



AIRWAY DEVELOPMENT INCORPORATED

ENHANCED RECOVERY AFTER SURGERY IN NIGERIA

A definitive, physiology-driven clinical guide for analgesia, hydration, anaesthesia, oxygenation and functional recovery.

RECOVERY BEGINS BEFORE ANAESTHESIA

This protocol replaces prolonged fasting, isolated opioid treatment and passive recovery with a coordinated pathway. Eligible elective patients arrive better hydrated and closer to euvolemia, receive preemptive multimodal analgesia, enter the operating theatre with oxygen reserves maximized, and begin drinking, eating and walking early after surgery.

Adult elective surgical pathway | Hospital implementation edition | September 2026

SECTION 01

Clear coconut water: preoperative hydration and metabolic preparation

Permitted clear coconut water ends unnecessary dehydration. Eligible elective patients arrive better hydrated and closer to euvolemia, require less replacement of fasting deficit, tolerate induction more smoothly and begin recovery with carbohydrate and electrolyte reserves already available.

HYDRATION SCHEDULE	250 mL on awakening, then comfortable sips until 1 hour before surgery
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TYPICAL COMPOSITION OF FRESH COCONUT WATER Per 100 mL: approximately 250 mg potassium, 105 mg sodium, 25 mg magnesium, 24 mg calcium, 3.7 g carbohydrate and 95 g water. A 250 mL serving therefore supplies about 625 mg potassium, 263 mg sodium, 63 mg magnesium, 60 mg calcium and 9 g carbohydrate. Concentrations vary with coconut maturity, cultivar and processing; the hospital uses a pulp-free, hygienically prepared product and records the volume. ELECTROLYTE PHYSIOLOGY Potassium maintains resting membrane potential, myocardial electrical stability and skeletal-muscle function. Magnesium supports ATP-dependent reactions, neuromuscular stability and potassium handling; it also limits NMDA-mediated excitability. Sodium sustains extracellular volume, while water expands the available fluid compartment. Together, these constituents restore thirst, support circulation and reduce the IV crystalloid required solely to correct prolonged fasting.	INSULIN RESISTANCE AND WOUND HEALING Surgical stress increases catecholamines, cortisol and inflammatory cytokines, producing hepatic glucose output and peripheral insulin resistance. Preoperative carbohydrate intake interrupts the prolonged fasting signal, reduces catabolic metabolism and preserves glucose utilization. Better metabolic control supports neutrophil function, collagen synthesis, protein preservation and tissue repair; this creates a more favorable wound-healing environment than dehydration plus starvation. IMPLEMENTATION STANDARD The holding-bay team verifies the one-hour stop time, records the volume and transfers that information to anaesthesia. Oral hydration becomes the first fluid intervention; IV fluid then replaces measured surgical losses and demonstrated intravascular need instead of an assumed fasting deficit. Restart clear sips early in recovery as part of the feeding pathway.
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KEY SOURCES: USDA nutrient dataset [22]; ASA fasting guidance [23]; ERAS LMIC abdominal/pelvic guideline [24]; carbohydrate-loading evidence [25].

SECTION 02

Preoperative holding bay: the operational center of ERAS

A dedicated holding bay converts ERAS from a list of intentions into a completed sequence. Oral hydration, preemptive medication, regional anaesthesia, monitoring, education and documentation occur before the operating room is occupied.

STANDARD TIMING	Patient arrives 90-120 minutes before theatre transfer
CORE WORKFLOW 1. Confirm identity, procedure, consent, allergies and medication record. 2. Record last solid intake and the exact coconut-water stop time. 3. Measure baseline blood pressure, heart rate, respiratory rate, oxygen saturation, pain and anxiety. 4. Administer observed oral ERAS medication at its scheduled time. 5. Establish IV access. 6. Provide patient education and expected milestones. 7. Perform regional anaesthesia with monitoring and light sedation. 8. Complete the documented handover to theatre. WHY THIS CHANGES OUTCOMES Correct timing produces therapeutic drug concentration before incision. Oral fluids produce better hydration and closer euvoemia before induction. A completed block reduces induction-to-incision delay and begins afferent blockade before tissue injury. Education reduces anxiety and gives the patient a clear plan to drink, breathe deeply, sit, stand and walk.	PHYSICAL DESIGN Place the bay immediately upstream of theatre. Provide reclining chairs with cup holders, oxygen, suction, a bag-mask device, standard monitors, capnography for procedural sedation, an emergency cart, IV supplies, medication storage, ultrasound, regional block equipment, hand-hygiene facilities and a visible timing board. Separate medication preparation from documentation and maintain direct anaesthesia supervision. RELIABILITY MEASURES Track the percentage of eligible patients who receive fluids to the correct stop time, receive each oral medication on time, enter theatre with a completed block, and have a complete handover. Review omissions every week. The holding bay becomes the hospital's visible standard for preparation, dignity, calm and coordinated care.

KEY SOURCES: ERAS implementation principles [24,26]; WHO surgical checklist resources [27].

SECTION 03

Melatonin: anxiolysis, sleep alignment and analgesic support

Melatonin establishes a calm preoperative state without building the pathway around benzodiazepine sedation. It reduces anxiety, supports circadian organization and strengthens multimodal analgesia before incision.

