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### Reduced Feasibility of Mitral Repair and Greater Risk of Failed Repair in Anterior Leaflet Prolapse or Flail

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Mitral valve repair and replacement (MVR) are effective treatments for mitral regurgitation (MR) from prolapse (MVP) and/or flail. Mitral repair preserves LV function and avoids anticoagulation but is not always feasible. To assess whether the location of mitral leaflet prolapse or flail impacts the feasibility of mitral repair, we examined pre- and postop (4–10 days) transthoracic echoes (TTE) on 203 consecutive surgical pts (mean age  $60 \pm 14$  years, 64 women). Pts were separated by which leaflet had prolapse or flail (anterior, posterior, or bileaflet). No pt with bileaflet involvement had anterior flail. Age, gender, preop LV and LA dimensions were not significantly different among the three groups. All pts (3%) with immediate failure of repair had a 2nd pump run for further surgery.

	Anterior leaflet n = 39	Posterior n = 103	Bileaflet n = 61	Total n = 203
% Flail	49	85	57	72
% Repair	59*	89*	87*	83
% 2nd pump	7.5	1.9	0	2.5

\*p < 0.005

The 3 groups (or repair vs MVR) did not differ in reduction in LV ( $62.7 \pm 8.5$  to  $53 \pm 13$  mm,  $p < 0.001$ ) and LA size ( $54 \pm 11$  to  $46 \pm 13$  mm,  $p < 0.001$ ) after surgery. Any flail (vs no flail) increases repair feasibility (85% vs 71%,  $p = 0.01$ ).

**Conclusion:** The feasibility of repair is higher for bileaflet (87%) or posterior (90%) than anterior leaflet prolapse or flail (59%),  $p < 0.005^*$ ; and is higher in flail than prolapsing leaflets. 2. Anterior leaflet flail or prolapse has higher risk of immediate failure after attempted mitral repair.

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### Patient Controlled Analgesia Post Cardiac Surgery Results in Shorter Hospital Stays

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Despite the easy accessibility and common use by orthopedic and gynecologic surgeons, physicians caring for postoperative cardiac surgery pts have been extremely reluctant to prescribe pt controlled analgesia (PCA) pumps for their pts. The major concerns expressed were respiratory depression, overdosing, inadequate pain control, and loss of "control" by the physician/nursing staff.

Sixty pts who were scheduled to undergo myocardial revascularization were selected and asked to participate in the study. Thirty were used as controls and received standard postoperative pain management. Thirty (age, sex, & procedure matched) subjects were asked to use the PCA pump postoperatively. Five pts in each group were "redo" surgeries.

Amount of MSO4 used during consecutive eight hour periods beginning four hours after endotracheal extubation

	1	2	3	4	5	6	7
Control Group	12 mg	8 mg	10 mg	10 mg	10 mg	10 mg	10 mg
PCA Group	7 mg	6 mg	6 mg	5 mg	5 mg	4 mg	3 mg

Average length of hospital stay for the control group was 7.2 days, while it was 5.8 in the PCA pump group ( $p < 0.1$ ). The reasons for earlier discharge were earlier participation in rehab, less nausea from decreased use of oral pain meds, better pain control, and less atelectasis and pulmonary infiltrates on CXR. Cost of hospital stay was significantly less in the PCA group ( $p < 0.01$ ). The major difficulty with this approach to postoperative pain management is reorientation of both the physician and nursing staff. Only one pt had a complications of over sedation with the PCA pump, because of family interference.

Overall, pts can be managed with PCA pumps post cardiac surgery safely, with less cost, and greater pt satisfaction than by the current standard of IV MSO4 and pain pills administered by a nurse.

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### Retrograde Cardioplegia Increases Diastolic Chamber Stiffness After Coronary Artery Bypass Surgery

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We have previously shown that diastolic dysfunction is frequent immediately after CABG. Although combined antegrade and retrograde cardioplegia (A + R) may improve myocardial protection, it can also lead to extracellular

edema and microvascular injury. To assess postCABG diastolic function after A + R cardioplegia, volume manipulation was used to create pressure-area curves before and after CABG in 20 pts. LV end diastolic area (EDAREA) was measured by transesophageal echocardiography as an index of end diastolic volume, and plotted against the PCWP as an estimate of mean LV diastolic pressure. A smaller EDAREA at a similar PCWP postCABG reflects a leftward shift in the pressure-area curve and increased LV diastolic chamber stiffness.

	A (n = 14)	A + R (n = 6)	p
EF(%)	$52 \pm 4$	$56 \pm 4$	NS
Wall thickness (mm)	$9.9 \pm 0.1$	$9.3 \pm 0.3$	NS
Bypass (min)	$80 \pm 5$	$74 \pm 7$	NS
Pre-Post $\Delta$ PCWP (mmHg)	$1 \pm 1$	$0 \pm 1$	NS
Pre-Post $\Delta$ EDAREA (%)	$-12.4 \pm 2.3$	$-22.5 \pm 3.0$	0.02

Increased LV diastolic chamber stiffness occurred in all pts postCABG, but was more pronounced with both antegrade and retrograde cardioplegia.

