The forgotten Barcroft/Edholm reflex: potential role in exercise associated collapse

Approximately 80% of the athletes who collapse in marathon and other long distance sporting events do so only after they have crossed the finish line. Although it is usually taught that “dehydration” explains this phenomenon, logic suggests this to be unlikely because any dehydration induced hypovolemia should cause cardiovascular failure when cardiac stress is highest—that is, during rather than after exercise when cardiovascular function is returning to the resting state. Instead it is clearly the act of stopping exercise that is the consequent, albeit paradoxical, cause of post-exercise collapse.

We have previously proposed that this form of exercise associated collapse is caused by the persistence into recovery of a state of low peripheral vascular resistance, compounded by removal of the skeletal muscle pump that maintains the right atrial filling pressure during exercise. According to this theory, the combination of a low peripheral vascular resistance and a sudden reduction in venous return would reduce stroke volume and cardiac output acutely, causing hypotension. Indeed this mechanism was used to explain the development of postural hypotension due to “dehydration exhaustion” in military personnel exposed to eight or more hours of exercise in desert heat without fluid replacement, in the classic studies of Adolph.

Thus Adolph wrote that “the ultimate failure of the circulation (in dehydration exhaustion) is of a peripheral type... Dilated blood vessels of the skin require additional blood to fill them; correspondingly, the available compensation for vertical posture are diminished, for the blood vessels of the legs compromise between dilating to carry heated blood and constricting to preserve a greater flow to the head” (p 235). He also reported that: “Lying down promptly relieves the circulations and the symptoms” (p 236). Similarly, Lee defined the physiological abnormality causing heat exhaustion as: “A low arterial blood pressure is the critical event, resulting partly from inadequate output by the heart and partly from the widespread vasodilatation”. He also noted: “Treatment is simple – recumbency, fluid and salt administration, gentle cooling, and rectification of any predisposing cause” (p 571).

However, these explanations fail to explain why a compensatory tachycardia is not a usual feature of this form of hypotension, or why the onset of symptoms occurs so rapidly and why they are reversed so rapidly when subjects lie supine in the head down position.

An historic study identifying a potentially forgotten reflex and published in The Lancet in 1944 suggests an alternative explanation. Barcroft et al1 studied the effects of rapid venesection of about 1 litre of blood in 12 minutes. Note that fainting is caused by a sudden reduction in peripheral vascular resistance resulting from an increase in forearm blood flow as right atrial pressure falls, the Barcroft/Edholm reflex. Redrawn from data in Barcroft et al.

A second study evaluated the authors’ hypothesis that it was the fall in right atrial pressure induced by blood loss that activated the reflex reduction in peripheral vascular resistance. A tourniquet was applied to the lower limb and inflated before the beginning of the venesection. Hypotension again developed but after a smaller blood loss of only about 550 ml. Release of the tourniquet immediately corrected the hypotension by reducing forearm blood flow (fig 2). The authors concluded that removal of the tourniquet produced a sudden infusion of blood from the lower limb thereby increasing the right atrial pressure, abolishing the reflex and normalising cardiovascular function.

Hence they proposed the existence of a potent skeletal muscle vasodilator reflex that is activated when the right atrial pressure either falls below some critical value or begins to fall at a particular rate. This reflex appears atavistic because it compounds rather than corrects the hypotension associated with blood loss. The similarity in the rapidity with which hypotension occurs with either progressive venesection or after the cessation of exercise, and without an associated tachycardia, suggests that this right atrial reflex may also cause exercise associated collapse.

To my knowledge, some contemporary texts of cardiovascular control during exercise do not specifically mention this reflex. For example, Rowell refers to a later 1945 paper in the Journal of Physiology by Barcroft and Edholm, in which the physiological basis for this skeletal muscle vasodilation was studied. However, the relevance of this specific reflex to the development of syncope is not directly discussed. Rather, Rowell presents the findings of...
Barcroft and Edholm as a possible example of neurogenic sympathetic cholinergic vasodilation in skeletal muscle.

