The Collapsed Endurance Athlete: A practical approach

EXERCISE ASSOCIATED COLLAPSE.

There is confusion in the literature over the diagnosis of exercise associated collapse, that revolves essentially around the following two concepts:

1. Any condition that causes collapse during exercise.
2. Collapse that cannot be attributed to a readily identifiable medical cause, such as heart attack or heatstroke.

For the purposes of this dissertation the following definition has been adopted:

Exercise associated collapse is:

The inability to walk or stand alone (unsupported) because of light-headedness, dizziness, weakness, faintness or syncope during or following participation in physical exercise.

A clinician on duty at an athletic event cannot choose whether he or she will only see the "sporting" causes of collapse. The collapsed patient is presented to the clinician and the management must follow.

For the average General Practitioner who becomes involved in managing a patient who has collapsed after or during endurance events, I would like to propose the following approach, which I have found useful, and which I think is practical.

Preamble:

Endurance events are popular and on the increase. When an athlete collapses there must be a cause for the collapse. Most athletes these days who partake in these events are fairly clued up about fluid and nutrition. Dehydration is only one of the causes of Exercise Associated Collapse but is not as frequent a cause as is often assumed. Gone are the days when a collapsed athlete in an endurance event is assumed to have collapsed as a result of dehydration and is immediately given a "drip" (intravenous fluid).

Most sporting conditions causing exercise associated collapse are usually not dire emergencies and can be approached and treated in the time-honoured way of first making a diagnosis before treating.

In the majority of cases an adequate history, clinical examination and investigations with the simple aids in doctors' bag will lead to a rational diagnosis and management.

Few of the athletes will require more sophisticated investigations.

THE PRACTICAL APPROACH TO THE COLLAPSED ATHLETE:

1. Emergency assessment and stabilisation.
2. Assessment of condition - diagnostic workup
2.1 History
2.2 Examination
2.3 Special investigations
2.4 Working diagnosis
3. Rational management.

1. Emergency assessment and stabilisation
   A. Airway
   B. Breathing
   C. Circulation
   D. Disability
   E. Evaluate

This is the basic approach to any medical emergency and detailed approaches will not be discussed here.

1.1 Airway and breathing
   Determine whether the airway is patent and ventilation is adequate. Manage emergency problems e.g. spontaneous pneumothorax etc. Oxygen is important.

1.2 Circulation
   BP, pulse and rhythm are important. Stabilise acute cardiovascular emergencies e.g. Myocardial infarction.

1.3 Disability
   Neurological Status
<table>
<thead>
<tr>
<th>AVPU</th>
<th>Glasgow Coma Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fully</td>
<td>Awake</td>
</tr>
<tr>
<td>Responds to Painful Stimulation</td>
<td>Verbal: 1-5</td>
</tr>
<tr>
<td>Eye:</td>
<td>Motor Response: 1-6</td>
</tr>
</tbody>
</table>

The crucial aspect is to make sure the athlete is neither confused, nor neurologically obtunded.

2. Assessment of condition diagnostic workup

After initial emergency evaluation and stabilisation, if required, the cause of the collapse must be sought. Rational management can only follow upon a rational approach to diagnosis and establishment of a diagnosis. We reiterate the point that exercise associated collapse in an endurance athlete does not equate with dehydration and a drip.

If the condition was not readily identifiable in the emergency assessment and stabilisation, an approach along the lines proposed by Meeuwisse leads to a working diagnosis and rational therapy. Obviously all readily identifiable medical conditions such as myocardial infarction will be treated along conventional lines.

2.1 History (using Meeuwisse's model)

Consider:

2.1.1 Intrinsic factors
   Factors peculiar to the specific athlete
   = The predisposed athlete +

2.1.2 Extrinsic factors
   Factors external to the athlete that affect the athlete
   = The susceptible athlete +

2.1.3 Inciting event
   = Injury or condition
2.1.1 Intrinsic Factors:
These are factors internal to the athlete i.e. those which the athlete brings to the event:
1. The athlete's genes i.e. somatotype, flexibility, age, gender, biomechanics, psychological status.
2. The athlete's illnesses and previous injuries
3. State of fitness
4. Nutritional status
These factors then leave you with an athlete who has a predisposition for certain injuries or conditions = The predisposed athlete.

