doi: 10.1093/ndt/gfp197

Letters and Replies
Advance Access publication 21 May 2009

Anaemia ERBP Position statement

Sir,
I read with interest the comprehensive and helpful position statement on anaemia management by the Anaemia Working Group of ERBP [1]. The undoubted quality of this review is, for me at least (and possibly for other fastidious nephrologists), slightly marred by a few errors which have unfortunately eluded the peer-review process:
1. The CHOIR study randomized patients to an upper Hb target level of 13.5 g/dL, and not 14.3 g/dL as stated [2].
2. The recently licensed ESA is called continuous erythropoietin receptor activator and not continuous erythropoiesis receptor activator as repeatedly stated [3].
3. Haematide is a peptide-based erythropoietin receptor agonist, and not a ‘non-peptidic erythropoietin receptor agonist’ as stated [4].

While these may be perceived as simple (even trivial) errors, nevertheless, I feel they deserve correction given the importance of this position statement.

Conflict of interest statement. None declared.

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doi: 10.1093/ndt/gfp202

Advance Access publication 1 April 2009

Post-conditioning to reduce renal ischaemia/reperfusion injury

Sir,
We read with interest the article by Serviddio et al. [1] on ischaemic post-conditioning to prevent ischaemia/reperfusion (I/R)-induced damages in a rat model of ischaemic acute renal failure. Conditioning consists, in short, of repeated sequences of ischaemia and reperfusion that are imposed to an organ. They can be performed before (pre-conditioning) or after a prolonged ischaemic episode (post-conditioning). Historically, pre-conditioning was first demonstrated to be beneficial by reducing the damage induced by the
Subsequently, post-conditioning has been demonstrated to reduce the infarct size by ~40% after an ischaemic myocardial injury [2]. As I/R-related lesions are very common in numerous other organs and especially in kidney disease, studies were performed to determine the potential application of post-conditioning in this area.

We assessed the effect of post-conditioning in a mouse model of ischaemic acute renal failure [3]. We observed that three cycles of 30 s of reperfusion followed by 30 s of ischaemia after an initial ischaemic period of 30 min produced renoprotective effects. Post-conditioning was associated with a significant improvement of renal function on Day 2 and a trend to increase mouse survival. Moreover, serum creatinine levels returned to baseline values at Day 8 in all surviving mice in the post-conditioning group [3]. Our results, which, to our knowledge, represent the first report demonstrating a beneficial effect of post-conditioning in ischaemia-induced renal failure, have been now confirmed by Serviddio et al. [1] in a rat model. Further, Serviddio et al. observed that post-conditioning improved the mitochondrial function after reperfusion and decreased peroxide generation and protein oxidation [1]. These results may suggest a common pathway involved in the protective effects of post-conditioning in myocardial and in renal ischaemia (inhibition of reactive oxygen species generation and intracellular Ca\(^{2+}\) overload [4], and inhibition of the opening of the mitochondria permeability transition pore [5]).

In conclusion, there is increasing evidence showing that conditioning may represent a protective manoeuvre for organs submitted to ischaemic injuries. Particularly interesting is the beneficial effect of post-conditioning in the kidney demonstrated by our data in mice [3] and presently confirmed by Serviddio et al. [1] in rats. Should these beneficial effects be demonstrated in humans, post-conditioning would be ideally applicable to renal transplantation. Renal grafts are forcibly submitted to ischaemia (due to the required conservation of the organ), and during the surgical procedure of grafting, the artery is accessible to the surgeon. At that moment, post-conditioning may easily be performed with an expected improvement in subsequent graft function.

Conflict of interest statement. None declared.

doi: 10.1093/ndt/gfn663

Advance Access publication 1 April 2009

Reply

Sire, We thank Dr Szwarc et al. for their comments on our recent paper [1]. They have observed, in a recent report [2], that three cycles of 30 s of reperfusion followed by 30 s of ischaemia after an initial ischaemic period of 30 min are renoprotective in a mice model of ischaemic acute renal failure in terms of creatinine and mortality.

Taken together, the data from our and Szwarc reports not only confirm that post-conditioning (PC) is an effective strategy to reduce I/R injury but also demonstrate that different algorithms may be equally applied.

The novelty of our approach was to demonstrate that the mechanism of protection exerted by this surgical technique is common in different tissues by preventing mitochondrial impairment.

In fact, we have previously demonstrated [3], in an *ex vivo* model of cardiac I/R injury, that PC protects mitochondria from oxidative stress by limiting the production of H\(_2\)O\(_2\) during reperfusion. In the present paper, we show that a same mechanism acts in the kidney, suggesting that PC may be an effective strategy in almost all tissues.

We agree with Szwarc et al. that PC seems able to overcome the limit of pre-conditioning, i.e. to know before the time of ischaemia. PC is under the operator’s control during surgery, but it is also applicable in patients showing acute ischaemic damage.

Studies demonstrating the efficacy of PC in human should be strongly encouraged, but we also think that the beneficial effect of pre- and post-conditioning in renal surgery should be explored.

Conflict of interest statement. None declared.


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