CORRESPONDENCE

Cognitive Deficits Following Intensive Care

by Joel Kohler, Dr. med. Friedrich Borchers, Prof. Dr. med. Matthias Endres, Dr. med. Björn Weiss, Prof. Dr. med. Claudia Spies, and Dr. med. Julius Valentin Emmrich, MPhil in issue 38/2019

Positive Experiences With Piracetam

Cognitive deficits after severe illnesses used to be called “unspecified organic or symptomatic mental disorder”. Cognitive deficits after intensive care are included in this definition. In my experience, an older but still very effective medication is piracetam. It facilitates the provision of oxygen and glucose to the brain cells. It can be administered orally (800–1200 mg) in the morning or at midday, or, initially, as an infusion (12 g) in the morning. If administered after 3 pm, it may cause disrupted sleep; (initial) overdosing may cause restless-

ness. Older women should be advised that piracetam may increase the libido—something that is often unwanted.

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Vitamin B1 Deficiency Should not Be Forgotten

The authors write that delirium in particular is a relevant risk factor for the development of cognitive deficits after intensive care, and that managing the delirium is therefore of great importance for preventing such deficits, which are often permanent (1).

We know that alcohol consumption and especially alcohol dependency increases the risk for many diseases. We know that people with alcohol dependency/addiction have to be treated more often in hospital and in intensive care. We know that the prevalence of alcohol-related disorders in hospital patients is far higher than in the general population.

But not only alcohol-related illness but also other states with nutritional deficiencies/malnutrition (for example, hyperemesis gravidarum) can result in thiamine deficiency, and the need for thiamine is increased in many patients treated in intensive care and also when glucose infusions are required. According to the DGEM guideline on clinical nutrition in intensive care medicine, patients with indications of an alcohol-related disorder or thiamine deficiency should be treated with vitamin B₁ from day one.

As a medical consultant in a geriatric acute and rehabilitation hospital in Berlin, I frequently see patients with severe cognitive deficits, who were transferred after treatment in intensive care. Often it is immediately clear from the third-party medical history or the physical and neurological examination results that such patients have a pre-existing alcohol-related disorder. Often clear symptoms—such as eye movement disorders or ataxias—indicate that the patient has had Wernicke encephalopathy, but this is not documented in the referral notes. Unfortunately in many of these cases, no vitamin B₁ was given. Severe cognitive disorders are unfortunately often the consequence.

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In Reply:

We thank our correspondents for their interest in our work and for their responses.

Our article presented a systematic review of studies available to date on long-term cognitive dysfunction after conservative intensive care treatment. It explains the prevalence, disease course, and the risk factors studied so far and pharmacological and non-pharmacological interventions, and discussed them on the basis of the available data (1).

We are grateful for the suggestion of possible pharmacotherapy using piracetam. The long-term sequelae of a critical illness (often months to years) that were the focus of our article are, however, not congruent with the (controversial) term “Durchgangssyndrom” (organic psychosis which is understood to last a few days) (2). The effects of treatment with piracetam have so far not been investigated in studies of the long-term sequelae of conservative intensive care treatment.

We thank our correspondent for mentioning possible pharmacological treatment using vitamin B₁. Alcoholism and associated syndromes and disorders—such as Wernicke encephalopathy or Wernicke-Korsakov syndrome—play an important part in intensive care medicine (3, 4). Studies thus far have, however, not been able to establish any association with the long-term cognitive effects of intensive care treatment for the group of patients with thiamine deficiency or insufficient treatment with thiamine.

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On behalf of the authors

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Conflict of interest statement

The authors of all contributions declare that no conflict of interest exists.

Erratum

The legend of *Figure 3* in the article “The Treatment of Proximal Humerus Fracture Using Internal Fixation With Fixed-angle Plates—A Randomized Controlled Study Comparing Carbon Fiber Reinforced Polyetheretherketone With Titanium by Patrick Ziegler and coauthors in issue 45/2019, contains an error. The following shows the figure with its correct legend (corrected part in *italics*):

Functional outcomes of the CFR-PEEK group and the titanium group at 6 weeks, 12 weeks, and 6 months after surgery, measured using (A) the DASH score (0 best score, 100 poorest score), (B) the Simple Shoulder Test (SST; 0 poorest score, 100 best score), and (C) the Oxford Shoulder Score (OSS; 0 = poorest score, 48 = best score). *The rectangles (boxes) are delimited by the first and third quartile. The length of the boxes represents the interquartile range. The continuous line in the middle of the boxes represents the median. The maximum length of the whiskers (error bars) above and below the boxes represents maximally 1.5 times the interquartile range. The error bars do not end exactly according to the length of the interquartile range but at the value that still lies within these limits. Outliers were excluded.*

MWR

CRF-PEEK, „carbon fiber reinforced polyetheretherketone“
DASH, „disability of arm, shoulder and hand“; Ti, titanium

FIGURE 3