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Energy expenditure and outcome in patients with multiple organ failure following abdominal surgery

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Abstract. A possible relationship between hypermetabolism (energy expenditure expressed as percentage above the estimated basal metabolic rate) and clinical outcome was investigated in 29 artificially ventilated patients with infection and multiple organ failure following abdominal surgery. The average energy expenditure and hypermetabolism were 126±19 kJ (30±5 kcal)/kg/24 h and 36±12%, respectively. Survivors of the intensive care period ($n = 20$) had a 15% higher hypermetabolism (41±11 vs 26±8%, $p<0.01$) than patients who died in the intensive care unit ($n = 9$). Six of the patients died after intensive care. On comparing hypermetabolism in survivors ($n = 14$) and non-survivors ($n = 15$) of the period of hospitalization, no significant difference was discernible. The results demonstrate that patients with multiple organ failure have a moderate hypermetabolism and suggest that the hypermetabolism is comparatively reduced in patients with a fatal outcome during intensive care.

Key words: Indirect calorimetry – Infection – Intensive care – Multiple organ failure – Patient outcome assessment – Prognosis

Trauma and infection induce an increase in energy expenditure (EE) which, in critically ill, artificially ventilated patients, amounts to a figure approximately 30–40% above the estimated basal metabolic rate [1–3]. This response may be diminished by several factors. For example, artificial ventilation has been suggested to reduce the work of breathing and hence EE [4, 5]. The use of sedatives and analgesics may also depress EE [6, 7]. Two investigations have suggested that a fatal outcome tends to be associated with a low hypermetabolism [1, 8] while a third fails to confirm such a relationship [9]. However, these studies either concerned spontaneously breathing patients [8] or did not discriminate between traumatized and non-traumatized, infected and non-infected patients [1, 9].

The purpose of the present study was to investigate a possible relationship between hypermetabolism and outcome in artificially ventilated patients with infection and multiple organ failure (MOF) following abdominal surgery.

Material and methods

Patients

Thirty-nine patients with infection and MOF following abdominal surgery were studied with indirect calorimetry in the intensive care unit (ICU). Infection was considered to prevail in the presence of positive blood cultures or abdominal abscess(es), as verified by laparotomy, or pneumonia verified by radiography. Patients were admitted to the study if they were expected to require artificial ventilation and total parenteral nutrition (TPN) for at least 1 week. Measurements were commenced when the patients had an inspired oxygen concentration below 50% and had a stable circulation as judged from bedside diagnostics, arterial blood pressure and arterial base excess. Measurements were continued until the commencement of weaning off the ventilator, or death.

Ten patients were excluded from the statistical analysis of data on the basis of the following criteria: less than 48 consecutive hours of indirect calorimetry (3 patients), inspired oxygen concentration above 50% (3 patients), technical problems with the indirect calorimetry (1 patient), withdrawal of life-supporting therapy during measurements (3 patients). In the case of patients who died during EE measurements, data from the final 24 h were excluded before analysis.

Preoperative diagnoses, postoperative complications and the number of reoperations are presented in Table 1. The severity of illness was assessed according to Apache II [10] on the first and last day of EE measurements. Due to the use of sedative drugs, the coma score was assumed to be equal to the mean of the other variables in Apache II.

Indirect calorimetry

EE and the respiratory quotient (RQ) were recorded continuously during aritificial ventilation using open circuit indirect calorimetry (The Engström Metabolic Computer®, Gambro Engström AB, Bromma, Sweden). This automatic device incorporates the Haldane correction in the calculation of oxygen uptake and carbon dioxide elimination. EE was computed from the oxygen uptake and the measured RQ and converted to STPD conditions, using corrections for the influence of ambient temperature, barometric pressure and humidity [11]. The accuracy of the inspiratory pneumotachometer was checked with a calibration sy-

