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# **ANAESTHESIA FOR CARDIAC HYDATID CYST SURGERY: NAVIGATING THE DUAL THREAT OF ANAPHYLAXIS AND CARDIOGENIC COLLAPSE A NARRATIVE REVIEW**

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## **ABSTRACT**

Cardiac echinococcosis accounts for fewer than 2% of hydatid disease, yet concentrates two feared perioperative catastrophes into one operation: parasitic anaphylaxis from antigen spillage, a distributive shock treated with epinephrine and volume; and cardiogenic or obstructive collapse from a mass lesion contacting the myocardium and coronary circulation, in which volume may be futile or harmful. Both present as sudden hypotension under conditions — drapes, general anaesthesia, heparinisation, extracorporeal circulation — that remove the cues normally used to tell them apart. The surgical literature is well developed; the anaesthetic literature consists of isolated case reports. This narrative review examines the immunology and true incidence of parasitic anaphylaxis, its recognition and treatment on cardiopulmonary bypass, the physiology of a compressive extracardiac mass, the discriminating role of transoesophageal echocardiography, the choice of perfusion strategy, and the overlooked systemic toxicity of scolicedal agents. A practical checklist is proposed.

## **KEYWORDS**

cardiac echinococcosis, hydatid cyst, perioperative anaphylaxis, cardiopulmonary bypass, transoesophageal echocardiography, cardiac anaesthesia

## MAIN ARTICLE

### INTRODUCTION

Cystic echinococcosis remains endemic across the Mediterranean basin, North Africa, the Middle East, Central Asia and South America, and patients from those regions present for surgery worldwide. The liver and lungs account for roughly 90 % of localisations; cardiac involvement is reported in fewer than 2 % of patients [1, 2, 3]. Because the left ventricle receives the richest coronary supply it is the most frequently involved chamber, followed by the right ventricle, the interventricular septum and the atria [2, 4, 5]. In the largest systematic analysis of the published literature, the majority of patients are symptomatic at presentation, most commonly with dyspnoea, chest pain or palpitations [3].

Surgical excision under cardiopulmonary bypass (CPB) is the accepted treatment and has been described in detail [2, 6]. The anaesthetic literature, by contrast, is sparse: a handful of case reports, no cohort data, and no consensus statement. This is a substantive gap, because the anaesthesiologist confronts a problem qualitatively different from routine cardiac surgery. Two entirely different catastrophes must be held in mind at once.

The first is **allergic**: anaphylactic shock from release of highly antigenic cyst content. It is distributive in mechanism, may occur without warning, may be precipitated by the surgeon's own manipulation, and is treated with epinephrine and aggressive volume expansion.

The second is **cardiogenic or obstructive**: collapse from a compressive mass lesion, ventricular wall failure, coronary compromise or arrhythmia, which the volume loading that treats the first may worsen.

Both present with the same initial sign — sudden, profound hypotension — under conditions that remove most of the bedside cues normally used to tell them apart: surgical drapes, general anaesthesia, systemic heparinisation and extracorporeal circulation. This review is organised around that diagnostic problem, around the tools that resolve it, and around what a team can prepare in advance.

### SCOPE AND SOURCES

This is a narrative rather than a systematic review. The literature was identified by searching PubMed and Embase for combinations of *cardiac hydatid*, *cardiac echinococcosis*, *hydatid* with *anaesthesia*, *anesthesia*, *anaphylaxis*, *cardiopulmonary bypass* and *transoesophageal echocardiography*, without date limits, supplemented by hand-searching the reference lists of

retrieved articles and of two recent systematic reviews of cardiac cystic echinococcosis [3, 5]. Because no comparative anaesthetic studies exist in this disease, the review necessarily draws on three distinct bodies of evidence: reports of cardiac hydatid disease, which supply the anatomy and the surgical constraints; the general literature on perioperative anaphylaxis, which supplies the epidemiology and the treatment algorithms; and the cardiac anaesthesia literature, which supplies the haemodynamic and monitoring principles. Where a recommendation rests on extrapolation between these bodies rather than on direct evidence, we have tried to say so.

