

Heat cramps: fluid and electrolyte challenges during tennis in the heat

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Sweat losses during tennis can be considerable. And while most players make a genuine effort to stay well hydrated to maintain performance and reduce the risk of heat illness, regular and copious water intake is often not enough. Besides an extraordinary water loss, extensive sweating can lead to a concomitant large electrolyte deficit too - particularly for sodium. Although a variety of other mineral deficiencies and physiological conditions are purported to cause muscle cramps, evidence suggests that, when a tennis player cramps in warm to hot weather, extensive and repeated sweating during the current and previous matches and a consequent sodium deficit are usually the primary contributing factors. Heat cramps often begin as subtle "twitches" or fasciculations in one or more voluntary muscles and, unless treated, can rapidly progress to widespread debilitating muscle spasms that leave an afflicted player on the court writhing in pain. If sufficient preventive measures are taken well before and during play, such cramping is avoidable in most cases. Appropriate and sufficient salt and fluid intake will enhance rehydration and fluid distribution throughout a player's body, so that heat cramps can be completely averted, even during long matches in the most challenging environments.

Introduction

Playing tennis in the heat presents a formidable challenge, even to the most fit players. Comments such as 'It's so hot, it's a joke', 'My feet were on fire', and 'I had the feeling that my brain was cooking' are not uncommon at notoriously extreme hot-weather events such as the Australian Open in Melbourne ⁽¹⁾. And throughout the world, many other tournaments are equally or more challenging, especially when the heat is combined with high humidity.

Through the efforts and emphases of tennis governing bodies ⁽²⁻⁴⁾ and a number of prominent national organisations ⁽⁵⁻⁷⁾, most players, coaches, and trainers acknowledge the importance of staying well hydrated in order to maintain performance and reduce the risk of heat illness ⁽⁸⁻¹¹⁾. However, regular and copious water intake is often not enough - in fact, excessive water intake can even be dangerous to a player's health ⁽¹²⁻¹⁴⁾.

Fluid losses

Adult and older adolescent tennis players generally lose between 1.0 and 2.5 litres of sweat during each hour of competitive singles play in warm to hot environments ⁽¹⁵⁾. As indicated in Table 1, on-court sweating rates can often be even much higher in fit, acclimatised players. With a very high rate of sweating, a player could readily incur a significant fluid deficit in a long match,

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even if s/he drinks considerably on each changeover. For example, a sweat loss of 3.0 or more litres per hour would likely lead to an accumulating body water deficit of at least 1.0 litre per hour (i.e., a loss of about 1.0% or more of prematch body weight per hour). If this continued over the course of a 4-hour match, this player would be facing a 4.0+-litre fluid deficit, even if on-court fluid intake was 2.0 litres per hour, which may be close to the upper limit for most adults with respect to tolerance and fluid absorption rate ⁽¹⁶⁾. Because, to fully rehydrate, one needs to ingest more than what is remaining as a post-exercise fluid deficit ⁽¹⁷⁾, an additional 6 litres or more of fluid (via drinking and food) would need to be consumed, in order to restore the 4.0-litre deficit. Such a requirement might seem to be a daunting task, if one expected to begin the subsequent match in a euhydrated condition, especially if the next match is scheduled for later that same day. This is often a perspective that many players do not fully appreciate - the extensive volume of fluid that must be replaced in a relatively short period. Which may help to explain why many tennis players *begin* a match with a significant fluid deficit (Bergeron, unpublished observations).

