Epinephrine produces a prolonged elevation in metabolic rate in humans $^{1–3}$

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**ABSTRACT**

Background: Epinephrine increases the metabolic rate and contributes to the hypermetabolic state in severe illness.

Objective: We sought to determine the effect of prolonged elevation of epinephrine on resting energy expenditure (REE).

Design: Thirteen healthy men were placed on a well-defined diet for 5 d. Beginning on the morning of the second diet day, the subjects were infused for 24 h with saline, then for 23 h with epinephrine (0.18 nmol·kg$^{-1}$·min$^{-1}$) to increase plasma epinephrine concentrations into the high physiologic range (4720 ± 340 pmol/L). REE and the respiratory quotient (RQ) were measured by indirect calorimetry in the postabsorptive state at the same time every morning.

Results: Infusion of epinephrine significantly increased heart rate and systolic blood pressure, but the response was transient (values after 23 h of epinephrine infusion were not significantly different from those on the day saline was infused). Infusion of epinephrine significantly increased REE by 12% and increased the RQ. These changes were apparent at the end of the 23-h infusion (REE: 97.5 ± 2.3 kJ·kg$^{-1}$·d$^{-1}$ with saline infusion and 108.9 ± 2.3 kJ·kg$^{-1}$·d$^{-1}$ with epinephrine infusion; RQ: 0.832 ± 0.012 with saline infusion and 0.879 ± 0.013 with epinephrine infusion). REE returned to baseline by 24 h after the epinephrine infusion ended, but the postabsorptive RQ remained modestly elevated. Infusion of epinephrine also produced a transient increase in urine flow and in urinary nitrogen excretion. This diuresis was compensated for by a drop in urine volume and nitrogen excretion after the epinephrine infusion was stopped.

Conclusions: Epinephrine produced a prolonged increase in REE in healthy subjects. The fuel for this increase in REE, determined by the RQ, was from increased carbohydrate oxidation, not from that of fat or protein.

**KEY WORDS** Energy expenditure, oxygen consumption, heart rate, blood pressure, nitrogen excretion, epinephrine, respiratory quotient, hypermetabolic state, men

**INTRODUCTION**

The hypermetabolic state seen in stress, trauma, and sepsis is characterized by increases in energy expenditure and nitrogen excretion that lead to wasting of lean body mass if not corrected by nutritional intervention (1, 2). The plasma concentrations of the hormones cortisol, glucagon, epinephrine, and norepinephrine are elevated during a period of injury, leading to the hypothesis that one or more of these hormones produces the hypermetabolic state seen in injury (3–5). However, it has been difficult to reproduce the hypermetabolic response in humans by infusion of these hormones. The effects of altered hormone concentrations seen in severe illness have been simulated in healthy subjects by using combined infusions of epinephrine, cortisol, and glucagon, as well as the infusion of the individual hormones (6–9). These studies showed that both epinephrine and cortisol increase energy expenditure in a dose-dependent manner (8–11).

Elevated concentrations of catecholamines are also associated with the stress that occurs from normal, everyday life (12). We (10) and others (11) showed that small elevations in plasma epinephrine, which are frequently encountered in daily life (500–600 pmol/L measured from venous blood), do increase resting energy expenditure (REE) in healthy subjects (10, 11). However, these studies of the acute effects of epinephrine were of short duration and we are uncertain whether chronic stress and, therefore, chronically elevated epinephrine concentrations result in a chronic increase in REE. It is possible that epinephrine’s effect on the metabolic rate may diminish with time with chronic exposure.

When we infused epinephrine to increase plasma epinephrine into the high physiologic range for 8.5 h to simulate the stress condition, we found that the elevation in metabolic rate slowly declined with time at the higher physiologic concentrations of epinephrine (9). Under those conditions, we estimated that metabolic rate would have returned to normal if the epinephrine infusion had been continued for 18 h. Bessey et al (6) infused 3 subjects with epinephrine for 60 h and found that the metabolic rate remained elevated. However, these results were suggestive, not...
conclusive. The specific aim of the present study was to determine whether the epinephrine-induced increase in REE diminishes or persists with continued exposure to high concentrations of epinephrine.