RECOMMENDED DOSE	Melatonin 3 mg PO or SL, 60-90 minutes before surgery
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PHARMACOKINETICS Melatonin peaks in 30-60 minutes after oral dosing. Variable bioavailability results from first-pass metabolism. CYP1A2 forms 6-hydroxymelatonin, and conjugated metabolites leave through urine. Immediate-release half-life is approximately 40-60 minutes, while receptor effects persist longer. MOLECULAR MECHANISM Melatonin activates MT1 and MT2 G-protein-coupled receptors in the suprachiasmatic nucleus, thalamus, spinal cord and peripheral tissues. MT1 signaling decreases adenylate cyclase/cAMP activity; MT2 signaling modifies neuronal excitability and circadian phase. Analgesia is reinforced through descending inhibitory pathways, GABAergic signaling and endogenous opioid interactions. Melatonin also suppresses NF-kB activation, decreases COX-2 expression and inflammatory cytokines, and directly scavenges reactive oxygen and nitrogen species.	HOW THIS IMPROVES RECOVERY Lower sympathetic activation reduces preoperative distress and the stress response. Better sleep-wake alignment reduces agitation and supports postoperative orientation. Antinociceptive and anti-inflammatory signaling lowers central sensitization, decreases pain amplification and reduces the amount of hypnotic or opioid required to achieve comfort. • Administer in the holding bay early enough for peak effect before transfer. • Document anxiety before dosing and comfort at operating-room entry. • Use as the non-benzodiazepine foundation of preoperative calming. EXPECTED PATHWAY EFFECT The patient enters the operating room calmer, more cooperative and physiologically organized. This improves positioning, regional block performance, induction quality and the transition into recovery.
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KEY SOURCES: Yousaf et al., Anesthesiology 2010 [3]; Hansen et al., Anaesthesia 2014 [4]; Madsen et al., Cochrane 2020 [5]; randomized 3 mg studies [6,7].

SECTION 04

Acycor Plus: dual-pathway preemptive analgesia

Acycor Plus is administered before incision so cyclooxygenase inhibition and central acetaminophen analgesia are active when surgical inflammation begins. The combination attacks pain at both peripheral and central sites and sharply reduces dependence on postoperative opioids.

RECOMMENDED DOSE

2 tablets PO: aceclofenac 200 mg + acetaminophen 1,000 mg, 60 minutes preoperatively

ACECLOFENAC PHARMACOKINETICS

Aceclofenac is rapidly absorbed and peaks in 1.25-3 hours. It is more than 99% protein bound and has a half-life near 4 hours. Hepatic metabolism forms 4'-hydroxyaceclofenac and diclofenac-related metabolites, which are eliminated mainly in urine.

ACETAMINOPHEN PHARMACOKINETICS

Acetaminophen peaks in 30-60 minutes. Glucuronidation and sulfation clear most of the dose; CYP2E1 forms a small amount of NAPQI that glutathione neutralizes. Plasma half-life is 2-3 hours.

MOLECULAR CONTROL OF PAIN AND INFLAMMATION

Aceclofenac preferentially suppresses COX-2 activity and reduces conversion of arachidonic acid to prostaglandin E2. Lower PGE2 decreases nociceptor sensitization, tissue hyperalgesia, vasodilation and inflammatory edema. Acetaminophen acts mainly within the central nervous system through prostaglandin/peroxidase modulation, descending serotonergic pathways and the AM404 endocannabinoid pathway. Together, they reduce peripheral inflammatory input and central amplification before those processes become established.

ERAS EXECUTION

Give both tablets as one observed dose in the holding bay and record the full 1,000 mg acetaminophen contribution on the medication chart. The postoperative analgesic plan then continues multimodal therapy without duplicating combination-product acetaminophen. Procure only an in-date, locally authorized Acycor Plus supply and verify current NAFDAC registration status.

KEY SOURCES: NAFDAC Acycor Plus product record [1]; Hinz et al., Clin Pharmacol Ther 2003 [8]; aceclofenac and acetaminophen pharmacology reviews [9,10].

SECTION 05

Gabapentin: prevention of central sensitization

Gabapentin is given before surgery to suppress excitatory neurotransmitter release during the period when tissue injury would otherwise amplify spinal pain signaling. In the selected major-pain pathway, it strengthens opioid-sparing analgesia and improves comfort with movement.

RECOMMENDED DOSE	Gabapentin 600 mg PO, 90-120 minutes before surgery
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PHARMACOKINETICS Gabapentin uses a saturable intestinal amino-acid transporter and peaks in 2-3 hours. Bioavailability decreases as the dose rises. It is not protein bound or hepatically metabolized; the kidney eliminates it unchanged. Half-life is approximately 5-7 hours. MOLECULAR MECHANISM Gabapentin binds the alpha-2-delta-1 auxiliary subunit of presynaptic voltage-gated calcium channels. This reduces calcium entry into activated dorsal-horn terminals and decreases release of glutamate, substance P and norepinephrine. The result is less excitatory transmission, reduced wind-up, less hyperalgesia and lower central sensitization after incision. Gabapentin does not act at the GABA-A receptor and does not function as an opioid.	ERAS EFFECT By diminishing spinal amplification of nociceptive input, gabapentin reduces early pain intensity and rescue-opioid exposure in procedures where neuropathic or high-intensity pain is expected. Lower opioid exposure reduces nausea, ileus and urinary retention and allows the patient to breathe deeply, cough, stand and walk sooner. OPERATIONAL STANDARD Administer the 600 mg dose in the holding bay with the rest of the procedure-specific multimodal plan. Confirm renal function when choosing the pathway, record the dose before theatre transfer and include sedation/ventilation assessment in routine postoperative monitoring. The dose is not repeated automatically.
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KEY SOURCES: Ho et al., Pain 2006 [11]; Tiippana et al., Anesth Analg 2007 [12]; Verret et al., Anesthesiology 2020 [13]; abdominal ERAS meta-analysis 2025 [14].

SECTION 06

Preoxygenation and denitrogenation

Preoxygenation replaces nitrogen in the functional residual capacity with oxygen. The lungs become an oxygen reservoir that continues transferring oxygen to blood during apnea, extending the time available for laryngoscopy and securing the airway.

