Kellogg School of Management at Northwestern University, the New Marketplace has already held its first major webcast meeting, portions of which can be viewed at the NEJM Catalyst website (catalyst.nejm.org).

The "Care Redesign" theme focuses on how to create and sustain the teamwork needed to provide high-value care, as well as the practical implications of organizing care to enhance health rather than simply provide sick care. Led by Amy Compton-Phillips, chief clinical officer at Providence Health Services, this theme has also already organized its first major meeting, covering topics such as team care for 21st-century medicine, the addressing of social needs in routine care, and bundled payments for chronic disease. Portions of that meeting are also viewable at catalyst.nejm.org.

In February, we will launch the third theme, "Patient Engagement: Behavioral Strategies for Better Health." Led by Kevin Volpp, a physician and behavioral economist from the University of Pennsylvania, this theme will focus on what is known about the use of incentives (financial and nonfinancial) to engage patients in improving their own health. A free webcast meeting will be held on February 25, 2016; information on how to register will be available shortly at catalyst.nejm.org.

The fourth theme will be "Leadership," led by Stephen Swensen, medical director for leadership and organization development at the Mayo Clinic College of Medicine. This theme will focus on the strategies and tools for engaging clinicians in enhancing the value of care and will be launched in late spring 2016.

The NEJM Catalyst Insights Council provides another avenue to draw on the expertise of clinicians, clinician leaders, and health care executives from across the country. The NEJM Catalyst Insights Council will select and regularly survey qualified executives, clinician leaders, and clinicians to contribute their perspectives and practical guidance on trends and issues in health care today. Survey results will be summarized and interpreted by NEJM Catalyst contributors and will be available at catalyst.nejm.org.

Like much in health care delivery today, NEJM Catalyst represents a new type of work, aimed at problems that are new to our times. We don't pretend to have a complete understanding of the best ways to accomplish this work, but we believe that bringing recognized experts together will give us the building blocks. We look forward to your input, and we hope that NEJM Catalyst will play a valuable role in helping the health care community create a higher-value health care system.

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Getting Warmer on Critical Care for Head Injury

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Unlike the successes for almost every other organ, resuscitation of the brain has been a fitful pursuit, punctuated by enthusiasm in early trials and subsequent disappointment. Even hypothermia after cardiac arrest, a seeming success, was tempered by a study in the *Journal* showing that cooling to either 33°C or 36°C gives the same results.[1] One strong point in the field had been the treatment of raised intracranial pressure, but trials involving patients with severe head trauma that were also published in the *Journal* have suggested that removing part of the skull to reduce intracranial pressure is ineffective[2] and that in resource-restricted countries a treatment strategy based on clinical and imaging findings is as good as treatment based on more elaborate direct monitoring of intracranial pressure.[3] The results of the Eurotherm3235 Trial, now reported in the *Journal*, may lead to the demise of hypothermia as yet another pillar of therapy for intracranial hypertension.[4]

In the new study, Andrews and colleagues

compared cooling with conventional measures (e.g., hyperosmolar therapy) for the treatment of severe closed head injury and found that the two approaches were equivalent in their reduction of intracranial pressure but that the group undergoing cooling had higher mortality and poorer functional outcomes. The ostensible purpose of using hypothermia was its fairly dependable effect in reducing intracranial pressure, and it has been thought to have an additional neuroprotective effect. Interpretation of these disappointing results is, as usual, complicated.

The above-mentioned Decompressive Craniectomy (DECRA) trial[2] and Benchmark Evidence from South American Trials: Treatment of Intracranial Pressure (BEST:TRIP) trial[3] and the current one are easily criticized but difficult to perform. All were pragmatic trials meant to provide practice guidance, but they involved critically ill patients with complex management problems that make it challenging to isolate the effect of a single treatment. When the results are unexpected, as they were in the hypothermia trial, detailed information regarding other treatments that were used to control intracranial pressure are required. For example, in the hypothermia group of the study, hyperosmolar infusions were implemented only if cooling failed to control intracranial pressure, but we do not have details pertaining to the manner and quantity of mannitol and hypertonic saline that were used. Furthermore, hypothermia resulted in a greatly decreased need for pentobarbital-induced coma. This may indicate that barbiturates provided the same or better metabolic suppression and neuroprotection as compared with hypothermia. For these reasons, it would be difficult to appreciate an effect of hypothermia alone on outcome.

The essential questions with respect to critical care that are raised by all these trials are whether control of intracranial pressure is still a key element for improving outcome after severe traumatic brain injury and what the threshold should be for initiating medical and surgical treatments. Is it necessary to maintain intracranial pressure below 20 mm Hg, as many guidelines suggest, even though cerebral perfusion is not compromised until it reaches much higher levels? All the aforementioned trials have been faulted because they initiated the active treatment when there was relatively mild and brief intracranial hypertension. Would the benefit have been more favorable for hypothermia in the Eurotherm3235 Trial if only patients who did not have an adequate response to what the authors term "stage 2" treatments (e.g., hyperosmolar therapy) had been enrolled?

Greatly elevated intracranial pressure is fatal, but our ability to control it, as noted in a previous editorial,[5] may be limited. The effects on mortality and functional outcome of all the treatments we currently use — hypothermia, craniectomy, drainage of cerebrospinal fluid, hyperventilation, and even hyperosmolar therapy — are probably smaller than we think, because severe head injury is an overwhelming and complex process involving neurons, glia, and vasculature. It may be that raised intracranial pressure compresses cerebral veins and creates a self-regenerating cycle of even higher pressure so that medical management becomes futile. This is not an excuse to allow elevated intracranial pressure to go unchecked, but it is an admonition to be discerning in the use of medical and surgical treatment, especially hypothermia. Given the diversity of protocols and treatments for severe head injury in different centers (e.g., the limited use of hypothermia in the United States as compared with Europe), the study by Andrews and colleagues is useful when interpreted with its limitations. Completely new approaches are needed for the control of intracranial pressure in order to design trials and treatments for severe traumatic brain injury.

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