TITLE: RISK FACTORS ASSOCIATED WITH POSTPARTUM BACKACHE IN OBSTETRIC PATIENTS

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Postpartum backache (PPB) is a minor but frequent problem after delivery. Despite the current opinion that epidural anesthesia does not increase the incidence of PPB, few prospective and comparative studies confirm this assumption. Furthermore, the respective role of other factors believed to be important in the development of PPB has never been systematically evaluated. The goal of this study was to prospectively evaluate, in a large obstetric population, supposed risk factors before delivery and then to observe the incidence of PPB. After Institutionnal Committée approval, we prospectively studied 1036 parturients who were seen for the first time at our outpatient clinic at 36 +/- 2 weeks of gestation and asked about previous episodes of backache. Demographic data (age, weight before pregnancy and weight gain, height, parity) were also recorded at this time. During labor or at delivery, parturients chose epidural anesthesia at their own request. Attending anesthesiologist was unaware of the goal of the study and perform epidural anesthesia as usual. During the first 3 days after delivery, the patients were asked to complete a questionnary about the development of PPB. The first step of the study was to compare the patients with (N = 336) or without (N = 803) PPB to determine the main factors associated with it. The second step of the study was to define the respective role of each factor in the subsequent development of PPB. Statistical analysis was performed using two-tailed t-test for unpaired data or chi-square analysis corrected for multiple comparisons as required. $p < 0.05$ was considered significant.

The 3 main risk factors ($p < 0.01$) associated with the subsequent development of PPB were: previous back pain, multiparity and epidural anesthesia. Epidural anesthesia does not significantly increase the incidence of PPB in primipara with or without previous back pain. However, epidural anesthesia increases the incidence of PPB in multipara with no previous history of back pain. Previous back pain was the strongest risk factor for PPB as neither multiparity nor epidural anesthesia increase the incidence of PPB in these patients We concluded that PPB could not be attributed to epidural anesthesia neither in primipara nor in patients with previous history of back pain.

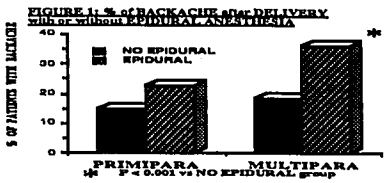

FIGURE 1: % of BACKACHE after DELIVERY with or without EPIDURAL ANESTHESIA

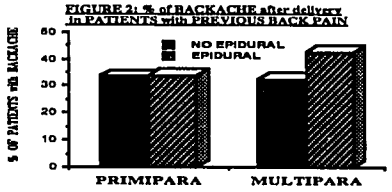

FIGURE 2: % of BACKACHE after delivery in PATIENTS with PREVIOUS BACK PAIN

TITLE: DECREASED METABOLIC RATE DOES NOT CAUSE HYPOTHERMIA DURING EPIDURAL ANESTHESIA

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Hypothermia during epidural anesthesia may result from increased environmental heat loss, decreased heat production, or redistribution of heat from central to peripheral tissues. We previously demonstrated that heat loss to the environment increases minimally.[1] In this study we tested the hypothesis that metabolic rate does not decrease during epidural anesthesia.

After approval from our IRB, tympanic membrane, skin temperatures (10 sites), and cutaneous heat loss (thermal flux transducers at 10 sites) were measured in 5 volunteers. Oxygen consumption was determined using a Deltatrac® monitor and converted to watts. After a 2-h control period in a 20°C environment, the epidural catheter was injected with 30-50 ml 3% chloroprocaine. Additional boluses were given at 10 min intervals to achieve sensory blockade to the T5 dermatome.

After 60 min of epidural blockade, tympanic temperature decreased 1.2°C, and average skin temperature increased 0.9°C; cutaneous heat loss increased only slightly. Oxygen consumption increased after epidural blockade (associated with shivering).

Metabolic heat production increased more than heat loss to the environment. We conclude that the observed central hypothermia can only be explained by redistribution of heat.

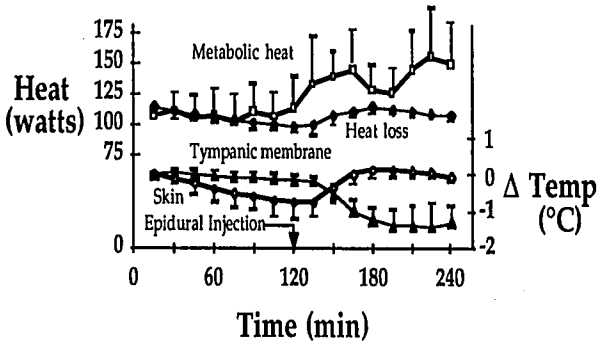

Fig. Metabolic heat production is calculated from oxygen consumption in ml/min assuming a caloric value of 4.82 kcal per ml oxygen. (One watt equals 0.86 kcal/h.)

References:

1. Sessler DI, Ponte J. Anesthesiology 71:A882, 1989.

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