DIAGNOSTIC CRITERION

Minimum expired O₂ (EtO₂) 80%; target EtO₂ 90% or expired N₂ 5%

THE PHYSIOLOGY

At room air, the functional residual capacity contains mostly nitrogen and only a limited oxygen store. Breathing 100% oxygen washes nitrogen from alveoli and raises alveolar oxygen concentration. During apnea, oxygen continues moving into blood while carbon dioxide accumulates slowly in alveoli; this concentration gradient supports apneic oxygenation. Greater oxygen content in the FRC delays arterial desaturation and protects the brain and heart during airway instrumentation.

TECHNIQUE

Use a tight-fitting mask with 100% oxygen and eliminate circuit leaks. Standard preparation is 3 minutes of normal tidal breathing or 8 deep vital-capacity breaths when time is limited. Elevate the head 20-30 degrees. Maintain airway patency and continue nasal oxygen during laryngoscopy when appropriate. In obesity, pregnancy, critical illness or low FRC, add CPAP/PEEP or noninvasive pressure support to recruit lung volume.

MEASURE SUCCESS - DO NOT GUESS

Expired oxygen directly measures denitrogenation. An EtO₂ of at least 80% confirms effective preoxygenation; continue toward 90%, the standard endpoint for maximal preoxygenation. Failure to reach the target means leak, rebreathing, inadequate flow, airway obstruction, low FRC or insufficient time. Correct the cause before induction whenever the clinical situation allows.

WHY SPO₂ IS INSUFFICIENT

A pulse oximeter reading of 100% confirms hemoglobin saturation but does not prove that alveolar nitrogen has been washed out. A patient can display SpO₂ 100% while retaining a poor oxygen reservoir. EtO₂ therefore functions as the diagnostic criterion for preoxygenation quality, while SpO₂ remains the downstream monitor of blood oxygenation.

KEY SOURCES: Nimmagadda et al., Anesth Analg 2017 [38]; preoxygenation review [39].

SECTION 07

Airway cart: a complete sequence for routine airway management

The airway cart supports every airway from preparation through confirmed ventilation and tracheal-tube securement. It is present in every operating room, emergency department, recovery room, intensive care unit and every other location where an airway is managed. The correct size of every required item is selected, opened when appropriate, tested and positioned for immediate use before induction begins.

READINESS STANDARD	One standardized, fully stocked airway cart at the patient's side before every airway intervention
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1. POSITIONING Place an intubation pillow under the head and elevate or ramp the patient until the external auditory meatus is horizontally aligned with the sternal notch. This head-elevated position improves mask ventilation and creates the most direct practical pathway through the oral, pharyngeal, laryngeal and tracheal structures. Confirm head and neck mobility and maintain neutral positioning when cervical-spine precautions apply. 2. PREOXYGENATION Apply a correctly fitting oxygen mask using 20 L/min fresh-gas flow. Prepare the breathing circuit, reservoir bag, oxygen source, suction and a self-inflating bag-mask device, and confirm effective tidal breathing on capnography and the reservoir bag. Continue preoxygenation until expired oxygen is at least 80% before administering sedative or induction medication. 3. MONITORING Apply and verify electrocardiography, a correctly sized non-invasive blood-pressure cuff, pulse oximetry, quantitative train-of-four monitoring, bispectral index, core-temperature monitoring and a gas analyzer with capnography, inspired oxygen, expired oxygen and anaesthetic-agent measurement. Display audible alarms and obtain baseline values before induction.	4. INDUCTION Prepare the propofol infusion and magnesium infusion with programmed pumps, patent intravenous access and the intended patient-specific doses. Apply the appropriate color-coded label to every syringe. Each label states the medication name, concentration, preparation date, preparation time and initials of the anesthesia provider. Induction begins only after positioning, preoxygenation, monitoring, suction, ventilation equipment and the primary and alternate airway devices are ready. 5. VENTILATION Select the mask and oral airway to the patient before induction; keep the oral airway immediately accessible. Prepare nasal airways when appropriate, oxygen tubing, circuit, reservoir bag, PEEP valve and suction. Confirm ventilation by continuous capnography, bilateral chest movement, appropriate airway pressure and improving or stable oxygen saturation. 6. AIRWAY SECUREMENT Prepare a tested LED laryngoscope with the correct blade, an appropriately sized cuffed endotracheal tube, syringe, lubricant, standard bougie, articulating bougie, videolaryngoscope with the correct blade, rigid video stylet and an appropriately sized intubating laryngeal mask airway with its compatible tracheal tube. Keep Magill forceps, tape or ties, a tube holder and a stethoscope ready. Confirm tracheal placement with sustained waveform capnography and document tube depth. Inflate the cuff with the minimum occlusive volume: at 20 cm H2O airway pressure, add air only until the audible leak just disappears. Measure cuff pressure and maintain 20-30 cm H2O to provide ventilation while preventing tracheal mucosal ischemia. Secure the tube before repositioning the patient.
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KEY SOURCES: ASA difficult-airway guideline [52]; Difficult Airway Society guideline [53]; international safe-anaesthesia standards [54].

SECTION 08

Preprocedural sedation before spinal and regional anaesthesia

Midazolam and fentanyl provide decisive anxiolysis, amnesia, analgesia and immobility for positioning and needle placement. The full sedation dose is administered only after five minutes of verified high-flow preoxygenation, complete physiologic monitoring and immediate readiness to ventilate the patient.