It is of interest that we had empirically discovered some years ago that the optimum management of post-exercise collapse is achieved by elevating the feet and pelvis of collapsed athletes above the level of the right atrium. The results of this manoeuvre are usually dramatic, with rapid reversal of hypotension and the symptoms of dizziness. According to the Barcroft/Edholm reflex, the adoption of this head down position would produce this dramatic response by immediately reversing the low atrial pressure that develops on the cessation of exercise in some susceptible athletes. Hence the effect would not result simply by increasing venous return and hence cardiac output, the more usual explanation, but also by reversing the skeletal muscle vasodilation induced by the Barcroft/Edholm reflex.

Finally, I would like to make the obvious point that the Barcroft/Edholm reflex explains why nursing in the head down (Trendellenberg) position would be the more logical approach to management of post-exercise collapse than the provision of intravenous fluids. The reason is that nursing in the Trendellenberg position rapidly increases the right atrial pressure whereas any effect of intravenous fluids on right atrial pressure is likely to be smaller and much delayed.

Furthermore the contribution of the Barcroft/Edholm reflex to exercise associated collapse can be very rapidly evaluated by studying the response of the athlete’s blood pressure to changes in posture. Failure of the blood pressure to rise when placed in the head down position must indicate that the Barcroft/Edholm reflex is not active and some other cause for the hypotension, such as myocardial dysfunction or a persistent reduction in peripheral vascular resistance for reasons other than this postulated reflex, must be considered.

**Figure 2** The sudden reduction in systolic blood pressure and the associated increase in forearm blood flow with progressive venesection is immediately reversed by release of a lower limb tourniquet. Redrawn from data in Barcroft et al.

| References |
|------------|-----------------|

| Table 1 details and skin fold thicknesses (mm) of the subjects |
|------------------|------------------|
| **Age (years)** | **Mean (SD)**    | **Range** |
| 23.62 (3.57)    | 18–31            |
| **Weight (kg)**  | **Mean (SD)**    | **Range** |
| 76.12 (8.34)    | 64.3–105.4       |
| **Height (m)**  | **Mean (SD)**    | **Range** |
| 1.81 (0.05)     | 1.7–1.92         |
| **Body mass index (kg/m²)** | **Mean (SD)**    | **Range** |
| 23.20 (1.79)    | 20.4–28.6        |
| **Biceps**      | **Mean (SD)**    | **Range** |
| 4.21 (0.93)     | 3.2–6.5          |
| **Triceps**     | **Mean (SD)**    | **Range** |
| 7.44 (2.46)     | 4–12.7           |
| **Abdomen**     | **Mean (SD)**    | **Range** |
| 10.67 (4.36)    | 5.3–21.6         |
| **Subscapula**  | **Mean (SD)**    | **Range** |
| 9.43 (2.05)     | 3.3–13.1         |
| **Suprailiac 1**| **Mean (SD)**    | **Range** |
| 7.13 (2.70)     | 3.4–14.8         |
| **Suprailiac 2**| **Mean (SD)**    | **Range** |
| 10.13 (3.68)    | 5.3–20.7         |
| **Thigh**       | **Mean (SD)**    | **Range** |
| 9.05 (3.24)     | 5–15.8           |
| **Calf**        | **Mean (SD)**    | **Range** |
| 6.96 (2.20)     | 3.7–12.9         |
| **Chest**       | **Mean (SD)**    | **Range** |
| 5.43 (2.14)     | 3.3–13.1         |
biceps. Table 2 shows correlations between the predictive fat measurements. There were strong correlations between the Tanita measurements and the other estimates. In addition, all the measurements, excluding biceps, triceps, chest, and suprailiac 1, also tended to increase in direct proportion with weight (p<0.001 for all values). Body density was found to correlate negatively with all the other measurements (p<0.05 for all values). A similar study to ours was conducted by Utter et al. in collegiate wrestlers in which three SFT measurements were compared with those obtained with a Tanita analyser (TBF-305). They also showed significant correlations. Table 2 shows a substantial negative correlation with the other measurements and the other estimates. In addition, all the measurements, excluding biceps, showed significant correlations between the Tanita measurements and other estimates. In addition, all the measurements, excluding biceps.

