximal heart rate were 21 ± 1 (SE) yr, 76.9 ± 2.0 kg, 3.49 ± 0.19 l/min, and 205 ± 3 beats/min, respectively. As required, the procedures and risks were fully explained to each participant before written consent was obtained and after approval of the study by the Office of Human Research (University of Waterloo, Waterloo, ON).

General research design. Measurements were made during a prolonged standardized cycle exercise test at an intensity equivalent to ~46% peak aerobic power (VO$_{2\max}$) on three occasions: under control conditions with no PV expansion (Con), after infusions of 277 ± 11 ml of Macrodex solution (Low), and after infusion of 554 ± 21 ml of Macrodex solution (High). The exercise tests were conducted at least 1 wk apart in ambient temperatures (22–24°C) and humidities (35–45%) and in randomized order. All studies were conducted at approximately the same time of day for each subject and between 4 and 6 h after consumption of a liquid supplement, consisting of one can of Ensure (1,045 kJ: 14.8% protein, 31.5% fat, and 53.7% carbohydrate; Ross Laboratories, Montreal, PQ). Only water (ad libitum) was allowed between ingestion of the liquid supplement before the exercise tests. All participants were instructed not to engage in any vigorous activity during the experiment and to follow a normal balanced diet. Only low- to moderate-intensity exercise was employed because the volunteers were untrained and probably incapable of sustaining exercise at higher intensities for the prolonged period required.

At least 120 min before the exercise test, the participants reported to the laboratory for preparation and for acute infusion of a 6% dextran solution dissolved in saline (Macrodex, Pharmacia Laboratories). The Macrodex solution, equilibrated to room temperature, was infused into an antecubital vein via a catheter (Angiocath 21) over a 60-min period. The volume of solution was calculated on the basis of estimated PV (1 kg ± 44 ml), with an 8% increment representing the Low and a 16% increment representing the High condition. The calculated expansion of plasma volume, estimated for hematocrit (Hct) determinations obtained while subjects were seated at the position amounting to 14.4 ± 1.8 and 21.2 ± 2.1% for the Low and High conditions, respectively. The difference in the percent PV expansion between the two conditions was significant (P < 0.05). For all conditions, including Con, in which no Macrodex was infused, the procedures were identical, with the bottle of infusate hidden from the participant. It has previously been established that the expansion of PV is lost within the first several days (30a). In this study, no differences were found among the resting preinfusion Hct values for each condition, suggesting that PV was comparable among conditions.

After the completion of the infusion, the participants were positioned upright on a cycle ergometer for ~30 min, during which time a number of preparatory measurements were taken. At the end of the 30-min period, blood was withdrawn for measurement of the plasma hormones and serum osmolality. Blood was also collected at selected intervals throughout the 2-h period of continuous exercise. No fluid was permitted either during the exercise itself or during the preparatory period after arrival at the laboratory.

O$_2$ uptake (VO$_2$) measurements. Exercise was performed on an electric cycle ergometer (Quinton 870, Excalibur Sport, Netherlands) calibrated before each test. For the VO$_{2\max}$ test, the protocol was identical to that previously described (18) and consisted of progressive increases in power output (15 W) each minute until fatigue. Ventilation and gas exchange were determined with the use of an open-circuit system (20). The value used for VO$_{2\max}$ was the VO$_{2\max}$ obtained, integrated over a 30-s period, during the progressive tests. For the prolonged cycle test, an absolute power output was selected that represented ~46% VO$_{2\max}$. Measurements of VO$_2$ were made at selected intervals (15, 30, 60, 90, and 120 min) throughout each test. For all conditions, the prolonged exercise test was conducted continuously and at the same absolute power output. As expected, no differences in VO$_2$ were observed among the conditions (unpublished observations).

