NEUROSCIENCE 2006

SECTION B

Press Book Part 2

Lay Language Summaries

Section B: Summaries from
Monday, October 16 – Wednesday, October 18

SOCIETY FOR NEUROSCIENCE

36th Annual Meeting
October 14 – 18, 2006
Atlanta, GA

SOCIETY FOR NEUROSCIENCE

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36th ANNUAL MEETING

October 14–18, 2006

Information from lay-language summaries is under embargo until after the time of presentation.

For more information, contact:

October 14–18:	Press Room, Georgia World Congress Center, Room B203, (404) 222-5025/5026
After October 23:	Mark Bowman or Sara Harris, (202) 962-4000

Please contact speakers either in person or by phone before writing about their work. Kindly send copies of stories based on this meeting to the Society for Neuroscience, 1121 14th Street, NW, Suite 1010, Washington, DC 20005, to the attention of Mark Bowman. Thank you very much.

The material contained in parts I and II of the *Press Book* reflects the authors' research and represents the opinions neither of the Society for Neuroscience nor of its Officers or Councilors.

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MONDAY, OCTOBER 16 MORNING SESSIONS

WEDNESDAY, OCTOBER 18 MORNING SESSIONS

THE EFFECT OF IMMEDIATE HYPOTHERMIA THERAPY ON NEUROLOGICAL RECOVERY AFTER CARDIAC ARREST IN RATS

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Program Number: 681.17
Session Date/Time: Tuesday, Oct.17, 1:00 PM
Room Number: Georgia World Congress Center: Halls B3-B5
Board Number: NN49
Presentation Time: 1:00 - 2:00 PM

Brain injury is a very common cause of disability in survivors of cardiopulmonary resuscitation (CPR) for cardiac arrest. Cooling comatose survivors down to 32-34° Celsius for 12-24 hours after successful resuscitation from cardiac arrest, which is called therapeutic hypothermia, has been shown to improve brain recovery in human clinical trials. Because therapeutic hypothermia is new, it is not clear what the optimal target temperature, starting time, and duration of cooling should be to derive the most benefit from this therapy. In humans, cooling is often delayed by at least one to several hours after successful resuscitation.

We previously created an experimental model in rats with induced cardiac arrest followed by resuscitation and treatment with therapeutic hypothermia. Cooling is achieved by spraying mixed cold water and alcohol solution and blowing cold air by electric fan. Using this model, we compared the timing of cooling between immediate initiation of cooling against cooling delayed by one hour, which is more typical of current practices in humans. In order to compare the effects of the two cooling times, we used 3 different measures of brain recovery. One measure, called the Neurological Deficit Score (NDS) is a series of tests for brain function that includes arousal, leg strength, presence of seizures, and reflex movements. In a second measure, we compared the recovery of electrical brain activity using electroencephalography (EEG). In our previous experiments, we showed that recovery of electrical brain activity by EEG after cardiac arrest is predictive of recovery of brain function later and that new computer-based techniques to enhance the interpretation of EEG can predict brain recovery.

We also observed that brain injury leads to EEG patterns that are simpler and more predictable and with brain recovery the EEG pattern typically becomes less predictable and more complex and chaotic. Based the idea that brain injury results in a reduction in information content of the EEG signal, we quantified the information contained in the entire EEG and the average of the EEG subcomponents: delta, theta, alpha, beta and gamma in terms of the Shannon entropy. In the third measure, we examined the rat brains under a microscope, in order to count the percentage of brain cells that were injured or dying due to cardiac arrest.

In the present experiment, we studied 4 groups of 8 rats, based on duration - either 7 or 9 minutes of cardiac arrest - and starting time for cooling. Half of the animals in the 7- and 9-minute cardiac arrest groups were cooled to 32-34° Celsius immediately after resuscitation and maintained at this temperature for 6 hours and the other half were cooled to the same target temperature after a delay of 1 hour and maintained at this temperature for 12 hours. During the experiment, we monitored and controlled temperature, blood pressure, heart rate, and oxygen exchange. After the experiment, we monitored brain recovery for 3 days using the EEG measure of recovery and serial examination of the NDS, as described above. We found that rats treated with immediate but shorter hypothermia had significantly better functional outcome, faster improvement in EEG recovery, and less brain cell

injury than those with later onset of therapy in the other group, despite a longer cooling duration. This improvement was especially seen in the animals subjected to more severe injury, the 9-minute cardiac arrest group.

The results of this experiment are important for 2 reasons. First, this experiment further validates using EEG recovery as an early marker for brain recovery. This technology is also being tested in human patients. If the same relationship is seen in humans, changes in the predictability of EEG patterns after cardiac arrest may be helpful as a real-time monitor of recovery that can be used to select patients for aggressive treatment and tailor therapies such as hypothermia. Second, the experiment lends further support to the theory that cooling should begin as soon as possible after CPR. If these experimental results are reproduced in human clinical trials, it might justify changing resuscitation strategies such that paramedics begin cooling in the field or ambulance en route to the hospital.