### Table 2 Correlation coefficients between the measurements

<table>
<thead>
<tr>
<th></th>
<th>Body density</th>
<th>Tanita</th>
<th>Zorba</th>
<th>Siri</th>
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<tbody>
<tr>
<td>Body density</td>
<td>-</td>
<td>-0.76</td>
<td>-0.90</td>
<td>-0.91</td>
</tr>
<tr>
<td>Tanita</td>
<td>-0.76</td>
<td>0.75</td>
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<tr>
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All p values <0.001.

We commend Professor Murrell for making a major novel contribution to the understanding, and thus potentially the treatment, of chronic tendinopathies. He has shown in patients with supraspinatus tendinopathy that a substantial proportion of the cellular compartment of the tendon shows nuclear fragmentation, a key characteristic of apoptosis.

Professor Murrell feels that apoptosis would necessarily be a "harmful" pathological event in tendon or might there be an optimal, basal level of apoptosis? We appreciate that excessive apoptosis—programmed cell death—is a feature of degenerative pathologies such as muscular dystrophy and Alzheimer's disease. In these conditions apoptosis may contribute to deterioration of the diseased tissue. On the other hand, it is a characteristic feature in physiological situations such as scar remodelling, in which coordinated death of myofibroblasts is a desirable outcome. A recent study does suggest that proliferation of tenocytes can enhance tendon remodelling, as proliferation of tenocytes after IGF-1 injection was accompanied by improved tendon stiffness and reduced lesion in equine flexor tendinopathy. Other studies have shown fibroblast apoptosis from excessive strain or, paradoxically, decreased loading.

We would also appreciate Professor Murrell's opinion on the significance of a third of tendon cells displaying a positive TUNEL assay, months after the onset of symptoms. Could apoptotic cells remain in tendon tissue for months after injury, or might the condition cause continual apoptosis and proliferation concurrently? If the latter were true, would apoptosis be a causal event of tendon degeneration or a secondary effect of scar remodelling? The fact that paratenonitis can directly cause tendinosis and loss of tenocytes suggests a possible causal role of apoptosis.

Does Professor Murrell see growth factors as playing a role in modulating apoptosis in the tendon, and could the effect of growth factors on tenocyte death and survival be tested in vitro or in model systems? Such investigations would be complicated by the many interacting conditions in the tendon which could promote or prevent apoptosis, including integrin-mediated signals, soluble factors, and cellular stresses such as hypoxia and reactive oxygen species. The distinct phenotypes of tenocytes within and among tendons would add yet another layer of complexity to the story.

Again, we commend Professor Murrell for this contribution. We will be very curious to know which caspases are activated in the supraspinatus and in other tendinopathies, and whether there would be an inhibitory effect of caspase inhibitors. By showing for the first time that nuclear fragmentation is a significant feature of rotator cuff tendinopathy, Professor Murrell has brought us one step closer to a more complete understanding of this condition.

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### References


### Lumbopelvic mechanics

It has annoyed us for a long time when patients present stating that they have been "core conditioning/core strengthening/pelvic stabilisation", etc. It is also annoying to find the same terms used in peer reviewed scientific articles with the assumption that they mean something to the readers. Maybe they do to others, and we are missing something! We would like to get some discussion going on this and are happy to open the batting.

