INTERNATIONAL ACADEMIC PUBLICATION DOSSIER

VIEN GUT MODEL

Integrated Outpatient Care for Complex Chronic Multimorbidity

Part B – OPERATIONAL DOCUMENTS

DOCUMENT B.5

ENABLING CONDITIONS AND PRIORITY PRINCIPLES

When several complex chronic diseases are present in the same patient,
the accompanying conditions are not controlled to reach separate targets,
but to keep the window of opportunity from closing

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AUTHOR & ACADEMIC LEAD

Nguyen Dinh Quang Independent medical researcher | Founder of Vien Gut | Overall designer of the HOW and DATA-to-operate operational layer

HOW AND DATA-TO-OPERATE DESIGN TEAM — VIEN GUT

Nguyen Dinh Quang Huy Contributed to the HOW and DATA-to-operate design | Operational management and transfer organization — Vien Gut Model

Huynh Phuoc Dai, Nguyen Son Patient-language editing | Communication data management, implementation, and transfer support — Vien Gut Model

ACADEMIC SUPPORT & WHAT (GUIDELINE) ALIGNMENT — INTERNATIONAL EXPERT GROUP

Thomas Bardin, Pascal Richette Co-authors of the EULAR recommendations, working with experts in cardiology, nephrology, hepatology, diabetes, imaging, and biostatistics at Université Paris Cité and Sorbonne University, France. They supported the transfer of WHAT from gout and comorbidity guidelines, aligned it with international standards, and supported the HOW design for the Vien Gut Model.

DATA MANAGEMENT TEAM — VIEN GUT

Truong Anh Duong, Huynh Hong Duc Data management and transfer support — Vien Gut Model

TREATING PHYSICIANS + MULTIDISCIPLINARY TEAM OF VIEN GUT GENERAL CLINIC

Clinical HOW implementation — risk stratification, window of opportunity assessment, longitudinal follow-up, risk control, polypharmacy management, and activation of the safe referral valve.

RESEARCH SITE

France–Vietnam Center for Research on Gout and Comorbidities
Vien Gut General Clinic — 13A Hong Ha, Tan Son Hoa, Ho Chi Minh City, Vietnam

POSITION OF THIS DOCUMENT IN THE VIEN GUT ACADEMIC DOSSIER

Document B.5 does not describe one single disease, and it is not a detailed treatment guide for each accompanying condition. B.5 is the closing document of Part B — the Operating Model. Its role is to define the place of enabling conditions and the principles of priority when several severe chronic diseases are present in one patient at the same time. If B.1 explains how the system is activated, B.2 explains the phase-based treatment plan, B.3 explains the necessary and sufficient conditions for the window of opportunity, and B.4 explains the patient-side participation capacity, then B.5 answers the last question of Group B: when several serious diseases act together, conflict with one another, and narrow the safety margin, how should the system set priorities so that the four validation targets can still remain possible.

Within the multi-layer architecture of the dossier, B.5 belongs to Layer 1 — Basic Architecture. It is also the direct bridge from Part B to Part C. Part C can only work in real life if enabling conditions are controlled well enough to keep the window of opportunity open. If these conditions are not controlled, and if disease–disease and drug–disease conflicts are not resolved, then crystal-free status, delayed dialysis, fewer heart-failure decompensations, and hepatic recompensation are unlikely to become lasting outcomes. This is why B.5 appears at the end of Group B: it gathers the earlier operating logic and brings it to the deepest level of clinical prioritization.

GUIDANCE FOR READERS OF B.5

• To understand the overall architectural statement of the dossier, read A.0.
• To understand the WHAT – HOW – DATA-to-operate framework, read A.1.
• To understand the definitions of the three core layers, read A.2.
• To understand the international evidence for the global HOW gap, read A.3.
• To understand the operational terminology and concepts such as the guideline paradox, reference-frame mismatch, clinical priority map, operating conditions, and clinical blind zones, read A.4–A.5.
• To understand the first visit and the identification of enabling conditions within the minimum safety core, read B.1.
• To understand the four-phase treatment plan, read B.2.
• To understand the necessary and sufficient conditions for the window of opportunity, read B.3.
• To understand the participation capacity of the patient and family, read B.4.
• To see how these enabling conditions are applied within each disease axis, read C.1–C.n.

