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Editorial

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Editorial

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It has become dogma in cardiovascular Mg2+-research that sub-clinical Mg2+-deficiency is at least a contributory factor to arteriosclerosis and related cardiovascular disease [1–3], that an infusion of Mg2+ is beneficial after an infarct [4, 5] or during and after cardiac surgery [6] and can be used in the treatment of cardiac arrhythmias [7, 8]. However, finding concrete evidence for these assertions is at times, rather like trying to locate the legendary grey man of Ben Macdhui in the Cairngorms, who on being sighted, simply vanishes into the mists of the Scottish Highlands. Adding to this difficulty is that trying to justify support in order to provide such concrete evidence can often be countered with the reply: “Interesting, but the evidence is at present insufficient to justify support.” It is the famous “catch 22” from Joseph Heller [9]. However, as this issue of the “Journal of Clinical and Basic Cardiology” shows, concepts and ideas are changing. When putting together this issue of the Journal, I wanted to get away from the “Magnesium Dogma” and I specifically asked the authors to take a very critical approach. The papers are divided into three main groups namely, 1) basic science, 2) magnesium and cardiovascular disease and 3) cardiac surgery. The articles identify not only the major gaps in our knowledge of Mg2+-homeostasis and regulation but also importantly delineate the direction of future research.

Papers

In the basic science group, my colleagues and I document in “Magnesium homeostasis in heart: a critical reappraisal” the abysmal state of our understanding of magnesium homeostasis in heart. Halestrap in his article “The mitochondrial permeability transition – a pore way for the heart to die” describes the importance this pore could have in reperfusion injury. Grunert and colleagues in their article “Intracoronary magnesium does not reduce myocardial infarct size, even if the Mg was infused before recovery. This clinical practice had no effect on the experimentally produced fusion” found that serum Mg levels similar to those used in Mg-aspartate and MgSO4 on infarct size in a canine model of regional ischaemia and reperfusion. This problem has to be solved and a direct lineate the direction of future research.

The second problem is to define the role of magnesium in the heart and that this was the cause of the failure to see a reduction in infarct size using Mg-aspartate and MgSO4. The question now arises that it could be the aspartate anion that is having a negative effect on the heart and that this was the cause of the failure to see a reduction in infarct size. This problem has to be solved and a direct comparison between Mg-aspartate and MgSO4 is now essential, for this has important clinical ramifications. The second problem is to define the role of magnesium in the mitochondrial permeability transition pore during reperfusion.

Small clinical trials are suggestive, but not always conclusive. Despite the cost and effort involved in large or multi-centre placebo-controlled trials it would appear that such trials have now become necessary to establish the actual role of Mg2+ in cardiac surgery, supplementation in established cardiac disease and in the recovery from AMI. Serious consideration must now be given to organising carefully controlled clinical trials of adequate statistical power, to convincingly settle the argument concerning the use of Mg2+ in the treatment of cardiovascular disease.

Until these problems are solved, the sterile arguments concerning the relevance of Mg2+ as a therapeutic agent in cardiovascular disease will continue. If this issue of the “Journal of Clinical and Basic Cardiology” is to achieve anything in the field of magnesium research, it will be in not only having identified the main areas where understanding is lacking but also in proposing possible solutions to the relevant questions concerning the role of magnesium in therapeutics.
References