Table 1. Preoperative diagnoses and postoperative complications

	The period of intensive care		The period of hospitalization	
	Survivors $n = 20$	Non-survivors $n = 9$	Survivors $n = 14$	Non-survivors $n = 15$
Preoperative diagnosis				
Malignant disease[a]	7 (1,2,1,1,2)	3 (0,0,1,2,0)	4 (1,1,1,1,0)	6 (0,1,1,2,2)
Infection[b]	4 (1,1,1,0,1)	3 (0,1,0,2,0)	3 (1,1,1,0,0)	4 (0,1,0,2,1)
Gastric ulcer	4	1	4	1
Aortic aneurysm	2	1		3
Diabetes mellitus (candidate for pancreatic transplant)	1	1	1	1
Liver cirrhosis		1		1
Chronic renal failure	1	2	1	2
Crohn's disease	1		1	
Colonic angiodysplasia	1		1	
Postoperative complications				
Acute renal failure[c]	5	5	3	7
Sepsis	12	8	8	12
Sustained septic shock	7	5	6	6
Pneumonia	5	1	5	1
Mediastinitis/pulmonary abscess	1			1
Abdominal abscesses	9	4	6	7
Phlegmone of the abdominal wall	1		1	
Peritonitis	1	1		2
Wound rupture	4		3	1
Gangrene of the foot		1		1
Intestinal obstruction	1		1	
Number of patients with 1, 2, 3 and 4 reoperations, respectively	7, 9, 3, 0	6, 0, 0, 1	4, 8, 2, 0	9, 1, 1, 1

Values in the table denote number of patients

[a] Values within parenthesis denote the number of patients with cancer of the oesophagus, stomach, pancreas, colon and the urinary bladder, respectively

[b] Values within parenthesis denote the number of patients with appendicitis, cholangitis/-cystitis, diverticulitis of colon, pyelonephritis/perinephric abscess and oesophageal rupture/mediastinitis, respectively

[c] All patients with acute renal failure received extracorporeal renal replacement therapy (4 patients received hemodialysis and 6 were treated with continuous arteriovenous hemofiltration). Statistical analyses (the Fisher exact probability test and Chi-square test) revealed no significant differences between values in survivors and non-survivors within each period

ringe. The carbon dioxide analyser was manually calibrated daily and the oxygen analyser every 4 h using an automatic calibration cycle.

Controlled mechanical ventilation with ventilatory settings in compliance with the optimal measuring range of the equipment were employed [11].

Nutrition

TPN was provided at least 2 days before measurements. During the period of measurements, energy (including amino acids) was administered in amounts corresponding to 105–125% of the 24-h EE. Non-protein energy was supplied as approximately 50% fat (Intralipid® 20%, KabiVitrum AB, Stockholm, Sweden) and 50% as glucose. The intake of amino acids (VaminGlucose® KabiVitrum AB, Stockholm, Sweden) was provided as anticipated to be tolerated on considering individual liver and renal function. Trace elements and vitamins were supplied daily. Enteral diets were not employed.

Appropriate analgesia and sedation were produced with morphine, pethidine, phenoperidine and benzodiazepines. No paralysing agents were used. Paracetamol was used occasionally in connection with body temperatures above 39 °C. Body temperature was measured in the axilla 16–24 times per day and body weight was determined daily using a bed scale (Datex 04 VM-104 A Datex OY, Helsinki, Finland). All patients were confined to bed and were studied at an ambient temperature of 21–23 °C. They were covered by a sheet and a blanket at body temperatures below 39 °C and by a sheet at temperatures above 39 °C.

Calculations

All weight-related parameters were calculated using the average body weight during the calorimetric study period. Weight determinations made in the presence of overt oedema were excluded. EE was related to body weight and was also expressed in percentage above the estimated basal metabolic rate according to Fleisch [12]. This is referred to as hypermetabolism in the text, Fig. 1 and Table 3. Energy balance (%) was calculated as 100× (EI-EE)/EE, where EI refers to the energy contents of administered nutrients, including amino acids, as specified by the manufacturs. The energy content of infused albumin and plasma was not taken into account.

Statistical methods

Data were analysed using the Wilcoxon two-sample test (Mann-Whitney), the Fisher exact probability test and the Chi-square test for several independent samples, when appropriate. Values are given as the means ± SD. In Tables 2, 3 and 4, the median values are given within parenthesis. A p-value < 0.05 was considered significant.