SUMMARY

B.5 makes one central argument: in patients with complex chronic multimorbidity, the accompanying diseases should not be seen simply as “comorbidities” sitting next to the main disease. They should be seen as enabling conditions — operating conditions that determine whether the model’s four validation targets are still realistically achievable. International evidence shows that complex multimorbidity is now a global reality, while modern guidelines and the evidence hierarchy are still mainly built around single diseases. As a result, the sickest patients often fall outside the true coverage of the guidelines, are excluded from many RCTs, and must be treated inside a structural HOW gap. B.5 describes that gap at the operating level: when several diseases are present together, they do not simply add up — they amplify one another through pathological loops, functional decline, and drug–disease conflicts.

On that basis, B.5 defines enabling conditions as an interconnected system rather than a list of separate diagnoses. It builds a matrix for resolving disease–disease and drug–disease conflicts, proposes priority rules across disease axes, summarizes the minimum safe control thresholds drawn from updated guidelines, and states the central hypothesis of the Vien Gut Model: if HOW and DATA-to-operate are structured tightly enough to resolve conflicts and support longitudinal follow-up, then the window of opportunity for conservative outpatient treatment can stay open wider and longer than in usual care for complex chronic multimorbidity. The anonymized cases DTH and LAU are presented as the most extreme boundary cases that the model can still keep in outpatient care, and three of the four validation targets — delayed dialysis, reduced cardiovascular decompensation, and hepatic recompensation — are proposed as future topics for multicenter validation.

BACKGROUND

In almost every clinic — from primary care to large university hospitals — doctors increasingly see patients who do not have just one chronic disease, but several severe chronic diseases from different specialties, with different types of organ damage, in the same physically weakened body, across many age groups. Barnett and colleagues showed that multimorbidity is not an exception but the rule; [9] WHO and other international health organizations also recognize it as a global reality. Yet the whole clinical system — medical training, specialty structure, guideline design, and research design — still mainly works in a single-disease way. So the gap is not a personal weakness of doctors. It is a structural gap in global medicine.

This is not only an LMIC problem. NICE has issued a separate guideline on multimorbidity, and WHO, OECD, and other international groups have all acknowledged that single-disease models are no longer enough for these patients. But after years of recognizing the problem, the world still lacks a practical operating model that shows not only what should be done, but how to do it when several severe diseases are present in one patient at the same time. That is the direct background of B.5.

AIMS AND SCOPE OF THIS DOCUMENT

Document B.5 has seven aims. First, to define clearly the patient group with complex chronic multimorbidity that this document addresses. Second, to explain why this group is barely represented in the evidence hierarchy and in single-disease guidelines. Third, to describe the guideline paradox and the structural break in the EBM chain when many diseases are present in one patient. Fourth, to define enabling conditions as an operating layer, classify them, and show the pathological loops they create. Fifth, to establish a matrix for disease–disease and drug–disease conflict resolution as a precondition of HOW. Sixth, to propose how HOW plus DATA-to-operate can widen the safety margin in three targets proposed for multicenter validation. Seventh, to summarize the minimum safe control thresholds for enabling conditions and the rhythm by which they should be followed in DATA-to-operate.

This document does not include detailed treatment protocols for each enabling condition; practical guidance for single-disease patients without cross-guideline conflicts; randomized clinical trials; emergency inpatient care; or detailed definitions of the four validation targets and their T2T thresholds. Those topics belong to the relevant specialty guidelines, B.2, Part C, and Part D. B.5 focuses on conservative outpatient management and on the operating architecture.

1. CLINICAL CONTEXT — A GLOBAL CHALLENGE WITHOUT A SYSTEM-LEVEL SOLUTION

In everyday clinical work, doctors increasingly see patients who carry several severe chronic diseases at the same time, across different specialties, with different degrees of organ damage, inside the same body that is already physiologically fragile. Barnett and colleagues showed that more than 42% of adults have at least two chronic diseases, that the rate is over 80% in people above 80 years of age, and that multimorbidity appears 10–15 years earlier in lower-income groups than in higher-income groups. WHO estimates that more than 60% of the global disease burden comes from noncommunicable chronic diseases, and a large part of that burden involves multimorbidity. Yet modern clinical medicine is still organized mainly around the single-disease model. This mismatch between multi-disease reality and single-disease systems is where B.5 begins.

