It was as early as 1906 that John Finney described that post-operative ileus occurs “just at the time when the surgeon has begun to be relieved somewhat of his anxiety, and to congratulate himself that once more his labors have been crowned with success; it is most disheartening to patient and surgeon alike” (1). Although this sense of discomfort for both patient and surgeon remains true today we have come some way in being able to predict the likelihood of its occurrence. Post-operative ileus is still recognised as an important sequela of abdominal surgery, being its most common complication. Because of its often unexpected clinical presentation, and the lack of effective therapies to prevent or abort an episode, post-operative ileus continues to receive a significant share of attention in the literature.

The first published accounts of “ileus” from the late 19th century were somewhat erroneous in that they in fact described post-operative mechanical bowel obstruction requiring reoperation (1). This perhaps foreshadowed the great degree of confusion that would come to characterise modern terminology of the condition, and several texts have since attempted to redefine or reclassify it. Because of its often unexpected clinical presentation, and the lack of effective therapies to prevent or abort an episode, post-operative ileus continues to receive a significant share of attention in the literature.

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There have been recent improvements in our understanding of post-operative gut dysfunction which is now believed to be multifactorial with inflammatory cell activation, autonomic shift, exogenous narcotic administration, electrolyte derangement, and surgical stress related modulation of gastrointestinal hormone activity all playing a part. The final common pathway for these factors is impaired motility and relative intestinal ischaemia (3). It is important to note however that these mechanisms have tended to be investigated in isolation and their individual significance in the pathogenesis of a complex clinical syndrome is unclear. Furthermore, their clinical corollary as predictors of ileus is limited. It seems reasonable therefore to consider the situation in reverse, vis-à-vis the retrospective or prospective collation of clinical information from peri-operative patient cohorts to determine risk factors for prolonged ileus.

Dr. Sugawara’s work in the recent issue of Journal of Gastrointestinal Surgery has addressed this issue by identifying clinical risk factors for PPOI following major abdominal surgery and going a step further to create a nomogram predicting its occurrence (4). A broad range of baseline patient characteristics, peri-operative factors, and post-operative outcomes were analysed from a prospectively maintained database of 841 patients. Variables associated with PPOI included: male gender, worse pre-operative performance status \( \geq 2 \), longer operating time, higher intra-operative blood loss, smoking history, colorectal surgery and open surgery. The latter three variables persisted as independent predictors of PPOI on multivariate regression. These findings are remarkably similar to previous literature.
and point strongly to the aetiologic roles of relative ischaemia, surgical stress, and narcotic consumption in PPOI, albeit by inference (5). The clinical upshot of this is clear—the growing body of evidence identifying these causative factors increases their value as therapeutic targets in future prospective work.

An important strength of Dr. Sugawara’s paper relates to the inclusion of all major abdominal surgery in the analysed cohort, with subsequent stratification by procedure type. This is in contrast to much of the previous literature which has largely focused on specific operative disciplines—most frequently, colorectal surgery. It is noted with interest that although colorectal procedures were found to have the highest rate of PPOI (12.4% vs. 7.8% of upper gastrointestinal procedures, 5.4% of hepatopancreaticobiliary surgery, and 4.8% of abdominal vascular surgery), the risk factors outlined above were representative of PPOI occurrence in the whole cohort. This suggests that disruption of colonic enteric continuity, perhaps coupled with increased bowel handling, plays an important supplementary role to the surgical stress response and neurohormonal derangements that appear to underpin ileus.

The most compelling aspect of this article however relates to the creation of a predictive tool for PPOI in the form of a nomogram. Independent risk factors for PPOI were input into a cross-validation model to generate a nomogram capable of discriminating PPOI risk of 2.5% vs. 19.6% in low vs. high risk strata with a moderate level of concordance. Although this model remains to be validated in an external cohort, it draws attention to the important role of cohort studies in improving identification of at-risk patients that in turn could allow rigorous institution of preventive measures. These might include the use of thoracic epidurals with local anaesthetic infusion, restrictive intravenous fluid regimens, gum chewing, rigorous monitoring and correction of electrolytes, and early mobilisation (all strategies that form part of contemporary multimodal ERAS protocols). Finally, it is worth noting that risk prediction tools for PPOI have been previously developed elsewhere (6,7), and ongoing research may be better served by collating data across units to either improve the discriminative power of new predictive tools or, perhaps more expediently, to validate the risk stratification capability of existing models. An important proviso to future work is that it is best undertaken prospectively, however, given that a standardised definition of PPOI is difficult to apply retrospectively. Continued application and revision of these scoring systems will hone their predictive accuracy leading both to better informed consent for patients, and to the ability to stratify patients for novel treatments to prevent PPOI.

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Footnote

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