

LETTER TO THE EDITOR

Response to ‘Changes in renal function during acute spinal cord injury: implications for pharmacotherapy’

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We have read with the greatest interest the letter submitted by Dr Silver¹ in relation to our recent publication in *Spinal Cord*.² Being a pioneer in the study of renal disorders associated with spinal cord injury (SCI), we greatly appreciate his comments. Dr Silver is correct. There are studies performed nearly 60 years ago that provide information on renal function in SCI that we failed to mention in our paper. We deeply regret having omitted references to the classical work of Silver and his team on the early stages of renal function after spinal cord transection.³ We had no intention of belittling such relevant work. Unfortunately, Medline searches performed using search terms such as ‘spinal cord injury glomerular filtration’ do not lead to these articles.

Our results in an experimental model of SCI are not consistent with the clinical observations of Silver and colleagues on creatinine clearance. This can be due to methodological differences and certainly warrant further investigation. Nonetheless, there is evidence pointing to the fact that there are pharmacokinetic alterations during acute SCI that are likely associated with changes in renal function, which can modify drug response.^{4,5} Such alterations have not yet been sufficiently characterized, as there has not been enough interest on these issues. We now intend to end with such inertia. Experimental models have proven to be useful to describe pharmacokinetic alterations due to SCI and can also be used for the characterization of the pathophysiological mechanisms underlying such alterations.^{5,6} Evidently, clinical pharmacology studies are absolutely required to establish if dosing regimens need adjustment in patients with SCI compared to able-bodied individuals, and how adjustments should be performed depending on the localization, intensity and time elapsed after SCI. Needless to say that patients can significantly benefit from this information, as pharmacotherapy for this patient group is still far from being optimal.⁷

We hope that our recent results² will bolster the interest of researchers in this field to study renal alterations, as well as other pathophysiological changes, and their impact on drug therapy in patients with SCI. Indeed, the investigations carried out by Silver and

co-workers in the sixties are of paramount importance for these purposes.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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- 1 Silver JR. Changes in renal function during acute spinal cord injury: implications for pharmacotherapy. *Spinal Cord* 2013; **51**: 934–935.
- 2 Rodríguez-Romero V, Cruz-Antonio L, Franco-Bourland RE, Guízar-Sahagún G, Castañeda-Hernández G. Changes in renal function during acute spinal cord injury: implications for pharmacotherapy. *Spinal Cord* 2013; **51**: 528–531.
- 3 Duggart JR, Guttman L, Silver JR. Comparative studies on endogenous creatinine and urea clearances in paraplegics and tetraplegics. *Paraplegia* 1966; **3**: 229–242.
- 4 Segal JL, Gilman TA, Thompson JF. Single-dose gentamicin clearance is a predictor of creatinine clearance in spinal man. *Am J Ther* 2010; **17**: 390–395.
- 5 García-López P, Martínez-Cruz A, Guízar-Sahagún G, Castañeda-Hernández G. Acute spinal cord injury changes the disposition of some, but not all drugs given intravenously. *Spinal Cord* 2007; **45**: 603–608.
- 6 Cruz-Antonio L, Flores-Murrieta FJ, García-López P, Guízar-Sahagún G, Castañeda-Hernández G. Understanding drug disposition alterations induced by acute spinal cord injury: role of injury level and route of administration for agents submitted to extensive liver metabolism. *J Neurotrauma* 2006; **23**: 75–85.
- 7 Segal JL, Pathak MS. Optimal drug therapy and therapeutic drug monitoring after spinal cord injury: a population-specific approach. *Am J Ther* 2001; **8**: 451–463.