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In response to manuscript: Stamyr K, Thelander G, Ernstgard L, Ahlner J, Johanson G. Swedish forensic data 1992–2009 suggest hydrogen cyanide as an important cause of death in fire victims. *Inhal Toxicol.* Feb 2012;24(3):194–199.

We read with interest the manuscript by Stamyer et al. which describes the cyanide levels of deceased patients exposed to structural fires [1]. This unique paper is important and adds more scientific support to what many clinicians feel is true, that cyanide is a common cause of toxicity and death after smoke inhalation. For many reasons, detection of cyanide toxicity is difficult, including accessibility of obtaining cyanide levels, ambiguity of cyanide related symptoms, and other reasons described by authors.

However, we felt two questions require clarification by the authors. The authors report that a spectrophotometric method was used to detect cyanide levels prior to 2002. However, many patients in Europe received hydroxocobalamin prehospital and in the hospital for presumed cyanide toxicity [2]. Hydroxocobalamin has light absorption characteristics that interfere with colorimetric and spectrophotometric laboratory measurements [3]. As examples, artificial elevations in creatinine, glucose, and bilirubin have been reported, as have decrease alanine aminotransferase, and unpredictable results for creatine phosphokinase, phosphate, and lactate dehydrogenase [4]. Urinalyses are often uninterruptable [3]. Thus, we are concerned that deceased patients in the database may have received hydroxocobalamin before death that could have caused spectrophotometric interference.

They authors did not state if hydroxocobalamin interferes with their spectrophotometric cyanide assay used for detection before 2002.

Secondly, hydroxocobalamin binds cyanide well. In particular, investigators have reported that once this antidote is infused in sufficient doses, cyanide levels are undetectable [5]. If patients in this study had received hydroxocobalamin, their cyanide levels may be reduced or undetectable as measured by either analytical method used for cyanide detection. The authors did not report which, if any, patients received an antidote, specifically hydroxocobalamin and thus potentially underestimating the proportion of patients that had detectable cyanide levels.

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