This gap is not a personal problem of generalists or specialists. Generalists see multimorbidity every day but lack an operating tool to coordinate several severe diseases at once. Specialists know one disease deeply, but their guideline does not explain HOW to manage that disease when the same patient also carries three or four other severe diseases. Health systems are organized by specialty, so the patient moves from one specialty to another while nobody keeps the full picture and nobody holds final responsibility for the overall plan. From this perspective, B.5 treats complex chronic multimorbidity as a structural problem of the whole system, not just a problem of individual cases.

2. DEFINITION — WHAT DO WE MEAN BY "COMPLEX CHRONIC MULTIMORBIDITY IN ONE PATIENT"?

Not every patient with several diagnoses belongs to the group discussed in B.5. This document uses the international concept of multimorbidity as a starting point, but adds one more layer from the Vien Gut Model: complex chronic multimorbidity. In B.5, this means a patient who has four or more severe diseases, target-organ damage, structural treatment conflicts, and usually at least one threatened vital axis. This extra layer is not used to make the language heavier. It is used to distinguish these patients from those who have several diagnoses but remain relatively stable and do not yet show major cross-guideline conflicts.

2.1. The seven dimensions of complexity — why this patient group is different

No.	Dimension of complexity	Meaning

1	Multiple specialties	At least 3–4 specialties are needed, and no single specialty can manage the case alone.
2	Different levels of disease severity	At least 2–3 diseases are already in severe or end-stage form, not just mild comorbidity.
3	Multiple pathological loops	The diseases do not sit side by side; they worsen each other.
4	Multiple metabolic disturbances	Several problems affect medication safety at the same time.
5	Multiple functional impairments	The treatment safety margin becomes clearly narrower.
6	Multiple chronic organ injuries	Each organ is both a treatment target and a treatment barrier for another organ.
7	Threat to a vital axis	At least one heart–kidney–liver–metabolic axis is close to collapse.

2.2. Age and physical reserve — extra factors that narrow the window of opportunity

This patient group does not appear only in very old people. At Vien Gut, many patients aged 45–65 already carry all of these dimensions because of late diagnosis, years of fragmented treatment, and major physical decline. This means complex multimorbidity cannot be identified by age alone. A middle-aged patient with low albumin, very low eGFR, rapid weight loss, destructive tophi, and heart failure may have a narrower safety margin than an older patient who still has good reserve. This is why B.5 always puts nutritional status, physiologic reserve, and frailty at the center of the analysis together with the disease labels.

3. ARE THESE PATIENTS REPRESENTED IN THE EVIDENCE HIERARCHY?

This is one of the most important questions in B.5, and the answer is: almost not at all. This is not because medicine has made no effort. It is because the structure of the evidence hierarchy was built for a different reference frame than the reality of these patients. RCTs sit at the base of modern high-level evidence, but to reduce noise and keep samples more homogeneous, they often exclude patients with severe multimorbidity, advanced CKD, severe heart failure, liver failure, complex polypharmacy, or very high risk. As a result, the evidence is produced in a population that is different from the patients doctors actually see. This is the mechanism that the Vien Gut Model calls a reference-frame mismatch.

Cohort studies cover reality more broadly, but they still usually focus on one main disease axis, while the other diseases appear only as control variables or small subgroups. Case reports can reach more complex cases, but they are not enough to create a HOW guideline. The result is that the patients with the highest medical need are the least well served by the evidence system. This is the clinical blind zone that B.5 must name before it can talk about solutions.