SUBJECTS AND METHODS

Subjects

Thirteen healthy men with a normal weight-for-height (Table 1) were studied at the New York Hospital–Cornell University Medical Center (NYH-CUMC) Clinical Research Center (CRC). All subjects were screened for medical illness through a medical history, a physical examination, and biochemical screening tests. Subjects were instructed about the purpose, benefits, and risks of the study and gave their written consent in accordance with protocols approved by the NYH-CUMC Institutional Review Board and by the CRC Scientific Advisory Committee.

Protocol

Each subject was admitted to the CRC on a Monday afternoon (day –3, Figure 1) and remained in the CRC for 6 d. During this period, subjects consumed a standardized liquid formula (1.5 MJ/container, Ensure Plus; Ross Laboratories, Columbus, OH) calculated to meet each subject’s daily energy requirement [1.5 times the basal metabolic rate as calculated with the Harris-Benedict equation (13)]. The energy composition of the diet was 53.3% carbohydrate, 32.0% fat, and 14.7% protein. Meals were consumed at ~0930, 1230, and 1800. Throughout the study period, subjects were ambulatory, but were confined to the CRC. Subjects were ambulatory during the 2 infusion days, but because of the infusion tubing and electrocardiogram leads, subjects’ mobility was restrictive and activity was less.

Before 0900 on day –1, catheters were placed into an antecubital vein for continuous infusion of 0.45% saline and retrograde into a superficial hand vein for blood sampling. The catheters were kept patent with a slow infusion of 0.45% saline. Before each blood sample was obtained, the hand with the blood sampling catheter was placed in a warming box (heated air kept at 50–55°C) for 15 min before every blood draw to produce arterialized venous samples. Both catheters remained in place for 48 h. Saline (0.45%) was infused for 24 h at a rate of 40 mL/h from 1000 on day –1 until 1000 on day 0, at which time the saline infusion was stopped and an infusion of epinephrine was started. Epinephrine was infused at 0.18 nmol·kg⁻²·min⁻¹ (≈2 μg/min) in 0.45% saline (volume infusion rate: ≈40 mL/h) for 23 h until 0900 on day 1. During the epinephrine infusion, subjects were followed continuously by use of an electrocardiogram monitor and by regular measurements of blood pressure with a sphygmomanometer.

Measurements

Energy expenditure

Each morning, oxygen consumption and carbon dioxide production were measured for 30 min by indirect calorimetry between 0800 and 0900 before breakfast on days –2, –1, and 2 and for 50 min on days 0 and 1. The indirect calorimetry measurements were also made for 30 min between 1700 and 1730 in the afternoon on days –1, 0, and 1 (Figure 1). The measurements were conducted with a Deltatrac indirect calorimeter with a flow-through hood system (SensorMedics Corp, Yorba Linda, CA). REE and nonprotein respiratory quotient (RQ) were calculated by using standard equations (14) from the oxygen consumption, carbon dioxide production, and urinary nitrogen excretion measurements obtained for the same period. Values obtained throughout each measurement period were averaged to produce a single number per measurement session for both REE and RQ.

Lean body mass

Each subject was given an accurately weighed 5-g dose of ²H₂O to consume before his evening meal on day –2. Urine samples were collected before consumption of ²H₂O and again on the next morning (2nd morning void). A small aliquot of each urine sample was frozen at −20°C until analyzed for ²H enrichment by isotope ratio mass spectrometry. ²H₂O enrichment was determined in triplicate for each sample after reducing 1-μL aliquots of urine over a zinc catalyst to hydrogen gas. Total body water (TBW, kg) was calculated from the increase in ²H enrichment (in ppm) measured in the second urine sample (E) compared with the urine taken before administration of ²H₂O (E₀):

\[
TBW = 10 \times \frac{n_d}{E} (E - E_0) \times 18/20
\]

where \(n_d\) is the administered dose (in g), \(E_0\) is enrichment (99.9% ²H) of ²H₂O, and the factor 18/20 converts the weight of ²H₂O to the weight of unlabeled water. Lean body mass was computed as TBW divided by 0.73 (15).

### TABLE 1

**Physical characteristics of the subjects**

| Age (y) | 30.0 ± 1.2 |
| Height (cm) | 180 ± 2 |
| Weight (kg) | 78.1 ± 2.8 |
| Lean body mass (kg) | 63.1 ± 1.9 |
| BMR (kJ·kg⁻²·d⁻¹)² | 98.8 ± 1.3 |

²Basal metabolic rate determined by using the Harris-Benedict equation (13).