E.g. A 23-year-old underweight amennorhoeic lady will be predisposed to a different spectrum of conditions than will a 55-year-old overweight ex rugby prop forward with osteoarthritis of a knee as a result of a cruciate ligament injury during his rugby career.

2.1.2 Extrinsic Factors
These are factors that impact on the athlete from without.
1. The course
2. The equipment e.g. running shoes, weight and/or make of canoe etc.
3. The climate
4. The time
5. The rules of the event
6. Pre- and intra-event nutrition and fluid intake

Once you add to a predisposed athlete (from the athlete's intrinsic factors) the extrinsic factors you are left with a susceptible athlete.

E.g. the downhill Comrades marathon run in high ambient temperatures on the 31st of May provides a different set of risk factors than an uphill Comrades run on the 17th of June in freezing cold and rainy weather.

2.1.3 The Inciting event e.g.
1. Going out too fast
2. Not drinking enough
3. Ingestion of medication

The susceptible athlete now is exposed to the INCITING event and the collapse occurs.

An example of the type of problem presented.
E.g. a burly macho 50 year old ex rugby player planning to run a 10-hour Comrades is passed during the run by a young slim female company representative who calls on him for business reasons. She jokingly calls on him to tag along and he responds. For the next 20 km he runs at a pace far too strenuous for his physique, training and osteoarthritic knee. When this begins to really hurt, he accepts some unknown "anti-inflammatory" from a well meaning fellow Comrades struggler, despite the fact that he has had peptic ulcers in the past. He now begins to feel nauseous, vomits a few times and is unable to keep any fluid down during the last 15 kilometres. He staggers into the stadium and beats the finishing pistol with 45 seconds to spare. When carried into the medical tent he is grey, drawn and ashen, but still grasps his medal triumphantly in his hand.

2.4 Establish working diagnosis

3. Rational management.
The 10 steps to take when presented with a collapsed patient.
1. Place patient supine on a bed or floor (unless contraindication e.g. vomiting)
2. Check Airway, breathing,
3. Check pulse and circulation
4. Assess neurological status (? confusion)
5. Temperature, BP, Pulse rate, Blood glucose
6. Elevate feet (Common cause of collapse is postural hypotension)
7. Comprehensive history a la Meeuwisse's schema
8. Clinical examination and special investigations
9. Make a provisional working diagnosis
10. Manage accordingly.

Helpful Aphorisms:
Here will follow some aphorisms that may help in the assessment. Some are direct quotations and are then referenced. Others are a combination of facts gleaned from the bibliography and practical tips gained from colleagues or from personal experience.

The event and many of these have postural Hypotension.

* Common Symptoms in collapsed athletes.

Feel weak; Exhaustion; Cramps in stomach; Cramps in the legs; Feeling hot; Feeling cold; Unable to stand; Fatigue; Dizzy; Light headedness; Nausea; Confusion; Nausea/Vomiting; Headache; Feel terrible.

* In a study performed during an 80km footrace all runners were dehydrated by the race activity with
a range of 1% to 7% and an average of 4.6%. Despite this their cardiovascular status in the supine position was not greatly compromised. The level of dehydration was unrelated to the degree of postural hypotension. 68% of the athletes in this study developed (asymptomatic) postural hypotension.4

* Clinically severe dehydration is where there is a fall in body weight of 5-10%. Most authors report no more than 2-4% loss of body weight during prolonged marathon running (10 references quoted by 2) and levels of dehydration are usually less in those who have participated in events of even longer duration.6

* Symptomatic Hyponatraemia occurs in only a small percentage of competitors in endurance events but it constitutes a significant amount of the collapsed athletes - approx. 10%.5

* Approximately 60% of athletes who end up in the medical tent after the Comrades marathon ingest some form of medication before or during the event. Approximately 30% of athletes who do not end up in the tent also take medication. (As yet unpublished research conducted at 1996 and 1997 Comrades marathon by Dr Andrew Paterson et al)