4. THE GUIDELINE PARADOX — WHY EVEN VERY GOOD DOCTORS STILL CANNOT FULLY RESOLVE THE CONFLICT

When one patient has severe gout, CKD G4, HFrEF, and Child–Pugh B cirrhosis at the same time, the doctor faces a treatment map that does not fit together. Gout guidelines suggest colchicine, NSAIDs, or corticosteroids for flares; but [2] KDIGO and ESC make NSAIDs clearly unsafe, while corticosteroids can worsen heart failure and metabolic instability. Gout care asks for ULT escalation to reach the urate target, while kidney guidance requires maximum caution with renally cleared drugs. Heart-failure care may require stronger diuretics to control congestion, but that can raise uric acid and lower GFR. Liver disease can also make many heart or gout drugs more dangerous. When each guideline is correct on its own but the combined result no longer works for the real patient, this is the guideline paradox.

The key point is that this paradox cannot be solved by writing “one more guideline” for every disease combination. Mathematically and methodologically, the number of disease combinations and severity levels rises exponentially. There are not enough RCTs, time, or resources to create strong evidence for every multimorbidity pattern. So this gap cannot be filled by classic guideline logic alone. It requires a structured operating architecture that can resolve conflicts at the bedside and across time. This is where B.5 connects with A.1, A.3, and A.4: the HOW gap is an architectural gap.

No.	Missing structural element	What happens when it is missing
1	One shared timeline that looks at all four disease axes together	Nobody sees the whole picture, so decisions rely only on a single-specialty snapshot.
2	One person with final responsibility	Nobody settles guideline conflicts, and the patient falls into a “no-one-decides” zone.
3	A continuous response mechanism between case reviews	The time between reviews becomes a blind zone where events can develop without timely response.

5. THE TWO ANONYMIZED CASES DTH AND LAU — THE LAST BOUNDARY THAT THE MODEL CAN STILL HOLD

B.5 does not present DTH and LAU simply as difficult cases. It presents them as the furthest boundary that the model can still keep in conservative outpatient care. The reason for choosing these two cases is clear: both threaten vital axes; both meet the seven dimensions of complexity; and both sit at the outer limit of what the Vien Gut Model can still manage without sending the patient fully out of outpatient care. Because they sit at that final boundary, they reveal the priority logic, the conflict-resolution matrix, and the role of DATA-to-operate in keeping the window of opportunity open.

5.1. The anonymized case DTH — the model’s outer limit

DTH combines several severe axes at once: decompensated F4 Child–Pugh B cirrhosis, end-stage CKD, very severe anemia, deep secondary adrenal insufficiency, and destructive tophaceous gout. The pathological loop is clear: cirrhosis lowers albumin and narrows drug safety; gout flares maintain systemic inflammation; CKD and electrolyte problems increase cardiovascular risk; hidden adrenal insufficiency means that any physiologic stress can suddenly push the patient into multi-organ collapse. In fragmented care, each specialty has a reasonable reason to delay or limit its own treatment, and together those delays can leave the patient with almost no treatment pathway.

What the Vien Gut Model did here was to set three preconditions: first identify and treat secondary adrenal insufficiency; assess every medication through the combined lens of CKD, cirrhosis, and adrenal insufficiency; and follow cortisol, electrolytes, albumin, and INR over time with the safe-referral valve always ready. On that basis, DTH remained in outpatient care for four years, with very low but still stable eGFR without RRT, clear improvement in the liver axis, and a meaningful reduction in tophus burden. B.5 does not use this case to claim that the whole problem has been solved. It uses the case to show that controlling enabling conditions can truly keep the window of opportunity open longer.

5.2. The anonymized case LAU — a heart–kidney–endocrine loop that requires precise polypharmacy management

LAU shows a different type of multi-axis loop: HFrEF, progressive CKD, low cortisol, severe peripheral vascular disease, and severe gout. In this case, the diuretic needed for heart failure raises uric acid and lowers eGFR; the beta-blocker needed for the heart can mask hypoglycemia or stress responses when cortisol is low; and NSAIDs are clearly contraindicated. In fragmented care, medication changes can connect into a chain of side effects that nobody fully sees.

The key lesson from LAU is that every medication change must pass through the conflict-resolution matrix before the final decision is made; follow-up must be frequent after each change; and cortisol must be part of the baseline dataset rather than something checked only after symptoms appear. This is another expression of the same message: enabling conditions are not “extra diseases on the side.” They help decide whether the patient still has or has already lost the outpatient window of opportunity.