## **THE ALLERGIC AXIS**

### **An unusual allergen, and an unpredictable one**

The hydatid cyst is not merely a space-occupying lesion but a reservoir of highly immunogenic material held under pressure behind a fragile membrane. Chronic exposure to *Echinococcus granulosus* antigens — antigen B in particular — drives a Th2-polarised response with production of parasite-specific IgE and IgG4, while the parasite simultaneously inhibits neutrophil chemotaxis and interferes with complement activation, permitting prolonged coexistence and progressive sensitisation of the host [7].

A patient may therefore harbour a substantial burden of specific IgE bound to mast cells and basophils while remaining entirely asymptomatic, with a normal eosinophil count and possibly negative serology. Breach of the cyst wall — spontaneous, traumatic, or surgical — delivers a bolus of antigen into a vascular bed already primed to respond. Both IgE-mediated degranulation and direct, non-IgE complement-mediated mechanisms are implicated, which explains reactions in patients without demonstrable specific IgE.

Two corollaries follow, and both matter more than they are usually given credit for.

**No preoperative test predicts who will react.** Neither eosinophil count, nor serology, nor a history of previous uneventful hydatid surgery excludes the possibility. Serology in particular is frequently negative in isolated cardiac involvement and cannot be used to stratify allergic risk. A history of previous hydatid surgery without incident is often treated as reassuring; it is not. It documents tolerance of anaesthesia, not tolerance of antigen, and through prior sensitisation may theoretically increase risk.

**Risk is not confined to gross rupture.** Anaphylaxis has been described with intact, non-ruptured cysts, presumably through microleakage across a permeable membrane, and during needle biopsy of cardiac, hepatic and subcutaneous cysts [8]. Preparing only for the moment

of deliberate surgical incision is therefore insufficient: the risk window opens with sternal retraction and pericardial manipulation.

### **Incidence: a necessary correction**

Narrative reviews and the anaesthetic case-report literature commonly quote rates of intraoperative anaphylaxis during hydatid surgery of 0.2–3.3 % [9]. These figures circulate widely but derive largely from small retrospective series and are vulnerable to publication bias, since uneventful operations are not reported.

The best available systematic evidence tempers them. Reviewing 124 publications covering 5,943 percutaneous treatment procedures on 5,517 echinococcal cysts, Neumayr and colleagues identified 99 reversible anaphylactic reactions and two lethal events — reversible allergic reactions complicating 1.7 % of procedures and lethal anaphylaxis 0.03 % [10]. Their conclusion, that the risk of severe anaphylaxis in cyst puncture has been widely exaggerated and may be lower than that following administration of certain antibiotics, is an important corrective to the tone of much of the surgical literature.

We draw two conclusions rather than one. Intellectual honesty requires acknowledging that catastrophic anaphylaxis is rarer than the case-report literature implies, and that fear of it should not by itself drive a patient towards a more morbid operative strategy. But a 1.7 % rate of clinically significant allergic reaction remains one to two orders of magnitude above the background incidence of perioperative anaphylaxis from all causes, which national audit data place near 1 in 10,000 anaesthetics [11]. For the anaesthesiologist that is the operative number: it makes anaphylaxis a realistic event to be rehearsed, while stopping short of justifying prophylaxis of unproven benefit.

Two caveats limit the transfer of these data. Neumayr's cohort concerns percutaneous puncture, predominantly of hepatic cysts, under conditions of controlled aspiration; open surgical excision with manipulation and possible uncontrolled rupture is a different exposure. And data specific to *cardiac* hydatid surgery do not exist in usable form. Whether cardiac localisation carries higher or lower risk than hepatic or pulmonary disease is unknown: the cardiac cyst lies in immediate contact with the systemic circulation, which favours rapid and massive antigen delivery, but cardiac cysts are typically smaller than hepatic ones and the operation proceeds under conditions of controlled perfusion. This uncertainty should be stated plainly rather than resolved by assertion.

### Preoperative risk reduction

**Albendazole** reduces protoscolex viability and intracystic pressure and is recommended by international expert consensus [12]. Evidence favours the longer course: in a prospective comparison, 5 % of patients treated for two weeks had viable cysts at operation versus none of those treated for four weeks [13, 14]. Critically, albendazole reduces the *parasitological* consequences of spillage — secondary echinococcosis — but there is no evidence that it abolishes the antigenicity of cyst fluid. It is not allergological prophylaxis and should not be presented as such.

