Quick Review: The Metabolic Cart
T Fujii, B Phillips

Citation

Abstract
This article gives a brief review of the metabolic cart.

TERMS & DEFINITIONS

CALORIMETRY

Direct calorimetry: Measurement of the amount of heat (energy) produced by a subject enclosed within a Chamber.

Indirect calorimetry: Measurement of the amount of heat (energy) produced by a subject by determination of the amount of oxygen consumed and the quantity of carbon dioxide eliminated

- The metabolic cart essentially measures the oxygen consumed and the carbon dioxide produced by the patient and then calculates (using the modified Weir equation) the energy expenditure for the patient
- Weir equation: $EE = (3.94 \times VO_2) + (1.1 \times VCO_2)$

Indirect calorimetry provides two pieces of information:

1. A measure of energy expenditure as reflected by the resting energy expenditure (REE)
2. A measure of substrate utilization as reflected in the respiratory quotient (RQ)

RESTING ENERGY EXPENDITURE (REE)

REE = 75% - 95% of total energy expenditure (diet-induced, environmental, activity)

- Metabolism in brain, liver, heart and kidney is relatively constant (60 - 70% REE)
- Variability in REE:
  - during the day 12%
  - increases with critical illness
  - day to day - 23%

Variability in REE can be due to:
- Size
- Gender
- Age
- Work of breathing (2 - 3%)
- Diet-induced thermogenesis
- Sleep
- Illness
- Starvation
- Fever (13% per degree C)
- Cold
- Activity
- Drugs

POINTS:
- REE correlates closely with fat free lean body mass.
- Work of breathing normally (2 - 3%) can be as high as 25% of REE with impending respiratory failure
- Diet-induced thermogenesis normally 8-10% drops
Quick Review: The Metabolic Cart

• to 4-8% with continues enteral feeds

• REE drops with sleep

• Most disease states increase REE

• 20-50% increase seen with elective surgery and trauma, 100% increase in REE seen with severe burns

• Increases in REE are seen in the flow phase of injury and can be effected by therapy

• Increases in REE can be expected to reflex severity of illness but the response plateaus at 2x the REE

• Increases in REE due to acute illness usually return to base line at 7 to 10 days

• Not all critically ill patients become hypermetabolic (35-60%)

• 15-20 % of ICU-patients are found to be normometabolic.

• 10 -20% of ICU-patients are found to be hypometabolic.

• Hypometabolic state may be do to the disease process: specific cancers , cachexia , spinal cord injuries paraplegics decreased REE by 10% quadriplegics by 30%

• Long-term starvation reduces EE by 30-40%

• Fever increases REE (13 % per degree C)

• Exposure to Cold /hypothermia increases REE by shivering and nonshivering thermogenesis

• General Activity is responsible for most of the variability in REE.

• Being awake and alert increase EE by 10%.

• Routine nursing care increases EE by 20 -30%.

Medications that affect REE:

• caffeine, aspirin increase EE

• catecholamines and pressor increase EE

• sedatives, analgesics, beta blockers

* general anesthesia decreases EE

** Acute hyperventilation increases EE, while hypoventilation decreases EE

PREDICTING REE

Harris-Benedict is correct 80-90% of the time in healthy, normal volunteers. In 10-14% it over-estimates EE. In obese volunteers, the equation predicts EE correctly only 40-64% of the time. In critically-ill patients the Harris-Benedict equation is correct only 50% of the time. For most disease processes Harris -Benedict underestimates EE. Multipliers for various disease states attempt to improve the accuracy of the Harris-Benedict equation (though these multipliers tend to overestimate EE when compared to indirect calorimetry).

• RQ: Derived from actual measurements of VCO2 and VO2

• RQ is the ratio of carbon dioxide produced to oxygen consumed (VCO2 / VO2)

• Reflection of which fuels are being oxidized

• “Non-protein” RQ (npRQ) excludes protein metabolism

RESPIRATORY QUOTIENT

Ratio of CO2 produced to O2 consumed.

\[
\frac{VCO2}{VO2} = RQ
\]

Carbohydrate: 1gm C + 0.83 L O2 0.83 L CO2 + 0.56g H2O + 4.17 Kcal

RQ = 1

Fat: 1gm F + 2.02 L O2 1.43 L CO2 + 1.07 g H2O + 9.3 Kcal

RQ = 0.70

Protein: 1gm P + 0.96 L O2 0.78 L CO2 + 0.41 H2O + 0.16g Nu + 4.3 Kcal RQ = 0.81

Glucose oxidation RQ = 1.0

Fat oxidation RQ = 0.7

Protein oxidation RQ 0.8

Lipogenesis RQ 1.3

(npRQ of 0.85 - 50 % fat and 50 % carbohydrate oxidation)

“OPTIMAL RQ”

• Nutrition support should probably provide a balance between carbohydrate and lipid with an RQ in the 0.8 to 0.9 range
Avoidance of RQ's > 1.0 which represents “overfeeding” and potential lipogenesis is a reasonable goal.

FACTORS THAT AFFECT RQ:

Those that increase RQ
- Hyperventilation
- metabolic acidosis leading to increases in carbon dioxide,
- overfeeding leading to lipogenesis
- exercise

Those that decrease RQ
- Hypoventilation
- Mild starvation with ketosis
- Diabetes with ketoacidosis or high rates of urinary glucose lose
- Gluconeogenesis
- ETOH metabolism
- Hypothermia via continued gluconeogenesis

Resting energy expenditure of critically ill patients varies widely over the course of the day and over the course of an illness. Measurements from 10 - 23% of an “average” REE can be seen within a 24 hour period. Test patient at rest in quiet, controlled environment. “Steady state” implies a 5 minute interval where the average V02 and the VC02 changes by less then 10% and the average RQ changes by less than 5%.

- Question validity of the test
- Steady state is not achieved
- RQ falls outside of the physiologic range of 0.67-1.3
- Measurements should fall with in the range of V02 (1.7 to 3.4 mL/min/kg) and VC02 (1.4 to 3.1 mL/min/kg)

METABOLIC CART SOURCES OF ERROR:
- FiO2 >60%
- Air leaks (chest tubes etc).
- Hemodialysis (Co2 loss via the dialysis coil )

Use of the metabolic cart can prevent over-feeding, and under-feeding by accurately measuring energy requirements. Overfeeding critically ill patients results in hyperglycemia, hepatic steatosis, R.E.S. dysfunction and increased septic complications. Under-feeding patients can lead to the complications of malnutrition. The use of a metabolic cart can reduce the amount of “unnecessary” TPN which is administered (use of a metabolic cart reduced TPN use from 33,000 liters to 26,000 liters in one study - Mullen et al., Proc Nutr Sos 1991. 50:239-44).

References
Quick Review: The Metabolic Cart

Author Information
Tisha K. Fujii, DO
Dept. of Trauma & Critical Care, Boston University School of Medicine, Boston Medical Center

Bradley J. Phillips, MD
Dept. of Trauma & Critical Care, Boston University School of Medicine, Boston Medical Center