ADULT PROTOCOL DOSE	Midazolam 2 mg IV + fentanyl 100 micrograms IV after 5 minutes of preoxygenation
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MANDATORY PREOXYGENATION Apply ECG, noninvasive blood pressure, pulse oximetry and continuous waveform capnography; establish a patent IV and confirm the airway equipment is ready. Place a tight oxygen mask and deliver 100% oxygen at 20 L/min for five full minutes before either sedative is given. Continue oxygen during positioning and block performance. This creates the largest practical alveolar oxygen reservoir before drug-induced hypoventilation or apnea can occur. WHY 20 L/MIN MATTERS Spontaneous inspiration follows a near-sinusoidal flow pattern: inspiratory flow accelerates from zero to a mid-inspiratory peak and then decelerates to zero. Peak instantaneous inspiratory demand is therefore much higher than the patient's average minute ventilation. A low supply flow empties the mask or reservoir during the inspiratory peak and pulls room air through mask leaks. A 20 L/min fresh-gas flow rapidly replenishes the reservoir, meets peak demand and prevents entrainment of nitrogen-rich room air, preserving a high inspired oxygen concentration.	MOLECULAR MECHANISM Fentanyl activates mu-opioid Gi/o-coupled receptors, opens potassium channels and reduces presynaptic calcium influx, suppressing nociceptive neurotransmitter release. Midazolam positively modulates the benzodiazepine site of GABA-A receptors and increases the frequency of chloride-channel opening, producing anxiolysis, amnesia and hypnosis. Combined treatment provides reliable comfort, immobility and amnesia for positioning and needle placement. ADMINISTRATION AND MONITORING After five minutes of preoxygenation, administer the adult protocol total of fentanyl 100 micrograms IV and midazolam 2 mg IV sequentially under direct anaesthesia supervision. Continuously observe respiratory rate, airway patency, EtCO2 waveform, SpO2, blood pressure, heart rate and level of consciousness. Capnography identifies hypoventilation before SpO2 falls. Keep bag-mask ventilation, suction, oral/nasal airways, naloxone and flumazenil immediately available and continue monitoring until ventilation and responsiveness return to baseline.
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KEY SOURCES: ASA moderate sedation guideline [28]; fentanyl and midazolam pharmacology [29,30]; preoxygenation physiology [38,39].

SECTION 09

Regional anaesthesia: complete technique platform

Regional anaesthesia blocks nociceptive transmission at its source. A successful block produces dense surgical-site analgesia, preserves ventilation and cognition, reduces nausea and opioids, and makes coughing, feeding and walking achievable immediately after surgery.

LOCAL ANAESTHETIC CEILING	Bupivacaine: 2.5 mg/kg (max 175 mg) plain; 3 mg/kg (max 225 mg) with epinephrine
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COMPREHENSIVE TECHNIQUE CATALOGUE

Neuraxial: spinal, epidural, combined spinal-epidural, continuous spinal, caudal. Head/neck: scalp, cervical plexus, trigeminal terminal branches, maxillary and mandibular blocks. Upper limb: interscalene, supraclavicular, infraclavicular, axillary, suprascapular, axillary nerve, and individual radial/median/ulnar/musculocutaneous blocks. Thorax/breast: thoracic epidural, paravertebral, erector spinae plane, intercostal, serratus anterior plane, PECS I/II.

ABDOMEN, PELVIS AND LOWER LIMB

Abdominal wall: TAP (subcostal, lateral, posterior), quadratus lumborum I/II/III, rectus sheath, transversalis fascia, ilioinguinal/iliohypogastric, wound infiltration and continuous wound catheter. Pelvis/perineum: sacral plexus, pudendal, paracervical and local infiltration. Lower limb: lumbar plexus/psoas, fascia iliaca, femoral, adductor canal/saphenous, obturator, sciatic (anterior, transgluteal, subgluteal, popliteal), IPACK, genicular, ankle and terminal nerve blocks.

MOLECULAR MECHANISM

Bupivacaine enters the axon, becomes protonated and binds the intracellular site of voltage-gated sodium channels. It stabilizes the inactivated channel state and prevents action-potential propagation. Small myelinated pain fibers are blocked before larger motor fibers. By stopping afferent traffic before and during incision, the block prevents dorsal-horn wind-up and reduces the neuroendocrine stress response.

ADJUNCTS AND EXECUTION

Use ultrasound, incremental injection and continuous monitoring. Epinephrine 1:200,000 (5 micrograms/mL) produces local vasoconstriction, slows systemic uptake and prolongs block duration. Preservative-free dexamethasone 4 mg added perineurally prolongs many peripheral blocks; IV dexamethasone 8 mg provides systemic analgesic and antiemetic benefit. Calculate the total bupivacaine dose across every injection and infiltration before the first needle enters the patient.

KEY SOURCES: Maximum-dose reference [15]; ERAS anaesthesia consensus [16]; procedure-specific regional analgesia evidence [17].

SECTION 10

Anaesthesia monitoring, including bispectral index

Anaesthesia monitors measure circulation, oxygenation, ventilation, temperature, neuromuscular function and cerebral activity. These signals are interpreted together to identify deterioration or inadequate anaesthetic depth immediately.

