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Effect of low temperatures on BAX and BCL2 proteins in rats with spinal cord ischemia reperfusion injury

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ABSTRACT. We evaluated changes in BAX and BCL2 expression levels after spinal cord ischemia/reperfusion injury (SCII) and hypothermia during operations in rats. Eighty rats were divided into four groups: Group A (N = 20, 18°C); Group B (N = 20, 28°C); Group C (N = 20, room temperature); and Group D (N = 20, sham operation control). Spinal cord ischemia was induced for 90 min. Hypothermia was induced 15 min before, and maintained during ischemia, followed by heating to normothermia for 30 min after reperfusion. Motor function of the lower limbs was evaluated according to the Tarlov score at 72 and 168 h. For each rat, spinal cord samples were taken at 6, 24, 72 h, and 1 week to evaluate the histopathological changes, neuronal

apoptosis, and BAX and BCL2 expression levels. Compared with normothermia, hypothermia significantly improved hind limb function; Group B achieved a higher score than Group A. Group D showed no neurologic deficiency, while the other groups showed various degrees. Group C exhibited greater neuronal apoptosis, higher BAX expression, but lower BCL2 expression than the other groups. Compared with Group A, BAX was expressed less and BCL2 more in Group B, and there was less apoptosis in Group B. Hypothermia preserves hind limb motor function and reduces neuronal death, thereby protecting rats from SCII. The spinal cord may be protected from SCII by inhibition of BAX and activation of BCL2. However, deep hypothermia may inhibit the expression of BCL2, resulting in a worse outcome than mild hypothermia.

Key words: Hypothermia; Spinal cord ischemia/reperfusion injury; BAX; BCL2