Results

All patients required TPN and artificial ventilation for at least 10 days. Demographic parameters, the durations of artificial ventilation, intensive care (IC), hospitalization

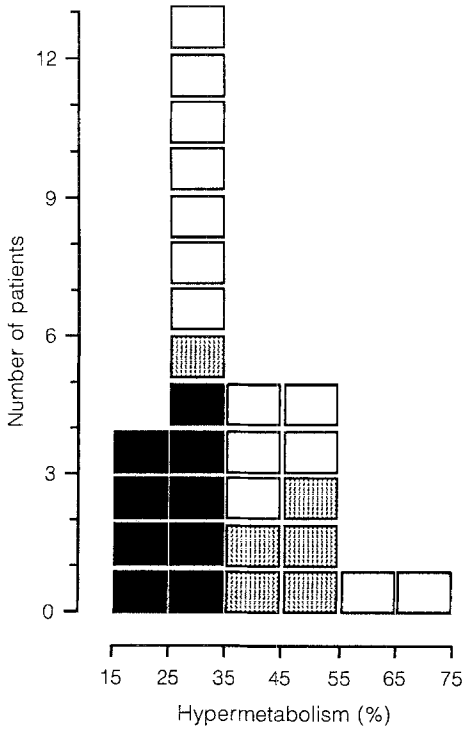

Fig. 1. Hypermetabolism (energy expenditure in percentage above estimated BMR according to Fleisch [12]). *Open squares* denote survivors of hospitalization ($n = 14$). *Solid squares* denote patients who died in the intensive care unit ($n = 9$). *Hatched squares* denote patients who died after intensive care, during subsequent hospitalization ($n = 6$)

and the calorimetric study period, and also the time lapse between the initiation of artificial ventilation and the beginning of the calorimetric study period, are presented in Table 2.

The average EE, hypermetabolism and RQ in all patients ($n = 29$) were 126 ± 19 kJ (30 ± 5 kcal)/kg/24 h, $36 \pm 12\%$ and 0.85 ± 0.03, respectively. Nine patients died in the ICU, and 20 patients survived the period of IC. Six of these patients died 20-85 days after IC during the subsequent hospitalization.

The period of intensive care

With regard to the average values for the whole period of EE measurements, survivors ($n = 20$) had a higher EE, whether related to body weight (132 ± 16 vs 113 ± 19 kJ/kg/24 h, $p < 0.01$) or expressed as hypermetabolism ($41 \pm 11\%$ vs $26 \pm 8\%$, $p < 0.01$, Fig. 1), than non-survivors ($n = 9$). The RQ was higher in survivors (0.86 ± 0.02 vs 0.83 ± 0.03, $p < 0.05$). The average energy supply was higher in survivors (148 ± 18 vs 129 ± 44 kJ/kg/24 h, $p < 0.05$), while energy balance did not differ ($p = 0.14$). The average supply of amino acid nitrogen was 49% higher ($p < 0.05$) in survivors (Table 3).

The average values for the first week of EE measurements revealed that survivors had a higher EE (134 ± 17 vs 114 ± 21 kJ/kg/24 h, $p < 0.05$) and hypermetabolism ($42 \pm 11\%$ vs $27 \pm 9\%$, $p < 0.01$) than non-survivors while the difference in RQ approached the level of significance ($p = 0.05$).

During the first day of EE measurements survivors displayed a higher EE (135 ± 23 vs 109 ± 21 kJ/kg/24 h, $p < 0.01$) and hypermetabolism ($43 \pm 15\%$ vs $21 \pm 12\%$, $p < 0.001$). However, there was no difference in RQ ($p = 0.20$).

The incidence of obesity, as defined by an average body weight exceeding the ideal body weight [13] by at least 15% (or a body mass index above 26), tended to be lower in survivors (2/20 vs 2/9). The differences in EE, hypermetabolism and RQ remained significant when data from obese patients were excluded ($p < 0.05$, $p < 0.01$ and $p < 0.01$ respectively).

Apache II scores were lower in survivors on the first day ($p < 0.05$) as well as on the last day ($p < 0.001$) of EE

Table 2. Demographic parameters and the durations of artificial ventilation, intensive care and hospitalization

	The period of intensive care		The period of hospitalization	
	Survivors $n = 20$	Non-survivors $n = 9$	Survivors $n = 14$	Non-survivors $n = 15$
Sex (male/female)	15/5	5/4	11/3	9/6
Age (years)	62 (66) ± 13	65 (69) ± 13	59 (61) ± 13	67 (67) ± 10
Height (cm)	174 (175) ± 9	171 (170) ± 8	174 (175) ± 9	171 (174) ± 9
Weight[a] (kg)	66 (69) ± 10	69 (72) ± 17	66 (69) ± 11	67 (71) ± 14
Weight loss[b] (%)	7 (6) ± 5	2 (0) ± 3**	6 (6) ± 4	5 (4) ± 5
Weight/ideal weight (%)[c]	101 (100) ± 16	109 (109) ± 21	100 (101) ± 14	106 (98) ± 20
Artificial ventilation (days)	32 (32) ± 15	17 (13) ± 8**	30 (28) ± 17	26 (26) ± 14
Intensive care (days)	40 (38) ± 18	17 (14) ± 7***	37 (34) ± 19	30 (26) ± 19
Hospitalization (days)	90 (78) ± 46	27 (26) ± 12***	80 (59) ± 45	62 (37) ± 52
Duration of indirect calorimetry (days)	15 (14) ± 10	8 (4) ± 8**	14 (13) ± 7	12 (8) ± 12
First day of the study in relation to beginning of artificial vent. (days)	8 (6) ± 8	7 (8) ± 7	8 (6) ± 9	7 (6) ± 7