**Antihistamine and corticosteroid premedication** is recommended in several case reports [8], but the evidence base is weak. The strategy is extrapolated from radiocontrast prophylaxis, where its efficacy is itself contested, and has never been tested prospectively in hydatid disease. Two objections deserve consideration. Premedication may attenuate early cutaneous and bronchospastic signs without preventing cardiovascular collapse, delaying recognition in a patient whose skin is already hidden beneath drapes. And it may generate false reassurance. Our view is that premedication is defensible as a low-cost adjunct, but must never substitute for preparedness: a premedicated patient is not a protected patient.

**The team briefing** has the strongest rationale and the lowest cost. Perioperative anaphylaxis is a diagnosis of pattern recognition under time pressure; shared expectation shortens time to recognition more effectively than any drug. The anaesthesiologist should state the anticipated risk, the surgeon should state when the highest-risk manoeuvre will occur, and both should agree on a verbal trigger phrase for suspected anaphylaxis.

### The recognition problem

Recognition is genuinely difficult, and every feature of the operative environment compounds the difficulty.

**Under general anaesthesia**, the classical prodrome — pruritus, flushing, dyspnoea, a sense of impending doom — is unavailable. National audit data indicate that hypotension requiring vasopressor support is usually the *first* sign of perioperative anaphylaxis, and that rash, the classical marker, is visible in only about half of cases and may be delayed, particularly in the most severe reactions [11]. In cardiac surgery the skin is almost entirely covered.

**In cardiac surgery specifically**, the differential diagnosis of sudden hypotension is long and mostly not allergic: surgical manipulation of the heart, arrhythmia, haemorrhage, mechanical compression of a chamber by the cyst itself, air embolism, and protamine reaction — the last

a recognised and sometimes catastrophic cause of collapse at a predictable moment of the operation [15]. Bronchospasm, a relatively specific sign, is masked when the lungs are deflated on full CPB.

**On cardiopulmonary bypass**, the diagnosis becomes harder still. Systemic vascular resistance falls, but vasoplegia after initiation of bypass is common and has multiple non-allergic causes [16]. Pulse contour and pulse oximetry are absent or unreliable during non-pulsatile flow. Capnography reflects pump gas flow rather than pulmonary perfusion. Haemodilution alters tryptase and histamine kinetics. Cutaneous signs remain hidden.

Certain features nonetheless favour anaphylaxis and should be actively sought:

- Profound hypotension with a **low** systemic vascular resistance in the presence of preserved or hyperdynamic ventricular function on transoesophageal echocardiography (TOE) — a distributive, not cardiogenic, pattern.
- On CPB, a fall in systemic pressure at constant pump flow, requiring escalating vasopressor doses, with no surgical explanation.
- A sudden fall in venous reservoir level reflecting capillary leak and third-spacing, often noticed by the perfusionist before the anaesthetist.
- Rising peak airway pressure or an obstructive capnographic waveform when the lungs are ventilated.
- Facial, conjunctival or oropharyngeal oedema, or urticaria on any exposed skin — worth a deliberate look under the drapes rather than an assumption.
- **Temporal association with cyst manipulation**, which is a strong clue in this operation and the reason the surgeon should announce puncture aloud.

### Treatment and escalation

Management follows established perioperative anaphylaxis guidance [17, 18], graded by the Ring and Messmer classification [19], adapted to the cardiac surgical context.

**Immediate measures.** Halt cyst manipulation, give 100 % oxygen, discontinue the volatile agent if a vasodilatory contribution is suspected, and give **epinephrine without delay** — approximately 10–20 µg intravenously for grade II reactions, 50–100 µg for grade III, and 0.5–1 mg for grade IV or cardiac arrest, repeated and escalated according to response with early transition to an infusion. Under-dosing and delay, not over-dosing, are the errors that determine outcome. Aggressive volume expansion is required concurrently; the capillary leak of severe anaphylaxis can sequester a substantial fraction of the circulating volume within minutes.

**When on CPB**, anaesthesiologist and perfusionist must act jointly. Pump flow can be increased to sustain systemic perfusion pressure while pharmacological therapy takes effect, and the venous reservoir provides an immediate volume source. This is, paradoxically, the safest moment for anaphylaxis to occur: extracorporeal circulation guarantees perfusion during a period in which the native circulation would otherwise fail. The corollary — that anaphylaxis occurring *before* cannulation or *after* weaning is considerably more dangerous — argues for keeping the circuit primed and the cannulation sites accessible throughout, rather than only during the period of planned bypass.

