University of Colorado Hospital tried to adapt this therapeutic windows.[13] Given that the levels obtained by Architect® are higher, the window has increased from 3-8ng/ml (with HPLC) to 4.5-13ng/ml (with Architect®).

Our study's most significant limitation is that we have included a small amount of measurements in the sample, which could not have been increased as Abbott Laboratories® stopped marketing the IMx® reagent. Furthermore, our study includes the most kidney transplant patients to date.

It confirms that the laboratories that determine the sirolimus levels should inform doctors when they make changes to the immunoassay employed, and the consequences that could arise. This information is of vital importance so that appropriate dose adjustments can be made. Furthermore, this information should be considered when conducting clinical studies or comparisons between different hospitals. Similarly, sirolimus therapeutic windows should be standardised for each of the techniques in use.

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Good practice guidelines on the use of erythropoiesis-stimulating agents in 2011

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To the Editor,

As coordinator of the Kidney, Dialysis and Transplant Programme in Cuba, I would be extremely grateful if you could publish this letter. I would like to highlight my opinions regarding the safe use of erythropoiesis-stimulating agents (ESA), and give my contributions on its optimal use, which is currently subject to debate.[1]

For me, introducing recombinant human erythropoietin (rhEPO) and ESA to clinical practice following replacement dialysis has been one of the most important advances in stage 5 chronic kidney disease (CKD) treatment. These techniques are the best example of how biotechnology has been successfully applied as a clinical treatment as it is used to correct severe anaemia linked with CKD, despite the adverse results highlighted by the most recent prospective and controlled studies.[2] Furthermore, we must remember that to do so we have to use supraphysiological doses of erythropoietin, justified by its non-haematopoietic effects.[3]

The reason why these studies report a greater risk to negative events, mortality and cancer makes us reflect upon important questions that are yet to be completely resolved:

1. Would the population with the greatest haemoglobin levels and worst results show other rhEPO effects and be likely to have to a homogeneous analysis?
2. Is the maximum rhEPO dose to be employed for each haemoglobin level clear?
3. Have we considered that rhEPO dose does not have to be increased to reach any haemoglobin level?
4. Are patients with adverse effects

and a higher ESA dosage those with an accepted 'accelerated atherosclerosis' and clinical or subclinical problems determining worse results in terms of mortality, previously hyporesponsive to the ESA (ferric state actually representing a deficit or decreased availability from the deposits, acute inflammation or chronic microinflammation, secondary hyperparathyroidism, among other factors)?

Recently, we are reaching a crucial moment and are currently analysing a prospective, phase IV, multicentre, open, non-controlled study, to assess the effectiveness of Cuban rhEPO. We are assessing haemoglobin levels and rhEPO doses employed over a period of 12 months, the type of response over time (variability), and adverse events. We included 617 patients from 15 nephrology departments throughout Cuba.[4]

This study highlights problems in controlling haemoglobin levels and rhEPO doses similar to those detected in other international studies.[5]

I have summarised my opinion based on the current evidence, as a strategy for guaranteeing efficient ESA use with minimum risks and in line with good clinical practice:

1. Avoid blood transfusions.
2. Start rhEPO treatment in renal anaemia patients with haemoglobin of 10g/dl.
3. Keep haemoglobin levels between 11.5g/dl and 13g/dl.
4. Never try and reach the latter by increasing rhEPO doses.
5. Question rhEPO doses over 8000U/week.
6. Use the best intravenous iron products available, depending on the elements of iron metabolism for each patient.
7. Increase the clinical method, scientific and rigorous search of the factors concerning a lack of response that are associated with ESA, undertake energetic and effective actions on this, and on those well identified mortality factors for patients with stage 5 CKD.

In summary, we must be careful in our prescription and assess the risk-benefit for each haemoglobin level, in accordance with each patient's characteristics and needs. We must consider that an inadequate EPO response or using it at a high dosage is a risk marker for mortality.

We must not forget that stage 5 CKD patients are becoming increasingly more heterogeneous with regards epidemiological and clinical aspects and related comorbidities.

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B) BRIEF CASE REPORTS

Listeria monocytogenes: an infrequent cause of peritonitis in peritoneal dialysis

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To the Editor,

Peritoneal infections are a serious complication in peritoneal dialysis and can affect the clinical state of the patient and technique viability.[1] Gram positive bacteria are most frequently involved (coagulase negative *Staphylococcus* [40%-60%], *Staphylococcus aureus* [10%-20%] and *Streptococcus* [10%-20%]). Of all peritonitis, 5%-20% are due to gram negative organisms. Other germs, which represent less than 5% of cases, are other bacteria, fungi and protozoa.[1]

There are not many cases of *Listeria monocytogenes* peritonitis published in the literature, and they generally affect immunocompromised patients.[2-12]

We present the case of a patient undergoing peritoneal dialysis due to heart failure resistant to diuretics. This is the first case of *Listeria monocytogenes* infection in the peritoneum in our hospital.

We present a 64-year-old man who underwent an operation for tetralogy of Fallot when he was younger. He later developed a severe right heart failure and eventually became resistant to diuretics. This caused