**Assessment of the likelihood of severity of collapse:**

<table>
<thead>
<tr>
<th>PROGNOSTIC FACTORS</th>
<th>MORE SERIOUS</th>
<th>LESS SERIOUS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timing of collapse</td>
<td>During the event</td>
<td>After the event</td>
</tr>
<tr>
<td>Neurological / Conscious status</td>
<td>Affected</td>
<td>Unaffected</td>
</tr>
<tr>
<td>Temp. (Rectal)</td>
<td>&gt;40°C</td>
<td>&lt;40°C</td>
</tr>
<tr>
<td>BP (Systolic)</td>
<td>Low &lt;100</td>
<td>Normal &gt;100</td>
</tr>
<tr>
<td>Pulse Rate</td>
<td>&gt;100</td>
<td>&lt;100</td>
</tr>
<tr>
<td>B/Glucose</td>
<td>&lt;4 &gt;10</td>
<td>4-10</td>
</tr>
<tr>
<td>Na</td>
<td>&lt;130 &gt;148</td>
<td>135-148</td>
</tr>
<tr>
<td>Wt loss</td>
<td>&gt;10%</td>
<td>0-5%</td>
</tr>
<tr>
<td>Previous collapse</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Underlying medical condition</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Medication taken</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>

**TO DRIP OR NOT TO DRIP?**

The infusion of intravenous fluids is not without danger as pointed out by Noakes.12,13

However, a comment by ERE in Sports Medicine digest May 1995 Page 6, sums up a lot of prevalent thought. "Most runners who collapse at the finish line don’t need IV’s. However, Noakes exaggerates the negligible risk of the common medical tent practice of giving 1-2 litres Dextrose/Normal saline to collapsed runners. There is not even a single published study in which giving 1-2 litres NS to an athlete who proved to be hyponatraemic harmed him or her clinically. Such athletes get into trouble from the huge amount of water (or other hypotonic fluid) they drink during the race e.g. 12 litres, not the small amount of normal saline they may get intravenously after the race."

My own personal experience confirms what Noakes says and I think the above statement is erroneous. After the 1996 Comrades, whilst conducting a study on

**Causes of collapse in endurance events**

<table>
<thead>
<tr>
<th>CARDIAC METABOLIC</th>
<th>RESPIRATORY</th>
<th>CEREBRAL</th>
<th>DRUGS</th>
<th>MISCELLANEOUS</th>
<th>TRAUMA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cor. art disease</td>
<td>Asthma</td>
<td>CVI</td>
<td>Stimulants</td>
<td>Postural hypotension</td>
<td></td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>Spontaneous pneumothorax</td>
<td>Seizure</td>
<td>Cocaine</td>
<td>Hyperventilaion</td>
<td></td>
</tr>
<tr>
<td>Congenital abnormalities</td>
<td>Valvular lesions</td>
<td>Pulm Embolism</td>
<td>Erythropoietin</td>
<td>Hysteria</td>
<td></td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Hyperthermia</td>
<td>Hypothermia</td>
<td>Overhydration</td>
<td>Stress Fracture</td>
<td></td>
</tr>
<tr>
<td>Hyperthermia</td>
<td>Hypothermia</td>
<td>Overhydration</td>
<td>Hypothermia</td>
<td>Hypoplasia</td>
<td></td>
</tr>
<tr>
<td>Overhydration</td>
<td>Hyponatraemia</td>
<td>Hypoplasia</td>
<td>Hypoglycaemia</td>
<td>Trauma</td>
<td></td>
</tr>
</tbody>
</table>
measuring Central Venous Pressures in collapsed athletes, we evaluated two athletes who had been treated in the medical tent with intravenous fluid and had not responded but deteriorated.

Both athletes were hyponatraemic and their CVP measurements were 18 and 27 cm H₂O respectively.