**Refractory shock.** Add norepinephrine and vasopressin, the latter acting through a receptor pathway independent of adrenergic desensitisation. Consider **methylene blue** (1.5–2 mg/kg intravenously over 20 minutes, or added to the CPB prime, with infusion if required), a guanylate cyclase inhibitor reported effective in vasoplegia of both post-bypass and anaphylactic origin [20]; it is a salvage agent, not a first-line one, contraindicated in glucose-6-phosphate dehydrogenase deficiency and to be avoided with serotonergic drugs. Consider glucagon in beta-blocked patients. Refractory anaphylaxis unresponsive to repeated adrenaline has been described in hydatid surgery specifically and carries high mortality [9].

Consider **extracorporeal life support** early. In cardiac surgery this is an unusually attainable rescue, because the patient is already heparinised, the chest is open, and cannulae are in place or immediately placeable. Veno-arterial extracorporeal membrane oxygenation has been reported to salvage refractory perioperative anaphylactic shock and anaphylactic cardiac arrest with neurologically intact survival [21]. In this operation, unlike most, the transition from failed pharmacological therapy to mechanical support can be measured in minutes rather than tens of minutes, and the decision threshold should be correspondingly lower.

Corticosteroids and antihistamines have no role in acute resuscitation and must never delay epinephrine; they may reduce the probability of a biphasic reaction and are given once the patient is stabilised.

### **After the event**

The diagnostic opportunity is time-limited and easily lost. Serum tryptase should be sampled at approximately 1–2 hours after the reaction, at 4 hours, and a baseline sample at 24 hours or later; the diagnosis rests on the *rise* relative to the individual baseline rather than on a single absolute value, and haemodilution on CPB may blunt the measured concentration [17, 18].

The anaesthetic record must document the timing of every drug administered relative to the timing of cyst manipulation. The distinction between a neuromuscular blocking agent reaction and a parasitic reaction cannot be reconstructed later without that timeline, and neuromuscular blocking agents remain the leading cause of perioperative anaphylaxis in national series [22]. Referral for allergological investigation should specify two questions rather than one: which anaesthetic agent, antiseptic or device material was responsible, and whether parasite-specific IgE is present. The second governs the risk of any future hydatid surgery in a patient whose disease is prone to recurrence, and it will not be sought unless it is asked for.

## **THE CARDIOGENIC AXIS**

### **Physiology and induction**

A large cyst adjacent to the ventricle behaves as a localised, chronic, extrinsic compressive lesion. Consequences depend on which structure is compressed and include impaired diastolic filling, regional wall motion abnormality, coronary compression, arrhythmia, embolisation of daughter vesicles on rupture into a cardiac chamber [23], and tamponade on rupture into the pericardium [4, 24]. Isolated intramyocardial cysts, particularly those involving the interventricular septum, may compromise haemodynamics purely by virtue of their anatomical relationships [25].

The essential point for the anaesthesiologist is that such a ventricle is **preload-dependent and afterload-sensitive**. Filling is mechanically limited, stroke volume cannot be augmented indefinitely by raising filling pressures, and cardiac output becomes rate-dependent within a narrow window — an attenuated, localised form of tamponade physiology.

Induction goals follow directly and are deliberately conservative. Maintain preload, correcting hypovolaemia and considering modest volume loading beforehand where the lesion is compressive. Avoid abrupt falls in systemic vascular resistance by titrating slowly with a vasopressor infusion already running rather than giving a large induction bolus. Maintain sinus rhythm and an adequate rate, since bradycardia is poorly tolerated when stroke volume is fixed and new atrial fibrillation may cause abrupt decompensation. Preserve coronary perfusion pressure where the lesion abuts a coronary artery. Anticipate an exaggerated fall in venous return with the transition to positive-pressure ventilation. Apply external defibrillator pads before draping, since arrhythmia may be provoked by manipulation, by the lesion itself, or by the subsequent repair.

Where imaging demonstrates severe compression, the logic that governs induction in cardiac tamponade applies. Options in ascending order of invasiveness are: arterial line and vasopressor infusion established awake; the surgical team scrubbed and the patient prepared and draped before induction; femoral vascular access secured under local anaesthesia; and femoro-femoral partial bypass instituted before induction. The principle is that loss of spontaneous ventilation and sympathetic tone should not precede the ability to support the circulation.