Blood measurements. Radioimmunoassay methods and, specifically, $^{125}$I labeling were used to measure the plasma levels of AVP (Peninsula Laboratories), Aldo (Coat-A-Count, Diagnostic Products), and $\alpha$-ANP (Peninsula Laboratories). For ANP and AVP, 3 ml of whole blood were transferred into chilled polypropylene tubes containing EDTA and aprotinin (ANP) and heparin (AVP), the tubes were centrifuged at 1,600 g at 4°C for 15 min, and the plasma was stored at ~80°C until analyses. The ANP and AVP were extracted from the plasma in a buffer containing trifluoroacetic acid and acetoni-trile, centrifuged and eluted by using a C18 Sep-Pak column (R1K-SEPCOLI, Peninsular Laboratories), and the eluent was dried by evaporation by using a lyophilizer. The radioimmuno-assays were performed by dissolving the residue in radioimmonoassay buffer and performing centrifugation, and single aliquots of each sample were incubated for 24 h at 4°C with antiserum followed by the addition of $^{125}$I-labeled-peptide (125I-ANP or 125I-AVP) and a second 24-h incubation. A third incubation for 90 min was performed after the addition of a second antibody and normal rabbit serum to each tube. Free and bound ANP and AVP were separated by centrifugation. The extraction of known amounts of ANP and AVP has been estimated at 66%. These procedures are essentially as outlined in the radioimmunoassay kit (Peninsula Laboratories). Our estimates of the interassay coefficient of variation were 2.5% for ANP and 4.8% for AVP.

For Aldo, the assay was performed on unextracted serum, stored at ~80°C after collection after separation from whole blood. On the basis of the Coat-A-Count procedures (Diagnostic Products), the detection level for Aldo is ~16 pg/ml and maximal binding is 40%. The antiserum is highly specific for Aldo, with little cross-reactivity to other compounds. The coefficient of variability for duplicate measurements was 5.5%.

Plasma catecholamines, both norepinephrine (NE) and epinephrine (Epi), were based on high-performance liquid chromatography (HPLC) separation techniques by using electrochemical detection (Waters 712 WISP) according to Weicher et al. (36) and as modified (16). For this assay, 3 ml of whole blood were collected in a tube containing ethylene glycol-bis-(β-aminoethyl ether)-N,N,N’,N’-tetraacetic acid and glutathione as antioxidant. The blood was then centrifuged at 2,000 g and 2°C and the plasma was removed and stored at ~80°C until analysis. All blood samples for a given assay and a given individual were analyzed in duplicate during the same analytical session. The percent change in PV both among conditions at rest and within a condition during exercise was calculated from Hct changes (measured in triplicate) by using the equation of van Beaumont et al. (34). All Hct values were corrected for trapped plasma (0.96) and venous-to-whole body Hct difference (0.91). Serum osmolality
was determined in duplicate by using a vapor pressure osmometer (Wescor model 5100C).

Statistical procedures. The effects of exercise time and experimental condition (Con, Low, High) were examined by using two-way analysis of variance procedures for repeated measures. When significance was found, post hoc analysis was used to determine the differences between specific means, according to the Newman-Keuls technique. A 95% level of confidence was established for all comparisons.

RESULTS

Plasma volume. The infusion of 277 ± 11 ml (Low) and 554 ± 21 ml (High) of 6% Macrodex resulted in a calculated expansion of PV obtained after subjects were sitting on the cycle ergometer for ~30 min of 14.4 ± 1.8 and 21.2 ± 2.1%, respectively. These elevations in PV on the basis of the supine position were different from both the Con condition and from each other. The effect of exercise on PV alterations within a condition is provided in Fig. 1A. Exercise, regardless of condition, resulted in a reduction in PV. The percent magnitude of the reduction that was observed was independent of the preexercise PV. Compared with the Con condition, PV remained higher throughout rest and exercise with hypervolemia (Fig. 1B; Con < Low < High). Plasma osmolality also changed with exercise (Table 1), but, as with PV, no differences were observed among conditions. Interestingly, the loss in PV was fully manifested during the initial 15 min of exercise.

Blood hormones. Plasma Aldo was altered by both exercise and hypervolemic state (Fig. 2). In the Con condition, the elevation of Aldo was most marked during exercise, with progressive increases noted at 30, 60, and 90 min. For the Low condition, initial increases were also observed at 30 min, but for the High condition initial increases were not observed until 60 min of exercise. With the exception of 30 min of exercise, the concentration of Aldo was dependent on the hypervolemic state (Con < Low < High). At 30 min, only the High condition was lower than the Con condition (Con = Low < High).

