SECTION B

Press Book Part 2
Lay Language Summaries

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

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

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October 14–18: Press Room, Georgia World Congress Center, Room B203, (404) 222-5025/5026
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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.
MONDAY, OCTOBER 16 MORNING SESSIONS

SYMPOSIUM: PERIPHERAL BASES FOR NEUROPATHIC PAIN: CUTANEOUS MECHANISMS FOR ACUTE AND CHRONIC NEUROPATHIC PAIN IN HUMANS, NONHUMAN PRIMATES AND RODENTS. F. Rice

SYMPOSIUM: CYTOKINE PROFILES IN ANIMAL MODELS AND IN HUMANS WITH CHRONIC PAIN. C. Sommer

SYMPOSIUM: SYNAPTIC EFFECTS OF ESTROGEN IN HIPPOCAMPUS AND PREFRONTAL CORTEX: IMPLICATIONS FOR COGNITIVE AGING. J. Morrison

SYMPOSIUM: THERAPEUTIC RNA INTERFERENCE FOR INHERITED DYSTONIA. P. Gonzalez-Alegre

MINISYMPOSIUM: DIVERSE FUNCTIONS OF NEURONAL ENDOSONES: FROM SYNAPTIC PLASTICITY TO PATHFINDING. B. Winckler

MINISYMPOSIUM: ERM PROTEINS REGULATE RESPONSE AND ADAPTATION TO SEMA3A. D. Benson

MINISYMPOSIUM: ENDOCYTOSIS AND AXONAL RETROGRADE TRANSPORT OF TETANUS TOXIN IN MOTOR NEURONS. G. Schiavo

MINISYMPOSIUM: SYNAPTIC SELECTION: A MODEL FOR SPECIFICITY UTILIZING INTRACELLULAR TRAFFICKING OF RECOGNITION MOLECULES. G. Phillips

MINISYMPOSIUM: ROLE OF EXOCYTOSIS IN NEURONAL MORPHOGENESIS. T. Galli

MINISYMPOSIUM: ENDOCYTOSAL TRAFFICKING OF AMPA-TYPE GLUTAMATE RECEPTORS. H. Hirling

MINISYMPOSIUM: NEUROIMAGING OF FALSE MEMORY. E. A. Kensinger

MINISYMPOSIUM: COGNITIVE NEUROSCIENCE INVESTIGATIONS OF FALSE MEMORY. B. Gonsalves

MINISYMPOSIUM: EXAMINING THE NEURAL BASES OF THE MISINFORMATION EFFECT. C. Stark

MINISYMPOSIUM: RECAPITULATION AND FALSE RECOLLECTION: MULTIMODAL IMAGING OF CORTICAL CONTRIBUTIONS TO FALSE MEMORY. I. Kahn

MINISYMPOSIUM: THE INFLUENCE OF EMOTION ON MEMORY DISTORTION. E. Kensinger

MINISYMPOSIUM: THE NEURAL BASIS OF AGE-RELATED CHANGES IN MEMORY DISTORTION. K. Giovanello

MINISYMPOSIUM: BDNF MODULATES DISEASE PROGRESSION IN A MOUSE MODEL OF RETT SYNDROME. Q. Chang

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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°C 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°C 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.