6. ENABLING CONDITIONS — DEFINITION, CLASSIFICATION, AND PATHOLOGICAL LOOPS

After defining the patient group and illustrating the issue with the two boundary cases, B.5 then gives its formal definition. Enabling conditions are comorbid diseases or states that are managed as preconditions for safely reaching the four validation targets, not as separate independent targets. This means that the level considered “well enough controlled” is not always the same as the ideal target in a single-disease guideline. Instead, it is the minimum safe threshold adjusted to the real multimorbidity context of that patient. This is one of the key differences between B.5 and the usual logic of simply adding one disease guideline to another.

No.	Enabling condition	Effect on the validation targets
1	Diabetes	Accelerates CKD and worsens the cardiovascular axis.
2	Hypertension	Damages both kidney and heart.
3	Chronic anemia	Makes heart failure worse and narrows the safety margin for many interventions.
4	Glucocorticoid-induced adrenal insufficiency (GIAI)	A hidden and highly dangerous enabling condition; under stress it can suddenly trigger multi-organ decompensation.
5	Chronic electrolyte disorders	Push heart failure and CKD toward faster decompensation.
6	Malnutrition / low albumin	Narrows the safety margin of many drugs.
7	Dyslipidemia	Accelerates the atherosclerotic axis.
8	High uric acid without overt gout symptoms	Can still affect the kidney and cardiovascular axes.

6.1. Pathological loops — why enabling conditions are not independent

B.5 emphasizes that enabling conditions do not exist as parallel and separate items. They create pathological loops. Anemia can worsen heart failure; worsening heart failure leads to higher diuretic doses; diuretics lower potassium and raise uric acid; gout flares increase inflammation and push the patient toward corticosteroids; corticosteroids raise blood glucose; and poor glucose control can accelerate CKD. Progressive CKD raises uric acid; higher uric acid causes more flares; and more flares plus inflammation can further worsen CKD. Decompensated cirrhosis lowers albumin; low albumin makes many drugs more dangerous; and hyponatremia and hypokalemia from resistant diuretic use can make the window of opportunity close faster. These loops explain why treating each enabling condition one by one in fragmented care often sees only separate links, not the loop itself. B.5 treats loop recognition and loop interruption as a required operational goal.

7. RESOLVING DISEASE–DISEASE AND DRUG–DISEASE CONFLICTS — A PRECONDITION OF HOW

One of B.5’s central arguments is that HOW plus DATA-to-operate is not only about longitudinal follow-up and trend reading. A more basic precondition is the ability to identify and resolve disease–disease and drug–disease conflicts before any treatment decision is finalized. Without that ability, longitudinal follow-up only records the downward slide instead of stopping it. Fragmented care usually builds the treatment

plan first and discovers the conflict only after harm has already happened. The HOW of the Vien Gut Model works the other way around: conflict resolution is a required step before the plan is made. DATA-to-operate does not only record what happened; it must also detect early signs that a conflict is starting to form. That is how the window of opportunity is widened.

7.1. Conflict-resolution matrix — eight common clinical conflict pairs

No.	Conflict pair	Why it conflicts	Typical direction of resolution
1	ULT (T2T) vs CKD G3b–G4	Oxypurinol accumulation when GFR is low	Lower dose, close eGFR follow-up, clear SLA.
2	Diuretics (heart failure) vs uric acid / GFR	Raise uric acid and reduce GFR	Adjust dose carefully and follow uric acid plus eGFR together.
3	NSAIDs (flare) vs CKD + heart failure	Clearly unsafe in CKD plus heart failure	Use colchicine or short-course corticosteroids instead.
4	ACEi/ARB (kidney) vs cirrhosis	Can lower blood pressure too much or worsen HRS risk in cirrhosis	Follow blood pressure and creatinine closely; stop if unsafe.
5	Corticosteroids (flare) vs diabetes / GIAI	Raise glucose and may reveal adrenal insufficiency	Use stress dosing logic and follow glucose plus cortisol.
6	Statins (cardiovascular axis) vs Child–Pugh C cirrhosis	Higher liver-related risk in advanced cirrhosis	Stop or reduce dose; follow liver tests closely.
7	Strong ULT target (<5 mg/dL / <300 µmol/L) vs severe liver disease	The liver safety margin is narrow	Start low, increase slowly, follow liver enzymes.
8	Beta-blockers (heart failure) vs GIAI	May mask hypoglycemia and stress responses when cortisol is low	Follow glucose and cortisol; warn the patient and family.