Both were severely neurologically obtunded and confused. One patient convulsed after referral to hospital (the doctor in Casualty ignored the referral letter, removed the central venous line and discharged the patient). The patient was insistent that he was fine and wanted to go home (His friends had brought him to the medical tent because he was "confused and not right"). Fortunately for the patient he had a convulsion just outside the doors of casualty. He spent the night in intensive care and after a massive diuresis was discharged the next day.

The ages of the athletes were 35 and 45 years old and they were experienced runners with many miles behind them.

At the 1996 'Dusi Canoe Marathon an athlete who was given a drip on the first day returned on the second day for another - as it made him feel so good. I declined on the grounds that clinically there was no indication. He was insistent so under the protection of Central venous pressure monitoring he was given a litre of fluid. His initial CVP was 8 cms of water and this increased to 15 cm of water after the litre. He suffered no clinical symptoms and in fact said he felt a whole lot better for it!

Following our experience at this Dusi a new ruling was made for the 1997 Dusi Canoe Marathon. Athletes could have intravenous fluid but could only qualify for a Finisher’s medal - not any other medal. In the 1996 Dusi we administered 253 litres of fluid. In the 1997 Dusi we administered 3 drips and one of them was for a fractured lower leg!

In young athletes with an expandable myocardium and an accommodating cardiovascular system it probably does not do harm to administer an intravenous drip of 1-2 litres. Youth and a forgiving physiological system will sort out the problem.

However, in older athletes this may not be the case and the procedure is potentially harmful.

In practice the histories of the athletes as to fluid ingestion are often not accurate, the clinical signs of hyponaaterialia are vague and non-specific and a patient with hyponatraemia very often is confused!

The excuse that this has been justified because the athletes were treated under difficult conditions in the field is not acceptable.

HENCE:

1. Administering intravenous fluid is definitely potentially harmful.
2. It should only be done following an attempt at a rational diagnosis and preferably with the help of objective evidence of fluid depletion i.e. documented weight loss, serum hypernatraemia, raised hematocrit or central venous pressure monitoring. One often does not have these facilities in a medical tent.
3. If one has administered IV fluid and the patient does not improve after 1-2 litres further investigation - CVP and Serum electrolyte/hematocrit are indicated before further fluid is given.

4. The majority of collapsed athletes do not need intravenous fluid. 12,13

The last word should go to Noakes as he and his colleagues have challenged conventional dogma and made endurance events safer for athletes:

"There is no evidence that, if the diagnosis is not readily apparent, withholding therapy for a few minutes will have any detrimental consequences. The wrong diagnosis and the wrong therapy can result in serious injury and even death." (Clinical Journal of Sports Medicine, 1995 5920:123-128)

"A course of treatment should be prescribed only when some basic investigations have led to a diagnosis that has a reasonable probability of being correct. By failing routinely to make the most simple measurements including heart rate, blood pressure, rectal temperature, and serum sodium and blood glucose concentrations in collapsed athletes, we have over treated many subjects, incorrectly treated others and produced iatrogenic, sometimes life-threatening, conditions in still others. The excuse that this has been justified because the athletes were treated under difficult conditions in the field is not acceptable. The collapsed athlete deserves optimum treatment based on the same clinical judgements, investigations and skills that should be applied to all medical conditions."

Kirkby, R
Family Physician
Piyadh, Saudi Arabia
Dear Colleague

Not too long ago I was caught in a situation, which could have cost me my life. Luckily I remembered what an aged colleague taught me years ago and this, I am convinced, saved me. I was called to the emergency room for a male patient with severe acute asthma. The patient really was in bad shape; he was fighting for air and was confused. I took out my stethoscope and started auscultating his chest. The next moment the patient got hold of my tie and started pulling. I grabbed his hand to try and free the tie, but the man was so strong that I could not open his clenched fingers. I could not even scream for help because I was choking. Suddenly the solution to the problem flashed through my hypoxic brain! I grabbed his wrist with my left hand and with my right hand I forcefully flexed the wrist. By doing so his fingers automatically relaxed and I could free my tie. What a close shave!

Since that day I never wear a tie when working in the emergency room.

The moral of the story?........
Should doctors wear ties?

Best wishes,
Patrick