![FIGURE 1. Epinephrine infusion protocol. Subjects were confined to the Clinical Research Center from the evening (□) of day –3 to the morning (●) of day 2 and consumed 3 meals/d at the times shown. Indirect calorimetry measurements were performed each morning before breakfast and in the afternoon just before dinner. Saline was infused for 24 h starting at 1000 on day –1 and ended at 1000 on day 0 when an infusion of epinephrine was started for 23 h. Blood samples were obtained before meals as indicated, and urine was collected for measurement of total body water and for six 12-h periods from 0900 to 2100.](https://academic.oup.com/ajcn/article-abstract/68/5/1046/4648611)
TABLE 2

Time course of plasma catecholamine concentrations

<table>
<thead>
<tr>
<th>Day and time</th>
<th>Epinephrine (pmol/L)</th>
<th>Norepinephrine (nmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before saline infusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day −1, 0900</td>
<td>300 ± 40</td>
<td>2.1 ± 0.3</td>
</tr>
<tr>
<td>During saline infusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day −1, 1200</td>
<td>270 ± 40</td>
<td>2.4 ± 0.4</td>
</tr>
<tr>
<td>Day −1, 1800</td>
<td>370 ± 95</td>
<td>1.9 ± 0.3</td>
</tr>
<tr>
<td>Day 0, 0900</td>
<td>310 ± 50</td>
<td>1.6 ± 0.2</td>
</tr>
<tr>
<td>During epinephrine infusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 0, 1200</td>
<td>3655 ± 405</td>
<td>2.1 ± 0.3</td>
</tr>
<tr>
<td>Day 0, 1800</td>
<td>3725 ± 320</td>
<td>1.7 ± 0.2</td>
</tr>
<tr>
<td>Day 1, 0900</td>
<td>4720 ± 340</td>
<td>2.0 ± 0.2</td>
</tr>
<tr>
<td>After epinephrine infusion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day 2, 0900</td>
<td>395 ± 60</td>
<td>2.2 ± 0.4</td>
</tr>
</tbody>
</table>

$^a$ ± SE; n = 10. Plasma epinephrine was significantly different among time points, $P < 0.0001$ by repeated-measures ANOVA.

$^b$ Significantly different from other epinephrine values, $P < 0.05$ (Student-Neuman-Keuls test).

$^c$ Significantly different from other 2 values during epinephrine infusion, $P < 0.05$ (Student-Neuman-Keuls test).

### Urinary measurements

Urine was collected in 12-h intervals from 0900 on the morning of day −1 until 0900 on the morning of day 2 (Figure 1). After the urine volume was determined, each urine collection was diluted to a standard volume and an aliquot of urine was frozen and stored at −20°C for later analysis for creatinine and total nitrogen. Total plasma creatinine concentration was measured colorimetrically by using the Jaffé method with kits from Sigma Chemical (St Louis). Total nitrogen in urine was measured by using a chemiluminescent analyzer (Antek Instruments, Houston).

### Catecholamine measurements

Blood samples were obtained at 1800 and 2100 on day −1; at 0900, 1200, 1800, and 2100 on day 0; and at 0900 on day 1 during the days of saline and epinephrine infusions (Figure 1). For measurement of catecholamines, 2 mL blood was placed in a tube containing glutathione. Blood samples were placed on ice; plasma was centrifuged at a g force sufficient for separation of blood components promptly at 4°C for 15-20 min, and samples were frozen and stored at −60°C. Plasma epinephrine and norepinephrine were measured by using the single-isotope derivative method (16). Plasma samples were unavailable for measurement in 3 of the studies, and catecholamine results are reported for only 10 subjects.

### Data analysis

Data are presented as means ± SEs. Data were analyzed for significant differences among factors by using SAS (17) version 6.12 for WINDOWS and repeated-measures analysis of variance (ANOVA). Repeated-measures ANOVA was performed by using the general linear models procedure. Factors that showed significant differences were then tested for differences from a control value (data obtained on the day of saline infusion) by using Dunnett’s t test, which controls for type I experiment-wise error ($\alpha = 0.05$) for all comparisons. The Student-Neuman-Keuls test ($\alpha = 0.05$), which also controls for the experiment-wise error rate, was used for comparisons of plasma epinephrine concentrations.