Values are means (medians) ± SD

[a] Average body weight during the study

[b] Postoperative

[c] Postoperative weight in percentage of ideal body weight for medium-framed adults according to the Metropolitan Life Insurance Tables [13]. Asterisks ** and *** denote significance levels of < 0.01 and < 0.001, respectively, for differences between survivors and non-survivors within each period

measurements (Table 4). The incidences of acute renal failure and sustained septic shock tended to be lower in survivors (Table 1). Impairements of organ function, as indicated by average values during the study for arterial blood pressure, inspired oxygen concentration, requirements for cardiotropic therapy and the serum concentrations of haemoglobin, creatinine, urea and bilirubin, tended to be less pronounced in survivors. Significant differences were detected in the serum bilirubin, haemoglobin concentration and in the arterial blood pressure (Table 4). The average body temperature tended to be higher (0.6 °C, $p = 0.14$) in survivors (Table 4). Survivors had lost more weight post-operatively (7±5% vs 2±3%, $p < 0.01$, Table 2).

Post-mortem examinations were performed in 6 of the patients who died. The findings were consistent with MOF and no other specific cause of death could be established.

Table 3. Energy expenditure, respiratory quotient and nutritional data

	The period of intensive care		The period of hospitalization	
	Survivors $n = 20$	Non-survivors $n = 9$	Survivors $n = 14$	Non-survivors $n = 15$
Energy expenditure (kJ/kg/24 h)	132 (132)±16	113 (115)±19**	132 (129)±15	121 (117)±21
Hypermetabolism (%)[a]	41 (36)±11	26 (29)± 8**	39 (35)±11	34 (34)±13
Respiratory quotient	0.86 (0.86)±0.02	0.83 (0.82)±0.03*	0.86 (0.86)±0.03	0.84 (0.86)±0.03
Energy supply (kJ/kg/24 h)[b]	148 (141)±18	129 (132)±44*	149 (141)±18	136 (135)±36
Fat supply (% of non-protein energy)	51 (51)± 9	52 (53)± 5	53 (51)± 5	50 (52)±10
Energy balance (%)[c]	12 (13)± 8	12 (13)±18	13 (13)± 7	11 (14)±14
Amino acid supply (g N/kg/24 h)	0.19 (0.18)±0.05	0.12 (0.13)±0.06**	0.19 (0.19)±0.04	0.15 (0.14)±0.06

Values are means (medians) ± SD
[a] Measured energy expenditure in percentage above estimated basal metabolic rate according to Fleisch [12]
[b] Including amino acids
[c] Energy intake in percentage above measured energy expenditure
Asterisks * and ** denote significance levels of <0.05 and <0.01, respectively, for differences between survivors and non-survivors within each period