We think of lumbopelvic mechanics as three distinct groups:

1. Intrapelvic stability
2. Peripelvic stability
3. Functional stability.

### Intrapelvic stability

This is dependent on the transversus abdominis contracting with intact posterior sacroiliac joint ligaments. The anatomy of the transversus abdominis is such that it has a major origin off the iliac crest and inguinal ligament/conjoint tendon to insert into the linea alba. Therefore it and the pelvic floor are the only muscles that give direct closure across the sacroiliac joint. The long lever arm involved gives it great mechanical advantage, as long as the sacroiliac joint ligaments are intact.
Problems arise from two mechanisms.

1. Loss of the active structures that provide intrinsic pelvic closure (the transversus abdominis and the pelvic floor muscles) because of
   - inhibition of the transversus abdominis caused by first onset low back pain;
   - pain inhibition through joint inflammation: “osteitis pubis”, hip joint pathology, and sacroiliac/lumbar spine joint dysfunction;
   - a tear of the conjoint tendon/inguinal ligament disrupting the origin of the inguinal ligament; and
   - past abdominal surgery inhibiting contraction or affecting the nerve supply to the transversus;
   - tearing of the pelvic floor muscles during child birth;
   - weakness of the pelvic floor muscles secondary to poor toilet habits.

2. Loss of passive structures
   - pelvic ligamentous laxity due to either body type (hypermobility) or external trauma (either single incident or prolonged postural loading), or hormone related ligamentous laxity (pregnancy);
   - laxity of the sacroiliac joint ligaments will cause loss of the closure moment at the sacroiliac joint. A very small loss of ligament strength here can have a profound effect on the closure moment. Is this how Vleeming’s posterior sling exercises work on post partum women? Further is this the mode of action of recto-therapy of the sacroiliac joint ligaments?

Peripelvic stability

Once the pelvis is stable and we have a firm foundation, we can look at the pelvis reacting with the rest of the body.

Pelvis on hip joint (pelvimensoral control)
- Does the knee roll inwards as the subject single leg squats?
- Does the pelvis dip as the patient reaches single leg stance?
- Is the lack of pelvifemoral control a strength or timing problem around the hip joint (gluteus medius and glutaeus maximus) or is it an inability to appropriately weight transfer on to that side because of hip, knee, or ankle problems, burnt out nerve root pathology, or just disuse of one side because of chronic injury.

Pelvis on lumbar (and thoracic) spine (lumbopelvic control)
- This is concerned with ability of the deep multifidus to contract to control the lumbar segments and the superficial multifidus to orientate the spine on the pelvis.1

Functional stability

Once the muscle strength and activation patterns are in place to allow force transfer through the pelvis, then linking these activities into normal activities and actions and conditioning the lumbopelvic complex can take place. This is really just an “on-field” extension of peripelvic stability. Once the athlete has all the necessary components to hold the pelvis stable on the femur and lumbar spine, can they coordinate that into their particular sporting or every day activity?

This type of stability is more concerned with technique, coaching, and video analysis. Also, as conditioning will no doubt affect the fatigue status of the athlete, high level physical conditioning allows the athlete to maintain a stable pelvis without physical fatigue, hence appropriate strength/endurance and power training is applicable to the type of athletic activity and the stage of the athletic season.

Most gym based strength, conditioning coaching, and fitness programmes fall into this category. However, it is our belief that, if the intrapelvic and peripelvic problems are not addressed first, that is where these patients break down. Conversely, if inadequate conditioning is performed before return to sport/competition, then the athlete will break down, as there is no transmission of their rehabilitation on to the field of play.

People present at various points along the continuum of disease. Some lack functional stability, and some lack peripelvic strength and coordination, but have a stable pelvis. Others have a pelvis that swings in the breeze. A recreational athlete with a sedentary occupation does not need the same level of intrapelvic strength and endurance as an Australian Football League on-baller or elite soccer player. Yet someone doing a lot of vacuuming and weight bearing with poor ergonomic and alignment values needs quite good intrapelvic and peripelvic strength without the need for the endurance of an elite athlete in these muscles.

We look forward to hearing the thoughts of others on this topic.