### RESULTS

The time course of plasma catecholamine concentrations is presented in Table 2. The plasma norepinephrine concentration was at a normal, baseline value throughout the study period. The plasma epinephrine concentration was also at a normal value before and during the day of saline infusion. The plasma epinephrine concentration averaged 4030 ± 320 pmol/L over the period of epinephrine infusion (mean of 3 time points; Table 2). This plasma epinephrine concentration was similar to that which we observed previously with a 2-μg/min infusion of epinephrine for 8.5 h (9). There was a small but significant increase over the course of the 23-h infusion in plasma epinephrine concentrations. Epinephrine concentrations returned to baseline after the epinephrine infusion was discontinued, ie, the concentration measured after the infusion was not significantly different from that measured before the epinephrine infusion.

Heart rate and blood pressure were measured at the end of the saline infusion period (morning of day 0) and during the epinephrine infusion period (days 0–1). The mean heart rate on the morning of day 0 was 62 ± 2 beats/min. The heart rate increased significantly by ≈11 beats/min during the first 14 h of the epinephrine infusion, but the heart rate measured at the end of the epinephrine infusion period was not significantly greater than the baseline value (Figure 2). Blood pressure readings were nor-

![FIGURE 2](https://academic.oup.com/ajcn/article-abstract/68/5/1046/4648611/6348611)
mal on the morning of day 0 at the end of the saline infusion period (systolic: 116 ± 3 mm Hg; diastolic: 72 ± 2 mm Hg). As expected (18), infusion of epinephrine increased the systolic pressure and decreased the diastolic pressure (Figure 2). Although the systolic pressure remained elevated through the first 14 h of epinephrine infusion, the suppression of diastolic pressure was temporary and not significantly different from baseline diastolic pressure after 8 h of epinephrine infusion.

REE measured in the postabsorptive state at the end of the saline infusion period on day 0 was 97.5 ± 2.3 kJ · kg⁻¹ · d⁻¹. The measured REE was 99 ± 2% of the basal energy expenditure predicted by the Harris-Benedict equation and similar to that measured previously in healthy subjects (9). The postabsorptive REE values measured each morning throughout the study are presented as the difference from those on the day of the saline infusion (day 0) for each subject (Figure 3). There was no significant difference in REE on the 3 d before the epinephrine infusion. Infusion of epinephrine increased REE by 12% over the 23-h epinephrine infusion. Although REE appeared elevated on the day after the epinephrine infusion ended (day 2, Figure 3), the increase was not significant. RQ measured in the morning at the end of the saline infusion was 0.838 ± 0.015 and was typical of fasting subjects. Although there was an upward trend in RQ, the RQ was not significantly different on the 3 d before the epinephrine infusion (Figure 3). Infusion of epinephrine increased the RQ significantly, indicating a shift toward increased oxidation of carbohydrate and less oxidation of fat. This elevation in RQ persisted for 24 h after the epinephrine infusion ended.

Energy expenditure and RQ were also measured in the afternoon of days 0, 1, and 2 at 1700 before subjects received their evening meal. Because the noon meal had been consumed <4 h before the indirect calorimetry measurement, the afternoon measurement was postprandial, not postabsorptive. REE measured in the afternoon during the saline infusion was 118.3 ± 2.1 kJ · kg⁻¹ · d⁻¹, which was significantly greater (22%, P < 0.001) than the postabsorptive REE value at the end of the saline infusion. The postprandial, afternoon RQ on day −1 was 0.880 ± 0.017 and was significantly higher (P < 0.005) than the postabsorptive RQ, but it was not significantly different from the RQ that would have been calculated if the subjects had been oxidizing exclusively the antecedent meal (the RQ calculated if the diet were combusted, often referred to as the food quotient, was 0.879). This increase in RQ indicated an increase in the contribution of carbohydrate oxidation to energy expenditure. Although energy expenditure was already increased during the postprandial period (compared with the postabsorptive period), infusion of epinephrine increased the REE by an additional 11% (Figure 4). The postprandial increase in RQ also occurred with epinephrine infusion. Postprandial energy expenditure and RQ returned to values not significantly different from those on the
day of the saline infusion within 6 h after the epinephrine infusion ended on day 1.

Urine samples were collected in 12-h increments on the day of the saline infusion, the day of the epinephrine infusion, and the day after the epinephrine infusion. Urine was collected in 12-h intervals from 0900 until 2100 (■) and from 2100 until 0900 (□). Repeated-measures ANOVA showed significant differences ($P < 0.0001$) in urine volume with time for 12-h and pooled 24-h samples and significant differences ($P < 0.05$) in creatinine excretion for 12-h and pooled 24-h samples, but individual sample differences in creatinine excretion were not significantly different from those on the day of saline infusion. *Significantly different from corresponding saline value, $P < 0.05$. $n = 13$.