### **Transoesophageal echocardiography**

TOE is the decisive monitor in this operation, and its role extends well beyond confirmation of the preoperative diagnosis. It is the tool that answers, in real time, the question that defines management: *is this hypotension distributive or cardiogenic?* Its value in the perioperative management of cardiac hydatid disease has been reported directly [26]. The study should follow the comprehensive examination protocol recommended by the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists [27], with the following case-specific emphases.

**Before bypass:** cyst dimensions, wall thickness and anatomical relations; the compressive effect on chamber filling; baseline biventricular function and regional wall motion, which becomes the reference against which post-excision function is judged; exclusion of pericardial effusion; and assessment of valvular competence, since a lesion distorting an annulus or subvalvular apparatus may produce regurgitation apparent only under anaesthesia.

**During excision:** immediate detection of new regional wall-motion abnormality; detection of intracavitary daughter vesicles or debris should the cyst communicate with a chamber; and detection of air or particulate embolisation.

**After excision:** confirmation of complete evacuation and absence of residual cavity or collection — reported as a principal indication for intraoperative TOE in this disease [26]; assessment of the repair for paradoxical systolic bulging, which signals a mechanically inadequate closure; comparison of regional wall motion in the territory of any adjacent coronary artery; and quantification of ventricular function to guide inotropic support during weaning.

**During any haemodynamic crisis:** the distinction between a hyperdynamic, empty, vasoplegic ventricle and a dilated, poorly contracting or compressed one is available within

seconds and directly determines whether the correct response is epinephrine and volume or a change in surgical strategy. No other monitor provides this discrimination in this setting.

### **On-pump, beating-heart or off-pump?**

Three strategies are described, and the choice is a genuine trade-off with anaesthetic consequences.

**Full CPB with aortic cross-clamping and cardioplegic arrest** offers a still, bloodless field and maximal control against spillage into a cardiac chamber. Its costs are ischaemia–reperfusion injury, a longer bypass time, and loss of the ability to assess contractile function during the critical part of the operation.

**CPB on a beating, perfused heart without cross-clamping** is a middle path, and has been used successfully for apical left ventricular lesions in contact with the distal left anterior descending artery [28]. Perfusion is guaranteed, the circulation is supported through the moment of maximal allergic risk, myocardial ischaemia is avoided, and — a point we consider underappreciated — the contracting heart remains available for continuous TOE assessment of regional function during excision and repair. For an apical, extracardiac lesion adjacent to a coronary artery, this combination is attractive.

**Off-pump resection**, reported for accessible extracardiac cysts, avoids bypass entirely with its attendant coagulopathy, inflammatory activation and vasoplegia, and has been advocated partly on the grounds that it removes CPB-related contributions to haemodynamic instability [29]. The anaesthetic objection is significant: a patient who develops anaphylaxis or ventricular rupture off pump has no circulatory backup, and cannulation must then be achieved in a collapsed or arrested circulation. Where off-pump resection is chosen, we would argue that the groin should be prepared and draped and the circuit primed as a matter of routine, so that the absence of backup is a deliberate and reversible choice rather than a structural one.

The choice should be made jointly and stated explicitly in the preoperative briefing, because it determines what rescue options the anaesthesiologist has at each phase of the operation.

### **Coronary jeopardy and the repaired ventricular wall**

Coronary compromise is well described in this disease: extrinsic compression of a coronary artery by a pericardial cyst causing narrowing [24], and myocardial ischaemia in young patients requiring bypass grafting [30]. A cyst lying against a coronary vessel without preoperative evidence of ischaemia is a favourable but not risk-free configuration, since the vessel remains liable to injury, kinking or inclusion in a suture during closure of the cavity [28].

Three measures follow. Continuous two-lead ST-segment analysis should be running with its baseline documented. Regional wall motion in the territory at risk must be compared by TOE against the pre-bypass baseline before the chest is closed, since a new abnormality after repair is more likely to reflect the repair itself than a spontaneous event, and is far easier to address before closure. And the concern should be communicated as a specific handover item, so that postoperative ST change or troponin elevation is interpreted against a defined anatomical question rather than dismissed as routine post-cardiotomy drift. Recurrence of cardiac hydatid disease, including with pericardial protrusion, is described, and reinforces the value of a documented baseline for later comparison [31].

