ENTERAL NUTRITION-ASSOCIATED INCRETIN EFFECT IN THE CRITICALLY ILL

Rationale: The effects of tight glycemic control (TGC) are beneficial in case of predominant use of parenteral nutrition (PN), but not when enteral nutrition (EN) is the predominant source of calories. This suggests the presence of an incretin effect, i.e. an increased release of endogenous insulin induced by EN. This effect would be supported if the amount of exogenous insulin required for glycemic control was increased during interruptions of EN, thereby reflecting improved insulin sensitivity (SI) due to the EN-related greater release of incretins.

Methods: A retrospective analysis using 52 patients on the SPRINT TGC protocol. All patients received EN for over 10 hours before EN was stopped for clinical reasons (ON/OFF) for at least 7 hours, and then restarted (OFF/ON). A validated computer model is used to compare SI 1 hour prior to stopping (or starting) and 3 hours after stopping (restarting) as a percent change to normalise across all patients. The 3 hour difference accounts for the clearance and rise of EN carbohydrates. BG values are compared to ensure no shift or bias. Diagnosed diabetics were excluded.

At the ON/OFF comparison SI is hypothesised to fall as incretin stops being secreted. At the OFF/ON it is hypothesised to rise.

Results: BG was unbiased across both changes (median change: ~0%). Median change in SI was: ON/OFF = -4.9% [IQR:-20, +23; 90%CI: -87, +46]% and 17 of 52 patients (33%) saw an unexpected rise in SI. Median SI change for OFF/ON = +20.2% [IQR:-5, +44; 90%CI: -50, +87]% and 15 of 52 patients (29%) saw an unexpected fall in SI. The before/after distributions of SI were different in both cases ($p<0.05$).

Conclusions: Changes in EN (OFF vs ON) result in significant changes in overall metabolic balance in the critically ill that should be accounted for in the delivery of TGC. These findings are consistent with the presence of an EN-related incretin effect in the critically ill.

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