**FIGURE 5.** Mean (±SE) urine volume and urinary creatinine excretion on the day of saline infusion, the day of epinephrine infusion, and the day after epinephrine infusion. Urine was collected in 12-h intervals from 0900 until 2100 (■) and from 2100 until 0900 (□). Repeated-measures ANOVA showed significant differences ($P < 0.0001$) in urine volume with time for 12-h and pooled 24-h samples and significant differences ($P < 0.05$) in creatinine excretion for 12-h and pooled 24-h samples, but individual sample differences in creatinine excretion were not significantly different from those on the day of saline infusion. *Significantly different from corresponding saline value, $P < 0.05$. $n = 13$.

FIGURE 6. Mean (±SE) urinary total nitrogen (UTN) excretion on the day of saline infusion, the day of epinephrine infusion, and the day after epinephrine infusion. Urine was collected in 12-h intervals from 0900 until 2100 (■) and from 2100 until 0900 (□). Repeated-measures ANOVA showed significant differences with time for 12-h and pooled 24-h samples ($P < 0.0005$, top; $P < 0.01$, bottom). *Significantly different from morning saline collection, $P < 0.05$. **Significantly different from 24-h saline collection, $P < 0.05$. $n = 13$.

The increased urinary total nitrogen on the day of epinephrine infusion was essentially canceled out by the decreased urinary total nitrogen on the day after the epinephrine infusion. Thus, the effect of increased nitrogen excretion with epinephrine infusion was compensated by less excretion after the epinephrine infusion. This change in urinary nitrogen excretion may have been related to the diuresis caused by the epinephrine infusion. To compensate for diuresis, urinary nitrogen excretion was expressed against creatinine excretion. The increase in nitrogen excretion with epinephrine infusion was attenuated when expressed against creatinine.

**DISCUSSION**

We have known for several decades of the importance of catecholamines to the hypermetabolic state seen with trauma, burn injury, and stress (20). Because of the complexity of injury and trauma, defining the alteration, hormonal or otherwise, that produces the metabolic effect has been difficult (1, 2, 4). Much of our understanding of the regulation of the hypermetabolic state in humans has come from studies simulating the injured state by infusion of hormones into healthy humans (5–7, 9, 21). These studies have shown clearly that elevating plasma epinephrine produces a hypermetabolic state with respect to increased energy...
expenditure (5). In our prior study (9), epinephrine was infused for 8.5 h at different rates into healthy subjects. A dose-response increase in REE with increasing epinephrine infusion rate was observed, as reported previously by others after shorter periods of epinephrine infusion (10, 11). However, the time course of REE during the infusion of epinephrine at the higher rate (0.18 nmol·kg⁻¹·min⁻¹) showed a significant decline over the 8.5 h of the epinephrine infusion, suggesting that epinephrine’s effect on REE was possibly transient. We predicted that if the decline in REE with time were true, then REE would return to baseline values after 18 h of epinephrine infusion (9). The present study was designed to test the hypothesis that epinephrine’s effect on energy metabolism is transient.

Energy expenditure is dependent on the metabolic state; therefore, hours of fasting and time of day are variables that can influence metabolic rate (9). To control for variables such as time of day, the present study was designed to measure REE at exactly 24-h intervals. The administration order of the saline and epinephrine infusions was not randomized because we anticipated a residual effect of epinephrine on metabolism after cessation of the infusion. However, our design included measurement of REE on the day after the epinephrine infusion to define residual effects of epinephrine, and also included measurements on the 3 d preceding the day of the saline infusion to define a day-to-day trend, if it existed. We determined in our subjects who consumed a standardized diet over the 3 d before the epinephrine infusion day (Figure 3) that REE and RQ were stable and did not change with time.

The data in Figure 4 show that infusion of epinephrine at 0.18 nmol·kg⁻¹·min⁻¹ to increase blood epinephrine concentrations into the high physiologic range increased postabsorptive RQ by 12%. REE returned to a value not significantly different from that on the day of the saline infusion on the day after the epinephrine infusion ended. These results clearly showed that infusion of epinephrine for an extended period of time (23 h) produced an extended elevation in the metabolic rate, answering the question of whether epinephrine’s effect on metabolic rate wanes when the duration of infusion of epinephrine is prolonged (9).