7.2. Conflict resolution must go beyond one pair at a time

B.5 adds one more important step: conflict resolution cannot stop at the A–B pair, because a solution that works for A–B may push axis C or D outside the safety margin. A classic example is using colchicine to solve the flare–CKD conflict, while the patient is also taking clarithromycin, which makes colchicine dangerously toxic. This means the matrix cannot look only at the direct drug–disease pair. It must look across the full medication map, the full disease map, and then be checked again with DATA-to-operate after the intervention. This is why B.5 is not only a document that identifies conflicts. It is also a document that designs how those conflicts should be judged and resolved.

8. HOW + DATA-TO-OPERATE CAN WIDEN THE SAFETY MARGIN — THREE TARGETS PROPOSED FOR MULTICENTER VALIDATION

8.1. Three targets proposed for multicenter validation

Validation target	Typical conflicts	DATA-to-operate to follow	Operational hypothesis
Target 2: Delayed dialysis	ULT toxicity when kidney function is low; ACEi/ARB-related hyperkalemia; diuretics lowering GFR; NSAIDs unsafe; corticosteroids worsening blood pressure and diabetes.	Serial eGFR, potassium, and uric acid; distinguish temporary eGFR dips from true CKD progression.	A dynamic safety margin may be preserved longer if conflict resolution is structured and continuous.
Target 3: Fewer cardiovascular decompensations	Diuretics raise uric acid; beta-blockers can mask hypoglycemia; NSAIDs are unsafe; flares act as systemic inflammatory triggers.	Weight, edema, electrolytes, uric acid, and flares followed together over time.	More precise medication adjustment may reduce decompensation.

Target 4: Hepatic recompensation	Low albumin gives the narrowest safety margin; statins may be limited; resistant diuretic use may cause electrolyte disorders; infection can close the window suddenly.	Albumin, INR, bilirubin, ascites, abdominal girth, and sodium over time.	May keep Child–Pugh B patients safely in outpatient care longer.

8.2. Why AI cannot replace a structured HOW

It is important to state clearly that current AI cannot replace a structured HOW in multimorbidity conflict resolution. AI can list drug interactions, but it cannot truly decide between guideline A and guideline B in one patient who is in Phase 1, whose eGFR is falling quickly, and whose cortisol is low. The core reason is that AI lacks the three elements that B.5 identifies as essential: one shared timeline that looks across all four axes, one person with final responsibility, and a continuous response mechanism between case reviews. AI can create the illusion that a HOW exists, when in fact there is no operating architecture behind the advice.

8.3. The integrated WHAT–HOW–DATA-to-operate software is still being completed

At the time of publication (March 2026), the integrated WHAT–HOW–DATA-to-operate software of Vien Gut is still under development, with completion expected by the end of 2026. This means that all the results discussed in B.5 — structured conflict resolution, time-series follow-up of enabling conditions, and widening of the safety margin — were achieved through human capability and manually organized processes built over nearly two decades, not through a closed digital system. This is both a limitation and a strong proof that the operating architecture existed and worked before it was digitized. A completed software layer will be necessary for transfer, scale-up, and multicenter validation, because no health system can be expected to rely on 18 years of one center's personal accumulated experience.

8.4. What this means for multicenter validation

The three targets — delayed dialysis, fewer cardiovascular decompensations, and hepatic recompensation — are proposed for multicenter validation precisely because they lie at the boundary where HOW plus DATA-to-operate may make the greatest difference compared with usual care. Once the integrated software is complete, centers joining the validation process will be able to work with the same WHAT–HOW–DATA-to-operate architecture without depending on one center's personal experience. The validation results will then answer the key question: can this operating architecture be reproduced and can it produce comparable results in different settings?