INTEGRATED STANDARD	ECG, NIBP, SpO2, EtCO2, gases, temperature, quantitative TOF and BIS 40-60
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ECG AND BLOOD PRESSURE ECG: Lead II emphasizes P waves and atrial rhythm; V5 improves lateral ST surveillance. Correlate electrical activity with the pulse and pressure. NIBP: Oscillometry derives pressure from cuff pulsations. Cuff-bladder width equals about 40% of arm circumference and length encircles 80-100%. A small cuff reads high; an oversized cuff tends to read low. PULSE OXIMETRY The probe transmits 660-nm red and 940-nm infrared light. Deoxyhemoglobin absorbs more red; oxyhemoglobin absorbs more infrared. The ratio of pulsatile to nonpulsatile absorption produces arterial SpO2. Verify the plethysmographic waveform. Motion, poor perfusion, probe position, nail products, ambient light and dyshemoglobins distort accuracy. CAPNOGRAPHY, GASES AND TOF Capnography confirms ventilation, airway continuity, respiratory rate and rebreathing. Gas analysis measures inspired/expired O2, CO2 and volatile agent; EtO2 >=80% confirms preoxygenation, with 90% as the target. Quantitative TOF uses four stimuli at 2 Hz: 0/4 confirms complete block when required for intubation, and T4/T1 must be >=0.90 before extubation.	PRINCIPAL EEG BANDS Delta: 0.5-4 Hz - deep N3 sleep and deep anaesthesia. Theta: 4-8 Hz - drowsiness and N1 sleep. Alpha: 8-13 Hz - relaxed wakefulness with eyes closed; propofol and volatile agents move organized alpha activity frontally. Beta: 13-30 Hz - wakefulness and light sedation; facial EMG can falsely increase high-frequency power. SLEEP, ANAESTHESIA AND BIS Awake EEG contains alpha and beta. N1 shifts toward theta; N2 adds 12-15 Hz spindles and K-complexes; N3 is delta-dominant; REM resembles low-amplitude wakefulness. With deepening anaesthesia, beta generally falls as alpha, theta and delta rise. Propofol and volatile surgical hypnosis commonly produces frontal alpha with slow-delta activity. Excessive depth produces burst suppression. HOW BIS ESTIMATES DEPTH BIS converts frontal EEG into a 0-100 index using fast-to-slow wave power ratios, frequency synchronization and burst suppression. More fast-wave power generally raises BIS; synchronized slow activity lowers it. Maintain BIS 40-60 and interpret it with raw EEG, signal quality, EMG and suppression ratio. Drugs, paralysis and artifact can alter the number. TEMPERATURE Maintain core temperature >=36 C. Distal esophageal and correctly placed nasopharyngeal probes are accurate; bladder is useful with adequate urine flow, while rectal and skin sites lag. Hypothermia impairs coagulation and neutrophil bacterial killing, increasing bleeding and infection.
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KEY SOURCES: Monitoring references [41-46]; BIS principles [55,56]; anaesthetic EEG and sleep-frequency relationships [58,59]; processed-EEG outcomes [61].

SECTION 11

Metabolic fresh-gas flow during anaesthetic ventilation

After induction, denitrogenation and circuit equilibration, a circle system is maintained at 0.5 L/min fresh-gas flow. Minimal flow conserves heat and moisture, preserves volatile anaesthetic inside the circuit, lowers cost and sharply reduces operating-room contamination and atmospheric release.

MAINTENANCE FLOW	Total fresh-gas flow 0.5 L/min in a leak-free circle circuit with CO2 absorption
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METABOLIC AND CLOSED-CIRCUIT PHYSIOLOGY An adult consumes approximately 200-300 mL oxygen per minute at rest. In a circle system, exhaled gas passes through the CO2 absorber and is rebreathed; only consumed oxygen, absorbed anaesthetic and unavoidable circuit loss must be replaced. A 0.5 L/min total flow approaches metabolic requirements and produces a near-closed circuit. Continuous inspired oxygen, inspired CO2, end-tidal CO2, agent concentration and circuit-volume monitoring confirms that the system is delivering the intended mixture. HEAT AND HUMIDITY CONSERVATION High fresh-gas flow continuously replaces exhaled warm, water-saturated gas with cold, dry pipeline gas and accelerates airway heat and moisture loss. At 0.5 L/min, most exhaled gas is recirculated after CO2 removal. Moisture and heat generated by the patient and the absorber remain in the circuit, warming and humidifying inspired gas, reducing mucosal desiccation, preserving ciliary function and improving airway comfort. Minimal flow therefore acts as an intrinsic heat-and-moisture conservation strategy.	COST, WASTE AND ENVIRONMENTAL PROTECTION Volatile anaesthetic use is directly proportional to fresh-gas flow, vaporizer setting and time. At high flows, large amounts of sevoflurane, isoflurane or other agent pass through the circuit once and then leave through the scavenging system or leaks. This rapidly drains the vaporizer and wastes one of the operating room's most expensive medication groups. Minimal flow keeps anaesthetic in recirculation, reduces agent consumption and lowers cost. It also reduces waste gas released into the operating room and decreases occupational exposure of nurses, surgeons, technicians and anaesthesia staff. OPERATIONAL SEQUENCE Use high flow for initial circuit wash-in, rapid concentration changes, emergency oxygenation and final washout. Once inspired and end-tidal concentrations are stable, reduce total flow to 0.5 L/min and adjust the vaporizer to maintain the target end-tidal agent/MAC. Continuously verify inspired O2, EtCO2, inspired CO2, end-tidal agent, circuit volume, absorber function and leaks. Use fresh, unexhausted CO2 absorbent approved for the workstation and install the canister correctly to prevent channeling. The circuit recirculates exhaled gas, but the absorbent removes CO2 before reinspiration. Confirm inspired CO2 remains zero. Any inspired CO2, elevated capnogram baseline, absorbent color change or suspected exhaustion requires immediate absorbent replacement to prevent CO2 rebreathing and hypercapnia.
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KEY SOURCES: Low-flow anaesthesia review [47]; minimal-flow airway heat/humidity review [48]; occupational-exposure study [49]; cost and emissions study [50]; OSHA waste-anaesthetic-gas guidance [51].

SECTION 12

Dexamethasone: anti-inflammatory analgesia and nausea prevention

A single 8 mg intravenous dose suppresses the inflammatory surge generated by surgery, reduces postoperative nausea and vomiting, improves pain with movement and accelerates return of gastrointestinal function after major abdominal surgery.