In contrast with epinephrine’s extended effect on metabolic rate, the epinephrine-induced increases in heart rate and systolic blood pressure were not sustained and returned to baseline values during the epinephrine infusion (Figure 2). Thus, the increase in metabolic rate occurred independently of the changes in hemodynamics. These results indicate that epinephrine may increase energy metabolism without producing a concomitant long-term change in heart rate or blood pressure. Although the use of a heart rate monitor to define activity-related changes in metabolic rate may be valid, alterations in metabolic rate due to prolonged alterations in catecholamine activity could be missed with a heart rate monitor.

The increase in metabolic rate with epinephrine infusion was accompanied by a significant increase in RQ, which was sustained 24 h after discontinuation of the epinephrine infusion (Figure 3). Using standard equations to convert the RQ values to rates of oxidation of carbohydrate and fat (14), we estimated the source of the energy used to increase metabolic rate by infusion of epinephrine. The percentage of energy derived from protein was calculated from the urinary nitrogen excretion data. Protein oxidation contributed 15–18% of the energy expended. The fraction of energy derived from carbohydrate rose from 40 ± 4% on the day of saline infusion to 57 ± 4% at the end of the epinephrine infusion. Conversely, the fraction of energy derived from fat dropped from 42 ± 4% at the end of the saline infusion to 28 ± 4% at the end of the epinephrine infusion. The shift in fuel source was toward increased utilization of carbohydrate and decreased utilization of fat. This pattern of fuel usage was similar to the pattern of dietary intake and to the postprandial RQ during saline infusion.

Although studies have shown that epinephrine induces glycogenolysis (22, 23), epinephrine-induced glycogenolysis is considered acute and of lesser importance than is epinephrine’s stimulation of gluconeogenesis (22–27). This conclusion makes teleologic sense because glycogen stores are limited and useful primarily for short bursts of energy. Thus, the increase in postabsorptive RQ during epinephrine infusion implies that the carbohydrate being oxidized was formed primarily from gluconeogenesis.

Because gluconeogenesis from amino acids requires disposal of amino acid nitrogen, gluconeogenesis will produce an increase in urinary nitrogen excretion. However, we did not observe a significant increase in urinary nitrogen excretion at the end of the epinephrine infusion. In our previous study of amino acid kinetics, we found no effect of epinephrine on amino acid oxidation (9). One explanation is that the increase in gluconeogenesis was not sufficient to increase urinary nitrogen excretion above normal variance. This conclusion does not appear to be warranted. Conversion of glucose into fat (ie, lipogenesis) increases the RQ. Lipogenesis is not measured when substrate oxidation is calculated from the RQ. Rather, when lipogenesis occurs and produces an increase in the RQ, the increase in RQ is interpreted in the calculations of substrate oxidation as an increase in the oxidation of carbohydrate (28). Thus, it is possible that lipogenesis from glucose would explain part of the rise in the observed RQ.

During an acute infusion of epinephrine, the increase in RQ can be attributed to glucose oxidation derived from glycogenolysis, but glycogen stores (both hepatic and muscle) are limited. Glycogenolysis could not be the source of the increase in energy burned during the fasting part of the day unless glycogen supplies were repleted during the meal-feeding portion of the day. We measured RQ in the late afternoon on the day of the saline infusion and on the day of the epinephrine infusion. Consumption of a mixed meal usually increases both the RQ and RQ because carbohydrate is preferentially oxidized from the meal (29). As expected, RQ and RQ were both greater in the postprandial state on the saline infusion day, compared with the morning postabsorptive values. The epinephrine infusion increased RQ and RQ even more (Figure 3), indicating that the epinephrine stimulation of RQ and RQ was not abated or obscured by meal feeding. The increase in RQ after meal feeding indicated that most of the carbohydrate in the meal was being oxidized, but did not rule out that glycogen stores were also being repleted in the postprandial state during epinephrine infusion.

In summary, we showed that an increase in blood epinephrine concentrations produced a sustained increase in energy expenditure, indicating that catecholamine elevation affects energy expenditure in both hypermetabolic situations of stress and trauma as well as in conditions of ordinary daily stress. The source of fuel for this increase in energy expenditure in normally fed subjects appears to be carbohydrate, not protein or fat.

REFERENCES