9. SAFE CONTROL THRESHOLDS FOR ENABLING CONDITIONS BASED ON UPDATED GUIDELINES

Enabling condition	Ideal threshold (guideline)	Practical threshold (multimorbidity)	Safe-referral trigger	Guideline source
HbA1c (diabetes)	<7%	Usually <8–8.5%	>9% or severe hypoglycemia	ADA/EASD
Blood pressure	<130/80	Flexible when CKD and cirrhosis coexist	Dangerous hypotension or >160	ESC / KDIGO
Hemoglobin	≥12–13 g/dL	Enough to keep a cardiovascular safety margin	<7 g/dL or severe symptomatic anemia	KDIGO
Potassium	3.5–5.0 mmol/L	3.5–5.5 depending on CKD and drugs	>6.0 or <3.0	KDIGO / ESC
Sodium	135–145 mmol/L	≥130 may be acceptable in cirrhosis	<125 or a rapid fall	EASL

Albumin	≥3.5 g/dL	Often ≥2.8 depending on context	<2.5 with edema and ascites	EASL
Morning cortisol	≥10 µg/dL	At least ≥5 with tight follow-up	<3 with physiologic stress	Endocrine Society
Uric acid	<360 µmol/L (T2T)	Flexible in severe CKD or liver disease	Ongoing flares plus progressive tophi	ACR / EULAR

10. SPECIAL CASE — GLUCOCORTICOID-INDUCED ADRENAL INSUFFICIENCY (GIAI)

B.5 gives GIAI its own section because it is a special enabling condition: it often causes few clear symptoms before the event, it is very common in severe gout patients with long-term corticosteroid exposure, and under physiologic stress it can trigger sudden multi-organ decompensation. This is a clear example of how an enabling condition can become an invisible breaking point if it is not actively built into HOW plus DATA-to-operate.

B.5 sets four required operating rules for GIAI: screen with morning cortisol in patients with a history of prolonged prednisolone use; define action thresholds when cortisol is low; prepare a stress-dose plan when procedures or physiologic stress occur; and make every medication change that affects glucose, electrolytes, or blood pressure pass again through the GIAI lens. The DTH case illustrates this clearly: deep cortisol deficiency was detected at the first visit, entered into DATA-to-operate immediately, and treated as a precondition before other decisions were made. B.5 uses this example to emphasize that hidden enabling conditions may not make the patient “look sicker” at first glance, but they still help determine whether the window of opportunity can remain open.

11. ENABLING CONDITIONS INSIDE DATA-TO-OPERATE — FOLLOW-UP RHYTHM AND ACTION THRESHOLDS

Another important contribution of B.5 is that it pulls enabling conditions directly into DATA-to-operate. This document makes clear that DATA-to-operate must follow not only the four validation axes, but also the enabling conditions, because these conditions are exactly what make the window of opportunity wider or narrower. B.5 therefore sets the follow-up rhythm by risk level and treatment phase, not by one fixed calendar. Metabolic data, electrolytes, hematology, liver function, morning cortisol, and visual longitudinal data all have their own follow-up rhythms and their own trigger signals that can shorten the interval when needed. In this way, enabling conditions become a living part of the operating system rather than a diagnosis list hanging at the margin.

The key point is that B.5 does not ask for the maximum number of tests. It asks the system to know what to follow, in which phase, at which risk level, and at what threshold it must respond. That is how DATA-to-operate changes from “data to store” into “data to act on.” Only then can enabling conditions truly be managed as an interconnected system.

12. STATEMENT ON EVIDENCE LEVEL AND DEGREE OF INFERENCE

B.5 includes an important statement that should remain clear: each layer of content has a different evidence level. The definitions of enabling conditions, many safe-control thresholds, and many common disease–disease or drug–disease conflicts are drawn from single-disease guidelines and international literature, so their evidence level is generally B–C within the context where they were produced. The conflict-resolution matrix, the priority rules, and the DATA-to-operate rhythm are clinical inferences built from single-disease evidence plus nearly 18 years of accumulated observation at Vien Gut; there are no direct RCTs covering this exact patient group. The DTH and LAU cases, and observations such as stable eGFR in CKD G5 or the ability to start ULT in Child–Pugh B disease, are single-center practice observations. The three targets proposed for multicenter validation in CKD G5, chronic heart failure, and

decompensated cirrhosis remain validation hypotheses that still need academic dialogue and multicenter testing. This transparent declaration is one of the reasons B.5 is academically stronger and more honest.