Player	Age (y)	Wt. (kg)	Temp. (°C)	% rh (%)	Sweat Rate (L/hour)	Fluid Intake (L/hour)	Sweat [Na ⁺] (mmol/L)	Na ⁺ loss (mEq/hour)	Na ⁺ loss (mg/hour)
AJ	17	84.5	30.6	60	2.6	1.3	23.0	59.8	1375
BJ	22	76.3	28.9	-	2.2	1.4	52.0	114.4	2630
GK	22	79.4	32.2	62	3.1	1.8	23.6	73.2	1683
GJ	21	85.7	32.2	62	2.9	1.4	53.4	154.9	3561
HR	21	77.4	35.6	50	3.0	2.4	56.1	168.3	3869
LC	17	80.7	33.3	58	2.5	1.4	83.0	207.5	4770
LY	24	77.1	31.1	35	2.0	1.1	40.0	80.0	1839
MC	18	77.1	29.4	-	2.1	1.0	32.3	67.8	1559
MD	19	86.5	32.0	64	3.3	1.9	32.1	105.9	2435
OA	24	85.6	33.9	55	3.4	2.3	60.8	206.7	4752
RM	28	86.6	28.9	50	2.8	1.5	44.1	123.5	2839
RR	15	90.4	29.4	-	2.0	1.4	44.9	89.8	2064
SR	49	80.7	33.3	45	2.5	1.3	39.2	98.0	2253
SJ	16	68.2	30.6	61	2.4	1.3	39.0	93.6	2152
WA	17	81.4	32.2	61	2.5	1.8	35.9	89.8	2065
WJ	20	80.8	35.6	50	2.8	1.9	48.0	134.4	3089
WM	28	78.5*	33.3	58	2.9	2.3	48.2	139.8	3214
Mean	22.2	81.0	31.9	55	2.6	1.6	44.5	118.1	2715
±SE	±1.9	±1.3	±0.5	±2	±0.1	±0.1	±3.5	±11.0	±252

Table 1: Sweat rate, fluid intake, and forearm sweat sodium concentration ([Na⁺]) and total estimated rate of loss (in mEq/h and mg/h) during tennis singles play. Player age and body weight (Wt.), as well as ambient temperature (Temp.) and % relative humidity (% rh) are also shown (if known). All players (17 males) listed here had a history of heat cramps during tournament play (not in these evaluated matches).

Electrolyte Losses

Again, the importance of hydration during tennis, especially in the heat, is well acknowledged; accordingly, most players make an effort to regularly consume fluids (albeit, sometimes not enough) on and off the court. Less emphasised and clarified, however, are guidelines to maintain electrolyte balance and the associated consequences of insufficient electrolyte intake.

Besides an extraordinary water loss, extensive sweating can lead to a concomitant large electrolyte deficit too. Tennis players who sweat profusely lose considerable electrolytes - particularly sodium and chloride. Other minerals in varying concentrations are also found in sweat ⁽¹⁸⁾. But, for most players, far more sodium and chloride are lost via sweating than any other electrolytes (e.g., potassium, magnesium, and calcium), even for players who are fit and acclimatised to the heat ^(15,19,20). Moreover, as sweating rate increases, the concentrations of sodium and chloride in sweat go up as well ⁽²¹⁾.

As implied by Table 1, substantial sodium losses can occur during a long match in the heat. Consequently, a sizable deficit in total body exchangeable sodium could readily develop over the course of several matches, especially if the player follows prevalent current dietary guidelines designed to prevent and treat high blood pressure ^(22,23) and maintains a low-salt diet (i.e., < 2400 mg of sodium intake per day) when playing in the heat. However, just as a very high rate of sweating (e.g., > 2.0 L per hour) can be difficult (if not impossible) to keep up with, it is also impractical to attempt to offset these accompanying high rates of sodium loss *during* play. Therefore, appropriate sodium and chloride (salt) intake to offset substantial losses and deficits of these particular minerals has to be primarily emphasised and integrated into a player's post-match rehydration plan (see Prevention below). Insufficient salt replenishment and a consequent deficit of exchangeable sodium increase the risk for (and often lead to) heat-related muscle cramps (heat cramps) - a progressive condition that can evolve from having merely a neutral effect on performance to leaving an afflicted player on the court writhing in excruciating and debilitating pain.

Heat Cramps

A variety of mineral deficiencies - namely calcium, magnesium, and potassium - have been proposed for causing muscle cramps during exercise ⁽²⁴⁻³³⁾. No doubt, any player who has a deficiency in one or more of these minerals or some other rare cellular dysfunction or pathophysiology ⁽³⁴⁾ could certainly present with muscle cramps or various other neuromotor problems. Accordingly, calcium, magnesium, or potassium supplementation (or other prophylactic approach) may be warranted. However, when a tennis player has muscle cramps in the heat, these particular minerals do not appear to be lacking in the diet or lost in great quantities from sweating during training or play (Bergeron, unpublished findings). Insufficient conditioning and fatigue can also cause a muscle cramp in an overworked muscle ^(35,36), but the cramp is usually localised and often readily resolved by a trainer using passive stretching, massage, or icing. With heat cramps, the affected areas are typically more widespread across a number of voluntary muscle groups and these same treatment strategies are usually ineffective.