Plasma AVP was also altered by exercise and hypervolemic state (Fig 3). With exercise, a generally small but significant increase occurred that was only evident by 120 min. The concentration of AVP was lower in both the Low and High conditions compared with Con. This was a general effect and not restricted to specific time points. No differences in plasma AVP were found between the Low and High conditions.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Time, min</th>
</tr>
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<tbody>
<tr>
<td>Control</td>
<td>282±1.7</td>
</tr>
<tr>
<td>Low</td>
<td>284±2.4</td>
</tr>
<tr>
<td>High</td>
<td>286±3.1</td>
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Values are means ± SE; n = 8 subjects. Control, no plasma volume expansion; Low, after 14% plasma volume expansion; High, after 21% plasma volume expansion. A significant main effect was found for times of 15, 30 < 90, and 120 min; 15 < 60 min, P < 0.05.

Table 1. Effects of acute plasma volume expansion and exercise on serum osmolality

Fig. 1. Changes in calculated plasma volume (PV) within each condition (A) and relative to control condition (B). Con, control; Low, low PV expansion; High, high PV expansion. Con < Low < High, P < 0.05.

Fig. 2. Effect of acute PV expansion on exercise-induced changes in plasma aldosterone. Values are means ± SE; n = 8 subjects. Abbreviations are defined as in Fig. 1. *Significantly different from Low, P < 0.05; †significantly different from Con, P < 0.05; ‡significantly different from 0, P < 0.05; ‡‡significantly different from 30 min, P < 0.05; ‡‡‡significantly different from 60 min, P < 0.05.
As with plasma Aldo and AVP, both exercise and PV expansion altered plasma \( \alpha \)-ANP concentration (Fig. 4). However, the effect was different from that observed for both Aldo and AVP. In the case of exercise, a general increase was observed at both 30 and 60 min compared with 0 min. Thereafter, the concentration decreased such that by 120 min of exercise, no differences were found from rest. Both Low and High conditions elevated the \( \alpha \)-ANP concentration over Con. This effect was observed at rest and persisted throughout exercise.

Induced hypervolemia blunted the exercise-induced increase observed in NE observed in the Con condition (Fig. 5). For both the Low and High conditions, the concentrations of NE were generally lower at all exercise time points. The only exception was at 15 and 90 min, where no differences were found between the Con and Low conditions. With the exception of 90 min, differences between Low and High conditions were not significant. In general, the effect of hypervolemia on plasma Epi followed a similar pattern to NE, namely, a blunting of the exercise-induced increase. However, in contrast to NE, the effect of the PV expansion was more delayed. Initially, differences were not seen until 60 min of exercise. As was generally found for NE, attenuation in the Epi response did not depend on the magnitude of the PV expansion.

**DISCUSSION**

The results of this study clearly indicate that at least for the three fluid and electrolyte regulatory hormones, Aldo, AVP, and \( \alpha \)-ANP, isosmotic expansion of PV significantly affects plasma concentration during prolonged low- to moderate-intensity exercise. Plasma volume expansion, however, appears to affect each hormone differently. In the case of Aldo, the effect is manifest during exercise itself, and the alteration that
occurs is directly related to the magnitude of the hypervolemia. In contrast, AVP is suppressed during exercise in a manner that is not volume dependent and that appears to occur at rest before exercise and to persist throughout the exercise. Alterations in resting concentration also appear to account for the difference in \( \alpha \)-ANP observed between the Con and hypervolemic conditions. However, in the case of \( \alpha \)-ANP, the effect of the hypervolemia, which also does not appear to be graded to magnitude, is to increase concentration. Although all of the fluid and regulatory hormones examined are stimulated by a complex of factors, PV level and osmolality appear to be of primary importance (4, 5, 7). Other factors such as systemic blood pressure, cardiac output and stroke volume, and thermal load may either directly or indirectly also modify the hormonal response (7, 12, 25, 35). Although rectal temperature was not altered with PV expansion in this study, both mean arterial pressure (MAP) and cardiac function were altered (unpublished observations). Acute PV expansion resulted in a lower MAP and a higher cardiac output and stroke volume.