13. LIMITS OF THIS DOCUMENT

No.	Content	Included in B.5?	Reference
1	Enabling conditions: definition, classification, and pathological loops	Included	B.5 — Section 6
2	Matrix for disease–disease / drug–disease conflict resolution	Included	B.5 — Section 7
3	Safe-control thresholds based on updated guidelines	Included	B.5 — Section 9
4	HOW + DATA-to-operate widening the safety margin	Included	B.5 — Section 8
5	Glucocorticoid-induced adrenal insufficiency (GIAI)	Included	B.5 — Section 10
6	Follow-up rhythm for enabling conditions	Included	B.5 — Section 11
7	Detailed treatment protocols for each enabling condition	No	Specialty guidelines
8	Detailed definitions of the four validation targets	No	Part C
9	First visit, four-phase plan, and window of opportunity	No	B.1, B.2, B.3
10	Role of the patient	No	B.4
11	Dialogue and validation framework	No	Part D

14. THE PLACE OF B.5 IN THE VIEN GUT DOCUMENT SYSTEM

B.5 closes the logic of Group B. B.1 identifies enabling conditions within the minimum safety core of the first visit. B.2 defines the phase and the follow-up rhythm within which these conditions must be controlled. B.3 tells us whether the window of opportunity is still open. B.4 shows how the patient's participation capacity directly affects the control of enabling conditions. B.5 gathers all of this and answers the hardest question: when many diseases are present together and obstruct one another, which intervention must be prioritized, which one must be slowed down, which data must be read more frequently, and what must the system do so that the four validation targets in Part C can still remain possible. For this reason, B.5 is both the closing document of Part B and the operating bridge to Part C and Part D.

15. CONCLUSION

Patients with complex chronic multimorbidity — carrying four to seven severe diseases across several specialties, with multiple pathological loops, multiple functional impairments, and threats to vital axes — are not adequately served by any single-disease guideline and are systematically under-represented in the modern medical evidence hierarchy. This is not a small gap. It is an architectural gap in global medicine. The guideline paradox and the structural break in the EBM chain explain why even when several good doctors are involved, the patient may still fall into a zone where nobody can make the final decision and may quickly move beyond the outpatient safety margin.

The Vien Gut Model proposes an architectural answer: add HOW and DATA-to-operate to the EBM chain, and manage enabling conditions as an interconnected system rather than as diseases standing at the side. The anonymized cases DTH and LAU show that even at the furthest boundary of the model, when enabling conditions are controlled, when disease–disease and drug–disease conflicts are resolved before treatment decisions are made, and when data are read as time series rather than snapshots, the window of opportunity for conservative outpatient treatment can stay wider and longer. On that basis, three of the model's four validation targets — delayed dialysis, reduced cardiovascular decompensation,

and hepatic recompensation in decompensated cirrhosis — are offered as invitations for future multicenter validation in Vietnam and across LMIC settings.

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PRACTICE PROVENANCE

Document B.5 is the product of nearly two decades of integrated clinical practice at Vien Gut. It did not come from theory alone or from a simple summary of guidelines. It came from direct operating experience with the most complex patients — the very patients for whom medicine usually has no guideline coverage.

• 2007–2010: the integrated outpatient model began to take shape; the first observations appeared about pathological loops and the limits of single-disease guidelines in complex chronic multimorbidity.

• 2014: contact with Prof. Thomas Bardin helped confirm the HOW gap in international medicine and supported the systematization of the operating concepts.

• 2017–2021: the conflict-resolution matrix, enabling-condition thresholds, and DATA-to-operate follow-up rhythm were progressively systematized from real clinical cases, including extreme boundary cases such as DTH and LAU.

• 2025–2026: the full academic dossier, including Document B.5, was written and systematized to prepare for academic dialogue and future multicenter validation.