RECOMMENDED DOSE	Dexamethasone 8 mg IV after induction and before incision
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PHARMACOKINETICS Dexamethasone distributes rapidly and has a plasma half-life of 3-5 hours, while genomic effects persist for 36-54 hours. Hepatic metabolism and renal excretion clear inactive metabolites. Administration before incision provides anti-inflammatory activity through the first postoperative day. MOLECULAR MECHANISM Dexamethasone crosses the cell membrane and binds the cytosolic glucocorticoid receptor. The complex enters the nucleus, increases transcription of anti-inflammatory proteins such as annexin A1, and represses NF-kB and AP-1. This decreases phospholipase A2 activity, arachidonic-acid release, COX-2 induction, prostaglandins, bradykinin-related signaling, IL-1, IL-6 and TNF-alpha. Reduced tissue inflammation means less nociceptor sensitization and less pain on movement.	MAJOR ABDOMINAL SURGERY BENEFIT In major abdominal and gynecologic laparotomy, 8 mg dexamethasone reduces 24-hour morphine consumption and pain with movement. In gastrointestinal surgery it shortens time to flatus, decreases abdominal distension and accelerates tolerance of liquids. These effects directly support coughing, ambulation, oral intake and removal of IV therapy. ANTIEMETIC MECHANISM AND PATHWAY POSITION Dexamethasone reduces central and peripheral inflammatory signaling to the vomiting center, modulates serotonin pathways and improves the effect of complementary antiemetics. Give it early because the genomic onset is slower than that of 5-HT3 antagonists. Pair it with another antiemetic class in a risk-based PONV plan.
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KEY SOURCES: De Oliveira et al., Br J Anaesth 2013 [18]; De Oliveira et al., Anesth Analg 2013 [19]; abdominal motility RCT [20]; laparotomy analgesia RCT [21].

SECTION 13

Propofol infusion: hypnosis, opioid sparing and antiemetic protection

Propofol infusion produces controllable hypnosis with rapid recovery and a lower emetogenic burden than volatile anaesthesia. Even a subhypnotic infusion of 30 micrograms/kg/min provides antiemetic exposure while allowing another agent to carry the primary anaesthetic.

DOSE FRAMEWORK	Antiemetic 30; sedation 25-75; general anaesthesia 75-150 micrograms/kg/min
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PHARMACOKINETICS Propofol reaches the brain within one circulation time and redistributes rapidly, producing prompt awakening after a bolus. Hepatic and extrahepatic metabolism form inactive water-soluble metabolites excreted by the kidney. High clearance and a distribution half-life of minutes support rapid titration and recovery. MOLECULAR MECHANISM Propofol positively modulates GABA-A receptors and at higher concentrations directly activates chloride current, hyperpolarizing cortical and subcortical neurons. It suppresses thalamocortical connectivity and consciousness. Antiemetic effects arise from reduced activity in the chemoreceptor trigger zone and nucleus tractus solitarius, modulation of 5-HT3 signaling and lower exposure to volatile agents. Its antioxidant phenol structure also limits lipid-peroxidation signaling.	PRACTICAL INFUSION STRATEGY Use 30 micrograms/kg/min as a subhypnotic antiemetic adjunct during balanced anaesthesia. For monitored sedation, titrate approximately 25-75 micrograms/kg/min to responsiveness and ventilation. For general anaesthesia, commonly use 75-150 micrograms/kg/min with analgesia and neuromuscular management, then step down as surgical stimulation falls. Titrate to clinical signs and processed EEG when available rather than maintaining one fixed rate. RECOVERY IMPACT Propofol reduces early PONV most powerfully when it replaces volatile anaesthesia as TIVA. Subhypnotic bolus and infusion studies support early antiemetic effects, although infusion evidence is mixed; therefore the 30 micrograms/kg/min strategy functions inside a multimodal PONV plan rather than as its only component. Stable dosing prevents awareness, excessive hypnosis, hypotension and delayed awakening.
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KEY SOURCES: Propofol PONV RCT [31]; subhypnotic infusion cohort [32]; consensus PONV guideline [33]; propofol pharmacology [34].

SECTION 14

Magnesium sulfate: analgesia, relaxation and physiologic control

Magnesium is a divalent cation and a high-value intraoperative adjunct. It reduces nociceptive amplification, decreases postoperative pain and opioid exposure, potentiates nondepolarizing neuromuscular blockade, moderates sympathetic hemodynamic responses and relaxes bronchial smooth muscle.