In this study, only PV varied among conditions in a manner that related directly to the amount of fluid infused. With subjects at rest on the cycle, compared with Con, the expansion in PV was estimated at 14 and 21%, respectively. With exercise, PV was reduced as expected (19). Because the percent loss was similar among conditions, the absolute reduction in PV was related to the initial level. However, even with the greater losses, the absolute volume remaining during exercise stayed proportional to the preexercise hypervolemic level. The greater loss of PV with acute expansion would appear to be determined principally by an elevated vascular hydrostatic pressure (37). Plasma osmolality did not differ among conditions, a finding that was expected and intended because the dextan was infused in a 6% solution of isotonic saline.

Our findings of an increase in AVP with prolonged exercise is entirely consistent with a number of previous studies (4, 11, 35). The difference in the magnitude of the response in untrained participants would appear to be due to the intensity of exercise and the environmental conditions. Previous studies have demonstrated severalfold increases when the exercise is performed at a higher intensity (4) and in hot temperatures (1, 2). Because both plasma osmolality and PV showed modest but significant alterations with exercise, conceivably both could be implicated in AVP release (4). The blunting of the AVP response with acute PV expansion appeared to be due to the lower resting concentration that persisted throughout the exercise. To some degree, the lower concentration could have been mediated by the diluting effects of the elevated PV. To our knowledge, no previous study has examined the changes in AVP during prolonged exercise after acute hypervolemia induced before exercise. However, other models have been used to alter exercise PV. One model that has been employed is to vary the hydration state before exercise by using diuretics (hypohydration) or isotonic water ingestion (hyperhydration). These experiments have demonstrated that when low-intensity exercise is performed in the heat with no fluid replacement, a pronounced blunting of AVP is evident both before the exercise and during the exercise itself (2). In another model, with use of the same exercise conditions, this group was able to demonstrate a complete inhibition of the AVP response when rehydration was permitted by oral ingestion of either water or an isotonic solution (1). Collectively, these studies have been interpreted to demonstrate the significance of both PV and osmolality in controlling AVP secretion.

Training also appears to depress the AVP response to exercise but in a manner that is dependent on the characteristics of the exercise. Convertino et al. (5) as well as others (11, 26) have been able to demonstrate that depressions in AVP after short-term training are dependent on exercise intensity, but not on the duration of moderate exercise (4). The latter finding is surprising because PV was significantly elevated with the training, and higher levels of PV appeared to be retained during the exercise itself (4). Osmolality, however, was unchanged with the training (4). The fact that these authors did not find decreases in AVP with training could also be due to the difference in the exercise protocol before and after training. The prolonged exercise test after training was apparently at a higher absolute intensity than before training (4). Unlike our study, these authors did not find depressions in preexercise AVP (4, 5). This could, however, be due to the magnitude of the training-induced PV response, which was only 12%. Other studies using acute volume expansion of comparable levels to our studies have also reported a depression in AVP at rest (13). It is possible that the lower MAP observed with acute PV expansion may have attenuated the AVP response, minimizing the actual reduction that resulted (11, 35).