Alternatively, extensive water and *sodium* losses from profuse and repeated sweating have also been often cited as primary contributing factors prompting the onset and development of heat cramps (13,20,24-26,28,32,37,38). Consistent with these previous reports and suggested scenarios, all of the players listed in Table 1 had a history of heat cramps occurring during tournament play, when concomitant heavy and repeated sweating was likely not matched with sufficient fluid and sodium intake. Severe heat cramp episodes often occurred in later rounds of play (particularly during second matches of the day, with some of the junior players), but not always. Moreover, certain players in this group claimed that heat cramps would sometimes even occur during indoor events or outdoors when the heat index was only moderate. However, in all cases, each player was reportedly sweating considerably and previous match play or a period of extensive training and practice closely preceded the cramping episode (circumstances that could promote significant cumulative fluid and sodium deficits).

As progressive dehydration continues, the extracellular fluid compartment becomes increasingly contracted (39). A loss of interstitial volume can cause a mechanical deformation of nerve endings and increase the surrounding ionic and neurotransmitter concentrations. This can, in turn, cause selected motor nerve terminals to become hyperexcitable and spontaneously discharge (37,40). A player's first indication of the onset of heat cramps is often very subtle, almost undetectable "twitches" or fasciculations in one or more voluntary muscles (such as the calves or quadriceps). These intermittent muscle twitches may be particularly evident while sitting during a changeover period. Players with a history of heat cramps recognise that soon (~20-30 minutes) they may be facing full-blown, severe muscle cramps, unless the match ends or something is done to address the problem appropriately. This is often the time when a perceptive player will request a trainer (if one is available). Even as the severity continues to evolve and cramps become more widespread, as a rule, only a limited number of localised muscle bundles at a time contract in a particular muscle. When certain muscle fibres relax, adjacent bundles subsequently contract, as a cramp seemingly "wanders" (41). Electromyography supports this notion, as periodic high-voltage discharges are shown to spread across an afflicted muscle, from one area to another, with irregular fasciculations occurring between cramps (37).

Notably, a deficit in total body exchangeable sodium often cannot be detected by just measuring serum or plasma levels (42). In fact, a cramping tennis player will often present with normal or slightly elevated plasma sodium, prior to receiving oral or intravenous fluids. This can mistakenly lead one to "confirm" the absence of a sodium deficit. Because sweat is hypotonic (compared to plasma), dehydration (within limits) will cause a proportionate adjustment in total body water (via each fluid subcompartment), with an initial primary emphasis on "borrowing from" (decreasing) the interstitial fluid volume (43), so that normal plasma osmolality is maintained. Thus, to a point, plasma volume is partially "defended", particularly during an intermittent sport such as tennis. Moreover, even a prematch assessment of plasma sodium may not be a valid predictor of an individual's susceptibility to heat cramps. Alternatively, a 24-hour urine collection indicating extreme renal sodium conservation would be more indicative of a significant exchangeable sodium deficit.

Table 1 indicates that players can have considerable variation in sweat sodium concentration. Sweat was collected from the forearm during play, using a regional sweat collection patch (Pacific Biometrics, Inc., Irvine, CA) and calculating sweat and mineral loss rates using previously described methods (20,44). Of course, with a high sweat rate and a concomitant high sweat sodium concentration (e.g., player LC), it's easy to appreciate the potential for an extensive deficit in total body exchangeable sodium from a long match. However, even with a relatively low level of sodium in sweat (characteristic of a fit, heat-acclimatised player), one can still incur a substantial mineral deficit when the sweating rate is high, given the opportunity (i.e., long-duration, repeated play on successive days and insufficient salt intake) to do so. Which is why many players who live and train in hot and humid climates are often surprised by their susceptibility to heat cramps. Notably, all players listed in Table 1 were acclimatised to the heat, at the times that these on-court evaluations were conducted.

Treatment

At the first signs of muscle twitching, a tennis player can avert or at least avoid a serious episode of heat cramps by consuming an appropriate salt solution - for example, 3 g (0.5 tsp.) of salt dissolved in 16-20 ounces of Gatorade®. Drinking half of this mixture on the current or next changeover (along with a little water), and the rest on the subsequent changeover, has been a proven effective strategy for many players in an effort to prevent further development of or relieve such cramping, so that play continues without interruption. If the situation is more urgent, intravenous rehydration (e.g., 0.5-1.0 L of normal saline) may be necessary and more promptly effective. Oftentimes, a player readily recovers, without any apparent detrimental effects and is able to perform well the next day.