RECOMMENDED REGIMEN	Magnesium sulfate 2 g IV infused over 30 minutes
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CALCIUM ANTAGONISM AND ANALGESIA Magnesium competes with calcium at voltage-gated channels and limits calcium entry into presynaptic nerve terminals. It also produces voltage-dependent antagonism of the NMDA receptor channel. Together, these actions reduce release of excitatory neurotransmitters, suppress dorsal-horn wind-up and central sensitization, and decrease amplification of the surgical pain signal. The result is lower postoperative pain intensity, reduced opioid consumption and a longer interval before rescue analgesia. NEUROMUSCULAR-BLOCK POTENTIATION At the motor nerve terminal, magnesium reduces calcium-dependent acetylcholine release. It also decreases postsynaptic membrane excitability. Nondepolarizing neuromuscular blockers therefore develop a deeper effect, may act more rapidly and remain active longer. Use quantitative train-of-four monitoring, titrate relaxant dosing to the surgical requirement and confirm a T4/T1 ratio of at least 0.90 before extubation. Magnesium administered after apparent recovery can recreate clinically important weakness.	HEMODYNAMIC CONTROL Magnesium reduces calcium-dependent contraction in vascular smooth muscle, promotes peripheral vasodilation and attenuates catecholamine release. Heart rate and arterial pressure fall toward a controlled range, and the sympathetic surges associated with laryngoscopy, intubation and surgical stimulation are blunted. Infuse the 2 g dose over the full 30 minutes; rapid administration produces unnecessary hypotension and bradycardia. BRONCHIAL SMOOTH-MUSCLE RELAXATION Magnesium decreases calcium entry into airway smooth muscle, limits acetylcholine release from cholinergic nerve endings and reduces mast-cell histamine release. Bronchial smooth muscle relaxes, airway resistance falls and bronchospastic tone is reduced during endotracheal-tube or laryngeal-mask airway management. Magnesium strengthens a complete airway plan that still includes adequate depth, gentle instrumentation, oxygenation and standard bronchodilator treatment when bronchospasm occurs. CONTRAINDICATIONS AND INTRAOPERATIVE CONTROL Do not administer routine magnesium to a patient with significant bradycardia, hypotension, advanced heart block, severe renal impairment, hypermagnesemia or myasthenia gravis. Correct hemodynamic instability first. Magnesium potentiates nondepolarizing relaxants and can cause prolonged or recurrent paralysis; establish quantitative train-of-four monitoring before infusion, reduce additional relaxant dosing as indicated and confirm T4/T1 at least 0.90 before extubation. Monitor ECG, blood pressure, heart rate, oxygenation and neuromuscular recovery throughout administration.
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KEY SOURCES: Perioperative analgesia umbrella review [63]; anaesthesia review [64]; neuromuscular-block meta-analysis [65]; airway smooth-muscle and asthma evidence [66].

SECTION 15

Goal-directed, balanced fluid care

Fluid therapy maintains perfusion without creating edema. Preoperative oral intake removes the routine fasting deficit, so IV fluid replaces basal physiologic need, measured blood loss and demonstrated volume responsiveness - not an assumed need for liters of crystalloid.

STARTING FRAMEWORK	Balanced crystalloid 1-3 mL/kg/h; 200-250 mL bolus only for a tested indication
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WHAT IS REPLACED Use a balanced crystalloid to cover modest insensible and urinary losses during routine surgery. Replace measured blood loss initially with targeted balanced crystalloid while evaluating hemoglobin, coagulation, perfusion and ongoing loss; use blood components when oxygen-carrying capacity or hemostasis requires them. Replace external gastrointestinal, drain and third-space losses according to their measured volume and composition. Oral intake before surgery substantially reduces the IV volume needed for fasting. GOAL-DIRECTED DECISION CYCLE Assess blood pressure trend, pulse, capillary refill, skin temperature, mental status, lactate when available and surgical field loss. In major surgery, use stroke volume, pulse-pressure variation or another validated flow variable. Give a 200-250 mL bolus only when the clinical picture suggests preload dependence, then continue fluid only if stroke volume or perfusion improves. Treat vasodilation with vasopressor after euvolemia is established.	WHY BALANCED SOLUTION Lactated Ringer's or another balanced crystalloid provides a chloride concentration closer to plasma and reduces hyperchloremic metabolic acidosis compared with large-volume 0.9% saline. The objective is a near-zero to slightly positive perioperative balance, stable perfusion, low lactate, warm extremities and no clinically important weight gain from salt and water. THE COST OF OVERHYDRATION Excess crystalloid expands the interstitial compartment and produces bowel-wall, lung, wound and peripheral edema. Bowel edema delays motility and oral intake; pulmonary edema worsens oxygen exchange; tissue edema impairs diffusion and wound healing. Gastric and intestinal edema promote nausea, vomiting and delayed emptying, increasing regurgitation and aspiration potential. Dilution worsens coagulation and hemoglobin concentration. Stop routine IV fluid as oral drinking becomes adequate.
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KEY SOURCES: ERAS gastrointestinal anaesthesia consensus [16]; ERAS LMIC guideline [24]; 2025 colorectal ERAS update [35]; ASCRS/SAGES guideline [36].

SECTION 16

Postoperative care I: early oral intake

Drinking and feeding restart within hours of surgery. Early oral intake restores normal gastrointestinal physiology, supplies substrate for healing, ends unnecessary IV therapy and becomes a measurable recovery milestone rather than a reward for bowel sounds.

RECOVERY TARGET	Clear sips in PACU; food within 4-6 hours; oral nutrition within 24 hours
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PHYSIOLOGIC BENEFITS Luminal nutrients stimulate vagal and enteric activity, gut hormones, bile flow and intestinal motility. Enteral feeding preserves mucosal structure, tight-junction integrity and splanchnic perfusion, limiting bacterial translocation. Carbohydrate supports ATP production and reduces proteolysis; protein supplies amino acids for collagen, immune proteins and muscle repair. Oral water and electrolytes restore circulating volume and permit IV discontinuation. METABOLIC AND FUNCTIONAL RECOVERY Early intake attenuates postoperative insulin resistance and nitrogen loss, supports glucose use by healing tissues and improves patient strength. Effective regional analgesia, dexamethasone, multimodal PONV prophylaxis and balanced fluid care remove the principal barriers to drinking. The patient experiences less thirst, less weakness and a faster return to normal behavior.	STEPWISE PROTOCOL In PACU, begin 30-50 mL clear fluid when awake with protective reflexes and controlled nausea. Advance to repeated sips, then a light meal according to the operation. Provide protein-rich oral nutrition within 24 hours for major surgery. Document time to first drink, total oral volume, time to first food and nausea score. Discontinue routine IV maintenance when oral intake meets hydration needs. ABDOMINAL SURGERY Early feeding after abdominal and pelvic surgery accelerates return of bowel function and shortens hospitalization without increasing anastomotic leak. Combine it with opioid-sparing analgesia, avoidance of routine nasogastric tubes, chewing and walking. Abdominal distension or nausea triggers evaluation and treatment; it does not reinstate indefinite fasting by default.
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KEY SOURCES: ERAS LMIC abdominal/pelvic guideline [24]; emergency laparotomy ERAS [37]; 2025 colorectal ERAS recommendations [35].