The effect of the induced expansion in PV on the fluid and electrolyte hormones was most clearly evident for Aldo where the suppressive effect was restricted to the exercise itself and proportional to the degree of expansion. This finding is consistent with a previous study (10) in which albumin was used to expand PV and exercise was conducted in the heat. Because osmolality was unchanged among conditions, the suppressive effect would have to be due directly to the PV level or secondary to some change initiated by the elevated PV. Normally, secondary effects are most often implicated. Several remain possible to explain the suppressive effect observed. A probable mechanism for the depression in Aldo observed in this study would appear to involve a reduction in renin production by the juxtaglomerular cells in the kidney as a consequence of improved renal perfusion (7, 25). According to this hypothesis, increases in PV could minimize the reduction in kidney blood flow typically observed during prolonged exercise (32). Reductions in renin concentration would be expected to reduce the amount of angiotensin I and consequently the amounts of angiotensin II and Aldo (25). This mechanism may also be potentiated as a consequence of the apparent reduction in the sympathetic drive that occurred, as indicated by the reduced
blood concentrations of both Epi and NE (7). Reductions in renal sympathetic tone have been shown to result in a lower β-adrenoceptor activation of the juxtaglomerular cells and lower renin secretion (7). Reductions in sympathetic activity could also reduce vasoconstriction of the renal arterioles and alter kidney perfusion. Although these possibilities would appear to represent the most probable mechanisms and appear to be supported by previous exercise and training studies (4, 5, 7), other possibilities may also exist. The production of Aldo under certain conditions can be uncoupled from the classic renin-angiotensin hierarchy (1, 2), indicating that other stimuli may intervene. Most exercise and training studies, however, indicate a parallel increase in renin, angiotensin II, and Aldo (7, 26). What can be ruled out is the possible role of osmolality and, in particular, potassium, in accounting for the effects of acute volume expansion on Aldo (25). Because MAP was depressed and carbon monoxide was elevated with acute PV expansion, these changes could also impact on the Aldo response either via alterations in kidney blood or via reductions in the sympathetic vasoconstriction of the renal arterioles and alter kidney perfusion. Although most training studies are consistent in demonstrating a decrease in Aldo during exercise at least at the same absolute intensity (5, 23), one study is curiously in contradiction (4). This is surprising because a previous study has shown that reductions in Aldo occur after training in the absence of changes in VO_{2max} and in the absence of changes in VO_{2} during the exercise itself (K. Shoemaker, H. Green, M. Ball-Burnett, and S. Grant, unpublished observations). Furthermore, the results of this study suggest that increases in PV should result in a lower Aldo response to exercise. The fact that no reductions in plasma renin were observed during prolonged exercise after short-term training is even more curious because elevations in PV were observed (4). It is possible, however, that the degree of PV expansion, ~12%, may not have been sufficient to alter the Aldo response. Moreover, because only renin was measured, it is conceivable that changes in Aldo may have been dissociated from the renin response (1). The most plausible explanation, however, was the difference in exercise intensity between the tests administered before and after training. As previously indicated, the absolute exercise intensity was apparently higher posttraining (4).

Our study has also found changes in plasma ANP with both exercise and acute PV expansion. At least with regard to exercise, most studies report increases in ANP, although the magnitude and pattern of change may be dependent on the characteristics of the exercise stimulus (12, 24, 30). In this study, an increase in ANP was observed followed by a regression toward rest values as exercise was prolonged. This is consistent with what has been reported previously (22) and may be directly associated with changes in atrial distension, the site of ANP production (6, 27). At the onset of exercise, central venous pressure (CVP) and, supposedly, atrial distension increase (32). However, as exercise progresses, CVP and, expectedly, atrial distension decrease (32). The higher ANP concentrations observed during exercise after PV expansion were clearly a carryover from the higher resting levels. Higher resting levels have been reported previously in persons with acute PV expansion (6, 9, 27). The increases in ANP previously noted during exercise after training probably reflect the elevation in PV that results from the training (6, 11, 25). Elevations in PV, whether training-induced or artificially induced, result in an expansion in CVP (3) and, in all probability, atrial distension. The data on cardiac function obtained in this study clearly support this possibility because cardiac output and stroke volume were elevated both at rest and during exercise (unpublished observations).

In summary, we have shown that elevations in PV appear to account for the training-induced reductions in Aldo observed during exercise and for the resting alterations in AVP and ANP observed at rest and that persist during prolonged exercise. It must be emphasized, however, that the PV increases represented the upper levels of those reported during training and heat acclimatization (15, 18). Moreover, in our experiments, the expansion was acutely induced over an ~60- to 90-min time frame. In contrast, training-induced PV expansion occurs over the first few days (4, 5, 15, 33). The implications of the different time frames remain to be elucidated as does the effect of the changes in cardiovascular function and blood flow that could affect hormonal concentrations by altering clearance rates. Remaining to be established as well is the role of numerous other adaptations that might result from training in general and from short-term training in particular.

The authors acknowledge the contribution of Pharmacia Laboratorios (Canada) for the donation of Macrodex. The authors appreciate the contribution of Dr. John Sutton, who died suddenly during the preparation of the manuscript.

This work was supported by the Natural Sciences and Engineering Research Council of Canada.

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Received 31 January 1996; accepted in final form 30 July 1996.

REFERENCES


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