Table 4. Clinical data, drug therapy and laboratory data

	The period of intensive care		The period of hospitalization	
	Survivors $n = 20$	Non-survivors $n = 9$	Survivors $n = 14$	Non-survivors $n = 15$
Apache II, first day of measurements	15 (15)±5	23 (25)±7*	16 (15)±6	20 (20)± 8
Apache II, last day of measurements	11 (11)±5	26 (28)±8***	11 (11)±5	20 (20)±10*
Systolic arterial pressure (mmHg)	119 (121)±13	103 (96)±19*	121 (121)±12	108 (105)±18
Heart rate (bpm)	102 (101)±14	95 (95)±13	104 (101)±13	96 (95)±13
Inspired oxygen concentration (%)	33 (32)±6	38 (37)±6	32 (31)±4	37 (37)±7*
Axillary temperature (°C)	38.0 (38.0)±0.4	37.4 (36.9)±0.9	38.0 (38.0)±0.4	37.6 (37.7)±0.8
High-dose steroid therapy[a]	2	3	2	3
Dopamine therapy[b]	4,0,0	1,2,1	3,0,0	2,2,1
Sodium bicarbonate[c]	15,4,1	6,0,3	11,3,0	10,1,4
Insulin therapy[d]	1,1,2	3,0,3	1,0,2	3,1,3
Morphine equivalents (mg/kg/24 h)[e]	67 (24)±104	17 (20)±11	73 (24)±117	31 (20)±48
Diazepam (mg/24 h)	10 (7)±11	23 (3)±49	9 (5)±11	18 (8)±38
Base excess (mmol/litre)	0.5 (−0.5)±3.3	−1.2 (−1.2)±2.8	0.8 (−0.8)±3.1	−0.7 (−0.3)±3
Serum haemoglobin (g/litre)	109 (110)±8	101 (99)±18**	110 (111)±9	103 (102)±14**
Serum urea (mmol/l, normal <5)	19 (14)±12	32 (34)±16	19 (15)±11	27 (26)±17
Serum prothrombin (%, normal >50)	38 (36)±7	36 (36)±16	35 (36)±7	39 (41)±13
Serum bilirubin (μmol/l, normal <26)	47 (30)±51	107 (91)±57**	36 (24)±48	91 (72)±56***

Values are means (medians) ± SD or refer to the number of patients [a,b,c,d] unless otherwise indicated
[a] The number of patients who received a bolus dose of methylprednisolone 1–2 g prior to measurements
[b] The number of patients who received dopamine doses 1–2.9, 3–5 and >5 μg/kg/min, respectively, during measurements
[c] The number of patients who received average doses of sodium bicarbonate of 0–9, 10–49 and 50–70 mmol per 24 h, respectively, during measurements
[d] The number of patients who received average insulin doses of 10–29, 30–50 and >50 U per 24 h, respectively, during measurements
[e] The sum of administered doses of morphine and morphine analogues, assuming that 1 mg of morphine is equivalent to 10 mg of pethidine and 0.1 mg of phenoperidine
Asterisks*, ** and *** denote significance levels of <0.05, <0.01 and <0.001, respectively, for differences between survivors and non-survivors within each period

The period of hospitalization after IC

EE, hypermetabolism and RQ values did not differ significantly between survivors ($n = 14$) and non-survivors ($n = 6$) when data on patients who survived the period of IC were grouped according to the outcome of hospitalization after IC. Nor was there any discernible difference in the other parameters, except a lower bilirubin level in survivors ($p < 0.01$).

The entire period of hospitalization

EE, hypermetabolism and RQ values did not differ significantly between survivors ($n = 14$) and non-survivors ($n = 15$) when data for all patients were grouped according to the outcome of hospitalization. This was so regardless of whether data during the first day, first week or the whole period of EE measurements were analysed.

Survivors had a lower Apache II score ($p < 0.01$) on the last day of measurement, a lower inspired oxygen concentration ($p < 0.01$), a lower serum bilirubin concentration ($p < 0.001$) and a higher haemoglobin concentration ($p < 0.01$, Table 4).

Discussion

The results demonstrate that artificially ventilated patients with MOF following abdominal surgery have a moderate hypermetabolism. This is in agreement with previous findings using continuous indirect calorimetry in similar patients [1–3]. The higher EE in survivors of the IC period, as compared to that in patients who died in the ICU, is in line with previous results, suggesting an inverse relationship between EE and mortality [1, 8]. A similar relationship is also suggested by previous data in patients studied during IC [14, 15] and in patients with burn injuries [16]. However, in these reports it is not clearly stated whether analyses of the relationship between EE and outcome refer to the outcome of the period of IC or to the entire period of hospitalization.

In the present study no firm relationship was found between EE and the outcome of hospitalization after IC. When investigating the relationship between EE measured during MOF and the outcome of the pathophysiological state of MOF we find it important, at least in the present study, to make a distinction between death during IC and death during the hospitalization after IC. First of all, the state of MOF had been essentially resolved in the patients who were discharged from the ICU and, consequently, it cannot be ruled out that the non-survivors in this group died as a consequence of circumstances which had little or no connection with the MOF during IC (e.g. later complications, cancer growth or even suboptimal therapy). Secondly, all therapeutic and diagnostic interventions were in our hands only during the period of IC.

Nor was a relationship found between EE and the outcome of the entire period of hospitalization. This is in agreement with a previous study investigating the relationship between EE measured during IC and the outcome of the entire period of hospitalization [9].