Evacuation of an intramural or adherent cyst also leaves a thinned segment of ventricular wall, and closure in layers is performed specifically to prevent aneurysmal dilatation [28]. Two consequences follow. In the immediate post-bypass period the repaired segment is the weakest point of the ventricle: systolic hypertension should be avoided, and the resulting tension between protecting the suture line and perfusing the myocardium should be resolved with the surgeon as a stated target pressure range rather than a general intention. And a suture line in ventricular myocardium is arrhythmogenic: ventricular ectopy should be anticipated in the first postoperative days, magnesium and potassium maintained in the high-normal range, and the threshold for continuous rhythm monitoring kept low.

### **WHEN THE TWO AXES COLLIDE**

The core difficulty of this operation is that its two catastrophes present identically and are treated differently. Both begin as sudden, severe hypotension. But anaphylaxis is a distributive shock requiring epinephrine and large-volume resuscitation, whereas compression, tamponade or ventricular failure is an obstructive or cardiogenic shock in which large-volume resuscitation may be futile or harmful and the definitive treatment is mechanical.

Three tools resolve the ambiguity, and all three must be prepared in advance:

1. **TOE**, which distinguishes the underfilled hyperdynamic ventricle from the compressed or failing one within seconds.
2. **The temporal relationship to cyst manipulation**, which is informative only if the surgeon announces the manoeuvre, and which should therefore be a rehearsed communication rather than an improvised one.
3. **Systemic vascular resistance and filling pressures**, interpreted together rather than in isolation.

Where the diagnosis remains unclear after these, we would argue for treating as anaphylaxis while simultaneously preparing mechanical support. Epinephrine given to a patient in cardiogenic shock is unlikely to be the decisive harm; epinephrine withheld from a patient in anaphylaxis frequently is.

### **THE OVERLOOKED HAZARD: SCOLICIDAL AGENTS**

Discussion of anaesthetic risk in hydatid surgery focuses almost exclusively on anaphylaxis, and the systemic toxicity of the agents used to prevent dissemination receives little attention. Hypertonic saline at 20 % is the standard scolicidal solution and is effective, but it is not inert. Severe and fatal hypernatraemia has been reported after irrigation of hydatid cysts; in one paediatric case serum sodium reached 170 mmol/L within six hours of hepatic hydatid surgery and the child died 20 hours later [32]. Povidone–iodine carries risks of iodine absorption and of hypersensitivity in its own right, worth remembering when a reaction occurs at the moment of irrigation rather than of aspiration.

Two measures address this at negligible cost. The volume of hypertonic saline used should be communicated to and recorded by the anaesthesiologist, and serum sodium should be measured after irrigation and again postoperatively. In cardiac surgery, where electrolytes are checked routinely, the failure mode is not the unavailability of the test but the absence of the thought.

### **A PROPOSED FRAMEWORK**

Table 1 summarises a minimum standard for anaesthetic management of cardiac hydatid cyst surgery, derived from the preceding analysis. An expanded version, with the rationale for each item, is provided as Supplementary Material.

### **LIMITATIONS AND RESEARCH PRIORITIES**

This review is narrative rather than systematic, and the underlying evidence consists overwhelmingly of case reports subject to publication bias, with no comparative data on anaesthetic technique in cardiac hydatid disease. The framework in Table 1 is a reasoned synthesis of general perioperative anaphylaxis guidance and cardiac anaesthesia principles applied to this setting; it has not been prospectively evaluated, and several of its components — notably antihistamine and corticosteroid premedication — rest on weak evidence that we have tried to identify as such rather than conceal. Recommendations that rest on extrapolation from hepatic and percutaneous series to open cardiac surgery are flagged in the text, but the extrapolation remains an assumption.

Four lines of work would materially improve practice.

**A multicentre registry of hydatid surgery in endemic regions**, recording anaesthetic technique, prophylaxis given, and the occurrence and grade of allergic reactions. This is the only realistic route to an incidence figure for open cardiac hydatid surgery specifically, and to any assessment of whether cardiac localisation differs from hepatic or pulmonary disease.

**A prospective evaluation of antihistamine and corticosteroid premedication.** The practice is widespread, is recommended in narrative reviews, has never been tested in this population, and carries a theoretical harm — masking of early signs — that has never been quantified. Given the low event rate, a registry-embedded design is more feasible than a randomised trial.

