TITLE: CEREBRAL BLOOD FLOW AND CEREBRAL SWELLING IN FULMINANT HEPATIC FAILURE

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Introduction: Fulminant hepatic failure (FHF) is frequently complicated by hepatic encephalopathy, and may result in brain herniation which has a high mortality.[1] Reports on cerebral blood flow (CBF) in comatose patients with FHF have been contradictory: one study showed decreased CBF,[2] while others suggested increased CBF.[3] In this study, we reviewed CBF and degree of cerebral swelling in FHF patients with grade IV and V coma, and noted their outcome.

Method: Six patients (14-48 years) who developed FHF and were admitted to the ICU between October 1989 and February 1990 were reviewed. All patients were ventilated and monitored by complete hemodynamic profile. Hepatic coma was graded according to the patient's clinical condition.[4] CBF was determined by either the Xenon-133 or the stable Xenon/CT method. Cerebral swelling was determined by the loss of gray/white matter distinction and appearance of small ventricles and cisterns on CT images.

Results: FHF was caused by paracetamol overdose in one patient and by undetermined factors in five. Clinical, CBF and CT scan data are presented in Table 1. CBF was half normal in patients #1, 2, and 3 (normal CBF 50-60 ml/100 g/min at $pCO_2$ of 40 mm Hg), relatively high normal in patients #4 and #5 and very low in patient #6. On the basis of CBF and CT scan results patient #3 was given mannitol to reduce cerebral swelling and patients #4 and 5 were hyperventilated. Patients #4 and 5 were hemodynamically unstable and required norepinephrine to maintain systolic pressure around 80 mmHg. Patients #1, 2, and 3 recovered neurologically after liver transplantation. Patients #4 and 5 died before donor livers became available. Patient #6, who had EEG activity during surgery, showed flat EEG 12 hours after surgery and was pronounced brain dead.

Discussion: Our limited experience demonstrates that CBF and cerebral swelling increase as FHF worsens, although the patients remain at the same level of hepatic coma (grade IV). Relatively low CBF in patients with FHF appears to be an appropriate response of the brain to low $CMRO_2$ as indicated by no cerebral swelling and good outcome. High normal cerebral blood flow appears to be a luxury perfusion as indicated by marked cerebral swelling and terminal outcome. We suspect that as FHF progresses further (grade V coma), marked cerebral swelling is followed by minimal CBF as the brain nears herniation. Thus, because of the presence of variable CBF and cerebral swelling, monitoring of cerebral circulation and metabolism is essential for appropriate management.

References:

1. GUT 20:20-23, 1979
2. Scandinavian Journal of Gastroenterology, 24:299-300, 1989
3. Advances in ammonia metabolism and hepatic encephalopathy, 1989, pp 567-70
4. Sherlock DS: Hepatic Encephalopathy. Chapter 8. In Diseases of the liver and biliary system. Blackwell Scientific Publications, Oxford, 1985, pp 91-106

TABLE 1. CLINICAL COURSE OF SIX PATIENTS WITH FHF

| PATIENT # | 1 | 2 | 3 | 4 | 5 | 6 |
|---|---|---|---|---|---|---|
| ONSET OF ILLNESS TO COMA (DAYS) | 20 | 20 | 16 | 13 | 2 | 2 |
| COMA GRADE DURING CBF STUDY | 4 | 4 | 4 | 4 | 4 | 5 |
| CBF (ML/100 G/MIN) /$pCO_2$ (MMHG) | 22/36# | 26/36# | 20/36* | 50/24* | 60/24# | 4/30* |
| CEREBRAL SWELLING ON CT SCAN | NONE | NONE | MILD | MARKED | MARKED | MARKED |
| LIVER TRANSPLANTATION | YES | YES | YES | NO | NO | YES |
| OUTCOME | LIVED | LIVED | LIVED | DIED | DIED | DIED |

* CBF STUDY DONE BY STABLE XENON/CT METHOD
# CBF STUDY DONE BY XENON-133

A212

TITLE: VASOCONSTRICTION DURING GENERAL ANESTHESIA MINIMALLY DECREASES CUTANEOUS HEAT LOSS

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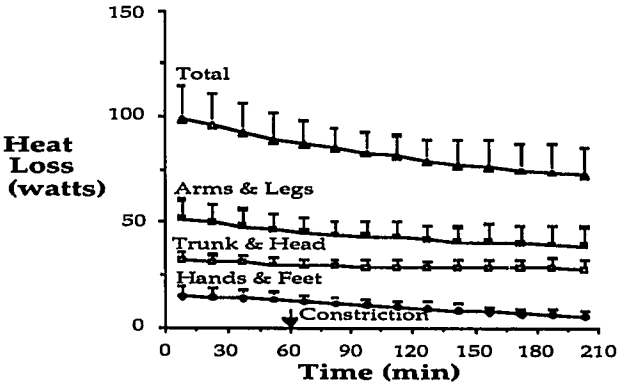
Thermal steady state (constant central temperature) is temporally associated with peripheral thermoregulatory vasoconstriction.[1] We tested the hypothesis that constriction during general anesthesia significantly decreases heat loss to the environment.

With approval of our IRB, we studied 4 minimally clothed volunteers in a 23.0 ± 1.0°C environment. Anesthesia was induced with isoflurane/$N_2O$ and maintained with isoflurane/air. Cutaneous heat loss (using thermal flux transducers) was measured from 10 area-weighted sites. Peripheral blood flow was evaluated using venous-occlusion volume plethysmography. Significant vasoconstriction was prospectively defined as a 10-fold decrease in fingertip blood flow. The preceeding 60 min were considered the pre-constriction control period.

Central temperatures at the time of vasoconstriction were 34.8 ± 0.3°C. Total heat flux, and flux from the arms & legs (upper arm, lower arm, thigh, and calf) decreased ≈ 16%. Heat loss from the trunk & head (head, back, chest, abdomen) decreased only ≈ 9%; in contrast, loss from the hands & feet decreased ≈ 38% (fig).

Overall heat loss was only minimally decreased by thermoregulatory vasoconstriction suggesting that thermal steady state results from other mechanisms (*i,e,*, nonshivering thermogenesis or redistribution of heat within the body).




**Legend:** Significant vasoconstriction occurred at 60 min. Regional heat losses indicated in the lower three curves add up to the Total Loss indicated at the top of the figure.

**Reference:** Støen R, Sessler DI. Anesthesiology, in press

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