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Muscle dysmorphia in weightlifters

I would like to raise a concern with regard to the article “Muscle dysmorphia: a new syndrome in weightlifters” by Choi et al. Although chronic obsessive behaviour related to fitness and weightlifting is genuinely a concern for both sexes, Choi et al fail, in my opinion, to present a valid argument for the construction of “muscle dysmorphia” as being a separate and distinct subcategory of body dysmorphic disorder. Inclusion criteria and procedures in the study by Choi et al were identical with those used in the study by Olivarzia et al—they used identical study populations. Both Choi et al and Olivarzia et al claim that differences exist between their cases (weightlifters preoccupied with their body image, namely insufficient muscularity) and controls (weightlifters not preoccupied with their body image) with respect to body image and a variety of other characteristics. However, in both studies, the aim of which was to distinguish muscle dysmorphia as a distinct clinical entity, individuals were classified as cases or controls on the basis of their body image perception. It is therefore hardly a surprise to find that men who obsess about their body image and who perceive themselves to be insufficiently muscular differ with respect to their body image and their perception of being insufficiently muscular from men who do not have the perception of being insufficiently muscular. Olivarzia et al discuss muscle dysmorphia in athletes with limitation, stating that, although this tautology exists, there are other factors that separate the two groups (higher lifetime prevalence of mood and anxiety disorders, use of steroids and other drugs, and higher scores on many Eating disorders inventory subscales). However, this evidence alone is not sufficient to indicate that these characteristics distinguish a muscle dysmorphia construct, as it is possible that, within this target population (male weightlifters), the proposed muscle dysmorphia characteristics in fact distinguish the other observed disorders. I would advise readers to take the conclusion of Choi et al that “...muscle dysmorphia may be one negative consequence of physical exercise behaviour ...” with extreme caution, as its validity as a distinct clinical entity has not yet been proven.

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References

A study day with Professor Stuart McGill. Further details: Yvonne Gilbert, BASEMS Secretary, Royal College of Surgeons of Edinburgh, Nicolson Street, Edinburgh EH8 9DW; email: y.gilbert@rcsed.ac.uk; tel: +44 (0)131 527 3409. Organised by BASEMS Scotland.


Keynote speakers include Professor Per Renstrom (SWE), Professor Peter Jokl (USA), Professor Savio Woo (USA), Dr Carol Otis (USA), Dr Mark Safran (USA), Dr Ben Kibler (USA), Prof Bruce Elliott (AUS), and Professor Ron Maughan (UK).

Further details: Dr Michael Turner, The Lawn Tennis Association, The Queen’s Club, London W14 9EG, United Kingdom; email: michael.turner@lta.org.uk

International XVII Puijo Symposium 25–28 June 2003, Kuopio, Finland

“A physical activity and health—gender differences across the lifespan.”

Further details: Ms Auli Korhonen, Project Secretary, Kuopio Research Institute of Exercise Medicine, Puijo Symposium Secretariat, Haaaniemntie 16, 70100 Kuopio, Finland; tel: +358 17 288 4422; fax: +358 17 288 4488; email: puijo.symposium@uku.fi

12th International Biochemistry of Exercise Conference 13–16 July, Maastricht, the Netherlands

Further details: Marllen van Baak; email: m.vanbaak@hb.unimaas.nl; website: www.biochemex.org/IBEC

The Fifth International Conference on Sport, Leisure and Ergonomics 19–21 November 2003, Burton, Cheshire, UK

A three day conference in affiliation with the Ergonomics Society.

Further details: Congress Secretariat, Sport, Leisure and Ergonomics, Research Institute for Sport and Exercise Sciences, Liverpool John Moores University, Henry Cotton Campus, 15-21 Webster Street, Liverpool L3 2ET, UK; tel: +44 (0)151 231 4088; email: K.George@livjm.ac.uk

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- Master of Physiotherapy by Coursework (Sports Physiotherapy) NOW CLOSED.
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