A positive relationship between EE and body temperature exists in normal subjects, with an increase of 10–13% in EE for each 1 °C [17]. Assuming a similar relationship between EE and body temperature in our patients, a 6–7% higher EE in survivors of the period of IC would be expected in connection with the 0.6 °C higher average body temperature in these patients, compared to the actual difference in EE of 17%.

The greater post-operative weight loss in survivors may reflect differences in body composition. In conditions of trauma, sepsis [18] and renal failure [19] the total muscle water and, conceivably, also the total body water [20, 21] may increase despite an absence of clinical evidence for hyperhydration. Consequently, discrepancies in hydration may have contributed to the differences in EE and hypermetabolism since these parameters were related to body weight. In addition, other differences in body composition (e.g. the relative weight of fat, muscle, skeleton and parenchymatous tissues) may have also contributed to the differences in body-weight-related EE. Accordingly, the incidence of obesity tended to be higher in non-survivors than in survivors of the IC period. However, the differences in EE, hypermetabolism and RQ remained significant, when data for the obese patients were excluded.

The higher supply of amino acids in survivors could have influenced the results since the thermogenic effect of amino acids in depleted patients may amount to 30–40% of the energy content given [22]. However, in septic patients who received amounts of glucose and fat, similar to those in the present study and a slightly higher amino acid supply (i.e. 0.191 g amino acids N/kg/24 h), oxygen consumption increased by only 2% [23]. A pronounced thermogenic effect of amino acids has been suggested mainly to result from an amino acid supply in excess of what can be retained [23]. Skeletal muscle mass, a major recipient of amino acids, may be extensively reduced in patients with MOF [18]. Consequently, a conventional amino acid supply may be excessive when related to the size of muscle mass [24]. However, an assumption of a thermogenic effect as high as 40% [22] would explain no more than 15% of the observed difference in EE. On recalculating the current data accordingly, the difference in EE remained significant.

Differences in physical activity, level of consciousness and therapeutic interventions, such as airway suctioning and physical therapy, may have a bearing on our results inasmuch as sedative and analgesic therapy has been shown to decrease [6, 7] and physical therapy to increase EE [25]. However, the administration of sedative and analgesic drugs was similar in survivors and non-survivors and both groups were critically ill, confined to bed and sedated.

Consequently, the contribution of physical activity to EE was considered to be minimal. Furthermore, since the non-survivors were more ill, as reflected by their higher Apache II score, phyical therapy was probably more frequent in this group. It is therefore unlikely that these factors have markedly contributed to the difference in EE between the two groups.

The lower RQ in patients who died in the ICU cannot be firmly attributed to differences in nutrient supply. It may reflect a comparatively higher oxidation of lipids, which is in line with previous findings in patients with sepsis that the proportion of lipid oxidation increases with the severity of the disease [26].

Patients who died in the ICU tended to have a more pronounced MOF, as indicated by various indices, and their comparatively lower hypermetabolism could thus reflect a more extensive derangement of the oxidative metabolism. This notion is supported by a previous report of a relationship between the decrease in muscle and liver intracellular concentrations of energy-rich compounds, on the one hand, and the severity of critical illness, on the other [27]. A recent report suggests that inadequate tissue oxygenation due to insufficient peripheral oxygen delivery is associated with a fatal outcome in patients with MOF and that such a condition may be difficult to recognize [28]. In the current study the lower haemoglobin concentration and systolic blood pressure in the patients who died during IC may have contributed to such a state, and it cannot be ruled out that an inadequate peripheral oxygen delivery is partly reflected as a lower hypermetabolism in these patients. However, the degree of acidosis was similar in the two groups as reflected by base excess values and the sodium bicarbonate requirements.

It has been suggested that measurements of EE may be useful for assessing the prognosis of individual patients [1, 8]. The present results demonstrate an overlapping in hypermetabolism between survivors and non-survivors. Consequently, the prognostic value of this variable is limited in patients with MOF. However, in a patient with a very high or very low hypermetabolism, information relevant to the prognosis during IC may be available.

In summary, the results demonstrate that artificially ventilated patients with infection and MOF following abdominal surgery exhibit moderate hypermetabolism. In such patients a fatal outcome during IC seems to be associated with a comparatively lower hypermetabolism. This may reflect a more pronounced reduction of the metabollically active cell mass in relation to body weight, and/or an impairment of oxidative metabolism.

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