Quinine administration continues to be regarded as an appropriate and favored option by some on-site trainers for treating heat cramps. Reportedly, quinine lessens motor end plate excitability, while increasing the muscle membrane refractory period; although, empirical evidence suggests both efficacy and no benefit (45). Besides a possible consequent decrease in performance related to such neuromotor changes, other potential adverse effects (e.g., hypoglycaemia, nausea, vomiting, and disturbed vision) should also be considered before administering or taking quinine on court.

Prevention

Prevention of heat cramps is clearly the preferred option for tennis players over treatment. Unfortunately, a variety of ineffective "remedies" are often prescribed to players (e.g., trace minerals, amino acids, quinine, bananas, water, and even pickle juice). Notably, all players listed in Table 1 (and myriad others) have been able to avoid heat cramps during competition and training, by appropriately increasing their daily salt and fluid intake in accordance with their individual respective losses incurred from previous play - even during events when the incidence of heat cramps (for other participating players) was high.

Beyond salting meals and choosing food items with high levels of inherent sodium (e.g., tomato juice, sauce, or paste, salted pretzels, canned vegetables

or soups, or pizza), it is sometimes more effective, convenient, and certain to consume appropriate salt containing beverage mixtures (4,46) between matches. For those players especially prone to heat cramps, a little salt (e.g., 1.5 g/L) can be added to their sports drink that is consumed on court during play. Salt tablets can also be effective, so long as they are ingested (perhaps after being crushed and mixed) with enough fluid. Ideally, any such dietary approach to offset sodium and fluid losses should be based on that player's measured specific rates of sweating and electrolyte losses during competition under the particular environmental conditions.

Hyponatremia

In an effort to avoid heat cramps, players are often encouraged to “drink more water”. Although performance and risk for heat illness are directly related to hydration status (8-11), heat cramps can still occur, even when a player has been drinking plenty of water or other low-sodium beverages. Complete rehydration and restoration of the extracellular fluid compartment requires sufficient fluid *and* sodium (47-49). Findings by Mitchell et al. (48) specifically highlighted that higher levels of fluid and sodium intake were needed to obtain the greatest recovery of the interstitial fluid compartment. In the presence of a significant exchangeable sodium deficit, rapid or repeated consumption of too much water (or other low-sodium or sodium-free drinks) will likely not resolve or prevent heat cramps (it may actually exacerbate the risk for heat cramps) and may readily promote hyponatremia - potentially, an extremely dangerous clinical condition (50). Early stages of hyponatremia may prompt a tennis player to feel nothing more than fatigue, slight nausea, and a headache; but it can get much worse. Player LC (Table 1) was feeling nauseous and weak and was cramping after winning a 4-h match played in extreme heat (~38°C). He was advised by on-site medical personnel to drink as much water as he could tolerate and return to his hotel and rest. After leaving the tournament site and consuming a considerable amount of water, he subsequently had a seizure in his hotel room and slipped into a coma. LC was classified as being severely hyponatremic (with an initial serum sodium level of 118.0 mmol/L). Given his tendency for a very high rate of sodium loss during play (Table 1), LC's state of severe hyponatremia was probably hastened by the combination of water excess and an extensive sweat-induced deficit of exchangeable sodium from the prior very long match. Notably, Vrijens and Rehrer (51) demonstrated that athletes do not necessarily have to drink excessive amounts of sodium-free fluid to become hyponatremic. However, if a player is constantly urinating (e.g., every 15-30 minutes), s/he may indeed be drinking too much (and consuming too little sodium). Moreover, frequent urination should not be considered an absolute indication of sufficient rehydration (i.e., complete restoration of all fluid compartments).

Recommendations

- Tennis players should arrive as early as possible to tournament being played in hotter and/or more humid conditions, so that they have the opportunity to acclimatise to the new environment (including conserving sodium).
- Players should drink plenty of fluids (water, juice, sport drinks, etc.) throughout the day; but they should also be careful to not “over-hydrate”.

- Players prone to heat cramps should add some salt to their diet and possibly include additional salt in their on-court sports drink.
- Certain players should consider having their sweat rate and sweat electrolyte losses measured, so that specific and effective strategies can be developed and applied for sufficiently and appropriately maintaining fluid and mineral balance.
- If heat cramps persist, players should consult with their doctor about potential other causes related to medications, an underlying illness or metabolic disorder, or other predisposing factors.

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