**Conclusion:** After CABG, combined A + R cardioplegia is associated with a more prominent increase in diastolic chamber stiffness than antegrade cardioplegia alone. Recognition of worsening diastolic function with A + R may be important for optimal pt management postCABG.

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### Resection vs PTFE Chordal Replacement for Repair of Mitral Valve Insufficiency

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Traditional management of prolapsing leaflets involves leaflet resection  $\pm$  native chordal repair. Uncertainty exists as to the role of chordal replacement with PTFE sutures. We compared the outcome of repair in 108 pts, 56 (52%) with #5 PTFE chordae (C) and 52 (48%) with resection (R). Both C & R had Puig-Massana ring annuloplasties. Mean age was  $61 \pm 16$  yrs, 53 were male (49%) and 79% of pts were NYHA III or IV. Sinus rhythm was present in 75 (69%) pts, atrial fibrillation in 25 (23%). Etiology was myxomatous: 68 (63%), rheumatic: 13 (12%) ischemic: 12 pts (11%). Other valve replacement  $\pm$  CAB were performed in C 16 (29%) pts, R 27 (52%) ( $p = 0.0132$ ). Clamp time was  $56 \pm 23$  min. for C,  $61 \pm 28$  min for R ( $p = NS$ ), bypass time  $78 \pm 30$  and  $84 \pm 30$  min ( $p = NS$ ). Mortality (30 day) was CR 1/56 (1.8%), R 3/52 (5.8%) ( $p = NS$ ). Post-op, mitral regurgitation was absent/mild in 104 (96%) pts, for CR 53 (95%), R 51 (98%) ( $p = NS$ ). PredischARGE mitral valve gradient was for CR,  $2.45 \pm 1.78$  mm and RT  $2.73 \pm 2.45$  mm ( $p = NS$ ). At follow-up of up to 5 years, 96% of pts were NYHA I or II. Reoperation was required in C 1/56 (2%) R 4/52 (8%),  $p = NS$ . Thus use of C produced results similar to R. C repair can be used in all pts with mobile leaflets and mitral regurgitation, especially when both anterior and posterior leaflets are involved.

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### The Effects of Protamine Sulfate on Myocyte Sarcolemmal Processes and the Relationship to Contractile Function

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Protamine sulfate (PROT), a polycationic peptide is used to reverse the anticoagulant effects of heparin in cardiovascular operations but has been associated with acute LV dysfunction. We hypothesized that the cationic nature of PROT would alter myocyte (MYO) contractile processes, sarcolemmal function ( $Na^+$ ,  $K^+$ -ATPase) and electrophysiology. Accordingly, isolated MYO contractile function (velocity of shortening; VEL,  $\mu m/s$ ) from 6 pigs was measured in the control state (no PROT) and in the presence of PROT (40 or 80  $\mu g/ml$ ; clinical doses of 2.5 and 5 mg/kg). Baseline (BASE) MYO VEL was obtained and then repeated following inhibition of the  $Na^+$ ,  $K^+$ -ATPase with 2  $\mu M$  ouabain (OUAB). In addition, indices of MYO membrane potential (resting, RMP; max upstroke velocity,  $V_{max}$ ; time to 90% repolarization, APD<sub>90</sub>) were measured in the control state and in the presence of PROT.

	BASE-VEL	OUAB-VEL	RMP (mV)	$V_{max}(V/s)$	APD <sub>90</sub> (ms)
CONTROL	$48 \pm 2$	$65 \pm 6^+$	$-79.1 \pm 0.8$	$139 \pm 5$	$163 \pm 9$
40-PROT	$31 \pm 2^*$	$32 \pm 3^*$	$-73.7 \pm 1.1^*$	$130 \pm 5$	$214 \pm 18^*$
80-PROT	$27 \pm 1^*$	$28 \pm 4^*$	$-73.0 \pm 2.1^*$	$114 \pm 5^*$	$219 \pm 6^*$

\*p < 0.05 vs Control, +p < 0.05 vs BASE

$Na^+$ ,  $K^+$ -ATPase OUAB binding (Ba; pmol/mg), affinity (Kd; nM) and hydrolytic activity (ACTIVITY;  $\mu gPMP/mg/hr$ ) were measured in the control state and with 40  $\mu g/ml$  PROT. **Summary:** PROT altered  $Na^+$ ,  $K^+$ -ATPase receptor binding but not hydrolytic capacity. Thus, the mechanism for changes in MYO function and electrophysiology is likely due to alterations in sarcolemmal conformation. These results provide a potential cellular mechanism responsible for the acute LV dysfunction associated with PROT administration.