Exercise Induced Collapse: "Hitting the Wall"

Background

1. "Hitting the Wall" / "Bonking"
    - Severe energy loss during prolonged exercise
    - Runners collapse around 20 mile point in marathon
    - Possibly due to depletion of muscle glycogen
        - Fat stores (even in leanest runners) are inexhaustible
        - Unable to utilize fat without carbohydrate as primer for metabolism

Pathophysiology

1. Old theory: exhaustion of carbohydrate fuel
    - Muscle glycogen or blood glucose falls to critically low level
    - Glycogen depletion contributes to muscle fatigue
        - At 80 % maximum capacity
    - Glycogen content of muscles dropped near zero in 90 min
        - Marathon at normal pace
    - Fuel consumption ratio:
        - 75 % carbohydrates
        - 25% fatty acids
    - Carbohydrate supplies fall
        - Body relies on fatty acids
    - Body stores 2,000 calories of glycogen in muscles and liver
        - Enough for about 20 miles
2. New theory: CNS fatigue
    - Muscle damage may cause "hitting the wall"
    - Protective mechanism
        - Mediated by interleukin-6
            - Brain decreases muscle stimulation
            - Discomfort/exhaustion when muscle damage approaches dangerous levels during prolonged exercise
        - Mediated by serotonin
            - Prolonged exercise increases serotonin production
            - Tryptophan-amino acid
                - Precursor to serotonin
                - Increase levels with muscle damage
                - Increased tryptophan
                    - Leads to increased serotonin
            - Increased serotonin causes brain to stimulate release of serum fatty acid
            - Increased serum fatty acid causes increased CNS fatigue
        - Mediated by insulin
            - Protein stimulates insulin release
            - Insulin speeds muscle cells' absorption of blood glucose by 50%
            - Transports amino acids into muscle
            - Decreased release of stress hormone cortisol
            - Stimulates blood flow to muscle

Diagnostics

1. Detailed history of syncopal event
    - Time frame of collapse
        - Around 20 miles
2. Serum blood sugar for hypoglycemia
3. Serum Interleukin-6 level for muscle tissue damage
    - Experimental only
    - Possible correlation with exercise intensity
    - Does not correlate with length of exercise
4. Serum serotonin/tryptophan level
    - Experimental only
5. Insulin level

Therapeutics

1. CHO supplementation during exercise studies
    - 1960s-subjects exercised to exhaustion
        - Consumed 200 g of glucose
        - Extended performance by one hour
    - 3 fluids: 4:1 CHO-protein solution, normal sports drink, water,
        - CHO-protein beverage: 30 min
        - Carbohydrate-only group: 20 min
        - Water only group: 14 minutes
    - Endurox R4/Accelerade
        - Use a 4:1 carbohydrate-protein ratio
        - Enough protein to stimulate insulin secretion
        - Not enough to stimulate peptide enzymes and induce gastric distress .

Training/Prevention

1. "Hitting the Wall" - multifactorial
2. Prevention needs to address diet and training:
3. Carbohydrate loading pre-event
    - 7 day pre-event cycle
        - Low CHO diet first three days to deplete glycogen stores
        - 70% CHO diet final four days
        - OR 75% CHO diet 7 days
            - Taper intensity of training 7 days prior to event
    - Both methods increased muscle glycogen up to 150%
4. Physiologic goal of training: optimize aerobic metabolism
    - Muscles increase utilization of oxygen
        - Due to increased size/number of mitochondria
        - Increased aerobic enzymes
    - Trained muscles better mobilize and use fat for energy
        - Preserves carbohydrate stores
    - Some muscle fibers can be adapted for aerobic or anaerobic exercise
        - For marathon, can adapt convertible muscles for aerobic/endurance work
    - Training increases:

- Number of capillaries
- Muscle nutrient supply
- Adaptation of cardiovascular/respiratory systems
  - Heart muscle: increased size, weight, blood volume
  - Resting and submaximal exercise heart rates decreased
  - Stroke volume-increases distribution of blood/oxygen to active muscles
- Maximal oxygen uptake (VO2 Max)
  - Quantitive measure of capacity for aerobic energy transfer (ability to do work)
- Variables that determine VO2 Max:
  - Heredity
  - Sex
  - Body composition (amount of lean body tissue)
  - Age
  - Training
- Can improve VO2 Max up to 20-25
  - Peaks 6 months-2 years after implementing endurance training
  - Typical marathoners can maintain pace using 75-80% VO2 Max for over 2 hr
  - Ultramarathoners able to work for prolonged periods at near 90% VO2 Max
- The ability to perform at higher VO2 Max
  - Probably related to anaerobic threshold
    - Lactic acid accumulation impairs performance
    - Anaerobic threshold can be increased by endurance training
  - Improved anaerobic threshold
    - Allows longer period before anaerobic metabolism dominates
      - Lactic acid accumulation affects performance

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