

“mandates” that turned out to have defective science behind them.

Dr. Wears goes a long way toward explaining why patients are still getting opiates for migraine headaches, antibiotics for viral illnesses, and unnecessary imaging studies. A crucial factor is staff time. Patient education takes time, and conflict with patients takes a lot more time. A busy emergency physician or nurse has very limited time, and until we figure out how to do the patient education ahead of time, we will bump up against the constant need to balance the time needed to do things the old way and the time needed to talk the patient/family/tech/private doc into doing things a better way. And so we had better be sure it really is a better way, and the battles must be fought selectively.

The accompanying paper, on changing from nebulizers to spacers for treatment of children with asthma in the ED, is a perfect example. It turns out that Hospital A eventually achieved this change by having full-time respiratory therapy coverage in the ED. The cost analysis was confined to the equipment, with no mention of the hospital’s cost for the extra personnel. The thrust of the article was that maybe by taking into account all the various reasons why Hospital B didn’t make the change, they laboriously could succeed in doing it for equivalent results. Nobody asks whether this is a fight worth fighting in an environment where the timely treatment of heart attack victims is getting worse, not better, in our overburdened EDs.

I’m all for evidence-based medicine, but I’d like to see a healthy skepticism toward the latest sure-fire scientific advances, and a careful selection of change efforts emphasizing those that actually improve patient outcomes without demanding time emergency physicians and nurses don’t have.

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*In reply:*

The Working Group doth protest too much, methinks<sup>1</sup> and the volume of their response confirms that academics who criticize the academy put themselves at risk.<sup>2</sup> However, I fear they have missed the main point.

The motivation for my comments was not to oppose scientific progress, or to yearn for a lost paternalism, or any of the other several assertions made, but rather a fairly simple observation: that knowledge translation seems to be more push

than pull, more the research community delivering its product than practitioners seeking it. As a social phenomenon, knowledge translation has been an activity of academics, not practitioners. One might think of it as a marketing effort for a product that we all agree has not been as successful in the marketplace as we had hoped. This situation suggests one (or more) of 3 possibilities: either the customers (clinicians in practice) have not heard our message; or, they have heard the message but have not been able to act on it due to a variety of barriers; or, the product isn’t very good—from the viewpoint of the customer and only the customer. Drs. Lang et al correctly point out that some very good work has been done addressing the first 2 possibilities<sup>3,4</sup> but the third issue has not been explored, because the product is assumed to be good if it meets the researchers’ standards. I think it would be more useful to address the third question from the viewpoint of the customer, and to do that we need to explore the differences between their perspectives. Dr O’Shaughnessy’s response plaintively addresses this gap between points of view.

The reason for the gap is that practitioners and researchers approach the same topic from different philosophical and scientific paradigms.<sup>5</sup> The dominant paradigm<sup>5</sup> of scientific activity held by researchers stems from positivism, and it clashes with the uncertain, pragmatic realities of clinical activity. The positivist perspective has led the research community to be devoted not to the production and distribution of fundamental knowledge in general, but rather to a particular view of knowledge that fosters selective inattention to practical competence and knowledge-in-action. Unfortunately, these types of practical but difficult-to-articulate activities are essential to professional practice, but are puzzling anomalies that do not fit the positivist paradigm, and thus are discounted if not discarded.<sup>6</sup> The problem is exacerbated by the tendency of the practice community to express itself in terms of intuition or “the art of medicine,” terms which tend to close off rather than open up discussion; and exacerbated again by the tendency of post-structural, non-positivist philosophies of science to stress the proliferation of meanings, the breaking down of existing hierarchies, the shortcomings of logic, and the failures of analytical approaches—elements that may strike modernist scientists as subversive or even destructive.<sup>7</sup> This is unfortunate, because a thoughtful discussion of the appropriate roles and potential values of the positivist versus the interpretivist paradigms in clinical work is, in my opinion, one of the most important intellectual issues in medicine today.<sup>8</sup> A post-structural research paradigm might narrow the gap in perspectives by leading to a philosophy of science focused more on practical results and their implications, grounded in the clinical workplace, and less on the generation of abstract meta-narratives serving to establish legitimacy.<sup>7</sup> That is, to research directed less at producing universal, abstract, conceptual truths, and more at local, timely knowledge of particular, concrete situations that can serve as reasonable guides for action for Dr O’Shaughnessy and her colleagues.<sup>2</sup>

It would seem important to begin a dialogue on how or whether shifting our paradigm might help both academics and practitioners. Such a discussion obviously cannot be held within the existing paradigm, but given sufficient openness to new thinking, it could help illuminate when and where new (or old) approaches are either useful or limited.

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## Response to "Use of Lipid Emulsion in the Resuscitation of a Patient With Prolonged Cardiovascular Collapse After Overdose of Bupropion and Lamotrigine"

*To the Editor:*

We read with interest the report by Sirianni et al of the use of lipid emulsion in the resuscitation of cardiovascular collapse.<sup>1</sup> However, we are concerned that readers may get the impression that the successful use of intravenous lipid emulsion in a patient with prolonged cardiac collapse from sodium channel blockade occurred only after the patient had received adequate boluses of sodium bicarbonate.

In the case presented by the authors there was clear ECG evidence of sodium channel blockade (QRS 0.122 and a

prominent terminal R wave in augmented ventricular lead right) in a patient that has a total of 3 cardiac arrests with a wide complex rhythm. During the first cardiac arrest, the patient appeared to respond to an intravenous bolus of sodium bicarbonate (50mEq) after a number of other options had been tried. Following return of circulation they report a QRS of 152 msec, well beyond the upper limit of normal. This would be a clear indication for further treatment with boluses of sodium bicarbonate in a poisoning suspected to be due to fast sodium channel blockade. During the second cardiac arrest it is not clear from the figure or text in the report at which point the NaHCO<sub>3</sub> boluses were administered, particularly in relation to the administration of intralipid. It is therefore difficult to determine what contribution bicarbonate and intralipid made to the return of circulation. We are concerned that the authors attribute the return of circulation to lipid emulsion and it may in fact be due to bicarbonate therapy. Despite this, the patient receives only 150 mEq of sodium bicarbonate (2.7mEq/kg) in total and remains acidotic (pH 7.196) with a low plasma bicarbonate (12.2mEq/L). It would appear that this patient may not have received sufficient amounts of sodium bicarbonate before being administered a dose of lipid emulsion.

While lipid emulsion may be familiar to intensive care unit staff, it is unlikely to be well known in the emergency department where these events are likely to occur. We would caution clinicians against the use of lipid emulsion as a first line agent in this situation and that fast sodium channel toxicity should be treated with boluses of sodium bicarbonate in a dose of 1–2 mEq per kg. In the context of cardiac arrest due to such agents, NaHCO<sub>3</sub> should be given early and titrated toward a high normal serum pH of 7.55.<sup>2,3</sup> Although most of the evidence for this treatment comes from tricyclic antidepressant overdose, it would seem reasonable to extrapolate this to other poisonings where the mechanism of toxicity also appears to be quinidine-like sodium channel blockade.

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