SECTION 17

Postoperative care II: early mobilization and walking

The patient sits, stands and walks on the day of surgery. Walking is an active physiologic treatment that restores ventilation, circulation, insulin sensitivity, gastrointestinal activity and functional independence.

MOBILIZATION TARGET	Sit out of bed 30 minutes on day 0; begin walking day 0-1; build toward 3 hours/day
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PHYSIOLOGIC BENEFITS Upright posture increases functional residual capacity and improves dependent-lung ventilation. Deep breathing and walking recruit alveoli and mobilize secretions. Calf-muscle pumping increases venous return and reduces venous stasis. Weight bearing preserves muscle protein synthesis, strength, balance and joint function. Movement stimulates intestinal motility and improves skeletal-muscle glucose uptake through contraction-mediated GLUT4 translocation, reducing insulin resistance. FUNCTIONAL SEQUENCE Once awake and hemodynamically stable: elevate the head of bed, sit upright, dangle legs, transfer to a chair, stand with assistance and walk a defined distance. Repeat short walks rather than relying on one prolonged effort. By postoperative day 1, set explicit time-out-of-bed and corridor-walking targets and record achievement at every nursing shift.	MAKE ANALGESIA SERVE MOVEMENT Pain management succeeds when the patient can cough, breathe deeply, rise from a chair and walk. Regional blocks, Acycor Plus, dexamethasone and selected gabapentin reduce movement-evoked pain. Rescue opioids restore function without creating avoidable sedation. Remove urinary catheters, IV lines, drains and nasogastric tubes early so equipment does not become the reason the patient remains in bed. SYSTEM DESIGN Explain walking targets before surgery and display them at the bedside. Provide non-slip footwear, a gait aid, staff assistance and a documented fall-risk assessment. Track time to first chair, first stand and first walk. Prolonged bed rest is treated as a clinical variance because it accelerates muscle loss, pulmonary complications, thromboembolism and dependence.
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KEY SOURCES: Emergency laparotomy ERAS [37]; ERAS LMIC guideline [24]; 2025 colorectal ERAS recommendations [35].

SECTION 18

The ERAS Nigeria challenge: every public hospital, every state

Every state will vigorously pursue implementation of the fundamental principles of Enhanced Recovery After Surgery in every public hospital as official hospital policy. Success means a written protocol, trained personnel, reliable supplies, bedside execution and continuous measurement - not an informal recommendation that is used only when convenient.

NATIONAL CHALLENGE	Every public hospital adopts, implements and audits the complete ERAS pathway
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THE ACCOMPLISHMENT AWARD Daniel Tapia will personally spend NGN 1 billion from his personal salary to purchase 1,000 fully equipped intubation mannequins and airway-equipment cases directly from factories in China. Beginning January 1, 2027, Daniel Tapia commits 90% of his personal salary after payment of all required taxes to this project until the NGN 1 billion commitment is fulfilled. This investment will allow 1,000 anesthesiologists at qualifying public hospitals to receive one complete mannequin and equipment case each, free of charge. Every case includes a videolaryngoscope with Macintosh size 3 and size 1 blades, laryngeal mask airways, oral airways, an intravenous-fluid pressure bag, a stethoscope and additional airway-training equipment. REQUIRED COMMITMENT Before receiving the free package, each recipient anesthesiologist must personally spend NGN 1,000,000 on coconut water for patients before and after surgery and must conduct and document airway training every week. Coconut water should ideally be obtained through the official Enhanced Recovery After Surgery coconut-processing facility. Within one year after receipt, the recipient anesthesiologist must donate the mannequin and every item of airway equipment to their hospital for permanent institutional use. Programme leaders will verify ERAS implementation, coconut-water expenditure, weekly training and completed hospital donation.	HOSPITAL POLICY CHECKLIST - PREPARATION [] ERAS policy approved by hospital leadership [] Multidisciplinary ERAS team and accountable lead appointed [] Preoperative holding bay administers the protocol on time [] Melatonin, two Acycor Plus tablets and gabapentin 600 mg incorporated as indicated [] Coconut-water hydration and sips until one hour before surgery adopted [] Regional-anaesthesia techniques and local-anaesthetic dosing standardized [] Dexamethasone 8 mg included when indicated [] Five-minute preoxygenation and expired oxygen at least 80% documented HOSPITAL POLICY CHECKLIST - ANAESTHESIA AND RECOVERY [] Preprocedural sedation, monitoring and airway readiness standardized [] Magnesium infusion, propofol infusion and multimodal analgesia implemented [] Goal-directed balanced fluids replace measured losses without overhydration [] Early postoperative oral intake begins promptly when clinically appropriate [] Early mobilization and walking are prescribed and documented [] Recipient performs weekly documented airway training [] Recipient spends NGN 1,000,000 on perioperative coconut water [] Recipient donates the mannequin and all equipment to the hospital within one year [] Quantitative monitoring, normothermia and BIS are used as specified [] Low-flow anaesthesia uses fresh CO2 absorbent and zero inspired CO2 [] A standardized airway cart is present wherever an airway is managed [] Outcomes and compliance are audited
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KEY SOURCES: Challenge requirements consolidate the clinical and operational standards established in Sections 1-17. Equipment eligibility and distribution remain subject to written programme verification and agreement.

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