**Characterisation of parasite-specific IgE in patients undergoing hydatid surgery**, correlated against intraoperative events. If a preoperative test with any predictive value exists, this is where it would be found; at present the honest position is that none is available.

**Systematic reporting of scolical agent volumes and postoperative electrolytes.** Hypernatraemia after hypertonic saline irrigation is preventable, its true incidence is unknown, and the data required to establish it are already generated routinely and simply not recorded.

## **CONCLUSION**

Cardiac hydatid cyst surgery is uncommon enough that few anaesthesiologists will manage more than one or two cases in a career, and dangerous enough that the first should not be approached without a plan. Its defining feature is the coexistence of two catastrophes that present identically and are treated differently: an anaphylactic shock whose probability is one to two orders of magnitude above baseline and which no preoperative test predicts, and a cardiogenic or obstructive shock arising from a mass lesion in contact with the myocardium and coronary circulation.

The anaesthetic conduct of this operation is best organised not around a technique but around a pair of explicitly stated objectives — antigenic containment with instant readiness to treat anaphylaxis, and preservation of loading conditions in a mechanically constrained ventricle — and around the single monitor, transoesophageal echocardiography, that allows the two to be told apart when the moment comes. Preparation is inexpensive; the alternative is not.

## **TABLES :**

Table 1 : Anaesthetic checklist for cardiac hydatid cyst surgery

| <b>Phase</b>   | <b>Action</b>                                                                                                       | <b>Rationale</b>                                                              |
|----------------|---------------------------------------------------------------------------------------------------------------------|-------------------------------------------------------------------------------|
| Preoperative   | Document biventricular function, compressive effect, valvular function, pericardial effusion and coronary relations | Defines both the haemodynamic plan and the territory at risk                  |
|                | Record explicitly that normal eosinophil count and negative serology do not exclude allergic risk                   | Prevents false reassurance from routine investigations                        |
|                | Document atopy, drug allergy, prior hydatid surgery and prior reactions                                             | Prior uneventful surgery does not confer protection                           |
|                | Albendazole for $\geq 4$ weeks where the clinical situation permits                                                 | Reduces protoscolex viability; benefit is parasitological, not allergological |
|                | Structured team briefing: anticipated risks, highest-risk operative moment, agreed trigger phrase                   | Shared expectation shortens time to recognition                               |
| Induction      | Arterial line before induction; large-bore venous access; vasopressor infusion running                              | Allows beat-to-beat detection and immediate response                          |
|                | Epinephrine drawn up in bolus and infusion concentrations before induction                                          | Delay, not dose, determines outcome in anaphylaxis                            |
|                | External defibrillator pads before draping                                                                          | Arrhythmia may be provoked by manipulation or by the lesion                   |
|                | Titrated induction preserving preload, rhythm and systemic vascular resistance                                      | The compressed ventricle is preload-dependent and afterload-sensitive         |
|                | CPB circuit primed and perfusion team present from incision                                                         | Risk window opens before cannulation                                          |
| Intraoperative | TOE from before bypass to decannulation, with documented baseline                                                   | Only monitor that distinguishes distributive from cardiogenic collapse        |
|                | Surgeon announces cyst puncture and aspiration aloud                                                                | Temporal association is a principal diagnostic clue                           |
|                | Continuous ST-segment monitoring with documented baseline                                                           | Coronary jeopardy where the lesion abuts a vessel                             |
|                | Serum sodium after hypertonic irrigation                                                                            | Fatal hypernatraemia is reported and preventable                              |
|                | Post-repair TOE: complete evacuation, repair integrity, regional wall motion versus baseline                        | Detects inadequate closure and new coronary compromise                        |

| Phase         | Action                                                                                                                               | Rationale                                                    |
|---------------|--------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------|
| Postoperative | Surveillance for biphasic anaphylaxis, ventricular arrhythmia and coronary compromise, each named in handover                        | Directs interpretation of postoperative signs                |
|               | Avoid systolic hypertension; agree a target pressure range with the surgeon                                                          | The repaired segment is the weakest point of the ventricle   |
|               | If any reaction occurred: timed tryptase, drug-versus-manipulation timeline, allergological referral including parasite-specific IgE | Governs risk of future surgery in a recurrence-prone disease |

CPB: cardiopulmonary bypass; TOE: transoesophageal echocardiography; IgE: immunoglobulin E.

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