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25 **Introduction**

26 Tricuspid valve dysplasia is an uncommon congenital malformation in small animals,
27 accounting for approximately 3% of congenital cardiac malformations in dogs
28 (Oliveira et al. 2011). It is more common in larger dogs, with Labrador Retrievers,
29 English Bulldogs and Golden Retrievers amongst others predisposed (Famula et al.
30 2005, Oliveira et al. 2011). A spectrum of valvular lesions are possible with the most
31 common being thickened, immobile septal leaflets that are effectively tethered to the
32 ventricular septum (Liu & Tilley 1976). The resulting valvular dysfunction leads to
33 progressive right atrial and ventricular volume overload, with chamber dilatation and
34 dilatation of the tricuspid annulus, which further exacerbates the valvular
35 incompetence. Treatment options for canine tricuspid valve dysplasia typically consist
36 of medical therapy for right sided heart failure (Adin 2008). In the human literature
37 both valve repair and valve replacement are reported with the decision based on the
38 specific valvular morphology and whether repair is feasible. In man, both techniques
39 have advantages and disadvantages. There is one report describing the surgical
40 treatment of tricuspid valve dysplasia in the veterinary literature to date (Arai et al.
41 2011) which documents the outcome of bioprosthetic valve implantation, under
42 conditions of cardiopulmonary bypass (CPB) in 12 dogs. In that study, dogs were
43 considered candidates for tricuspid valve replacement if they had severe tricuspid
44 valve regurgitation associated with clinical signs of cardiac compromise such as
45 severe exercise intolerance and ascites, and required on-going medical therapy (Arai
46 et al). Ten of the dogs survived surgery with a further two euthanatised at 10 and 13
47 months post-operatively due to inflammatory pannus formation and consequent
48 failure of the bioprosthesis. The purpose of the study reported here is to describe the
49 short and long term outcomes in a further cohort of dogs.

50

51 **Materials and Methods**

52 Similar to Arai et al.'s description (2011), tricuspid valve replacement was undertaken in
53 dogs that had severe tricuspid valve incompetence associated with right heart failure and
54 whose owners fully accepted the risks associated with this treatment, along with the
55 financial obligations associated with surgery. Written client consent was obtained from all
56 owners. Data were collected from the medical records of all dogs that had undergone
57 tricuspid valve replacement under CPB at the RVC between 2006 and 2012. Data gathered
58 included: signalment, clinical signs, previous and current medication, echocardiographic
59 findings, duration of anaesthesia, CPB and cross clamp time, type and size of valve used,
60 pre- and post-operative complications and time to discharge. Follow up data were obtained
61 from the medical records for subsequent visits to our referral centre, and long term
62 outcome was obtained from either the medical record if the patient was known to be
63 deceased, including post mortem data if applicable, or by referring veterinarian or owner
64 contact. Minor complications were defined as those requiring no surgical intervention;
65 major complications were those requiring surgical intervention or resulting in death.

66 The technique for tricuspid valve replacement has been previously reported by Arai
67 and others (Arai et al. 2011). The protocols for anaesthesia and cardiopulmonary
68 bypass used in this study, have also been reported previously (Griffiths et al. 2005,
69 Orton et al. 2001). All dogs were administered peri-operative antibiotics (cefuroxime
70 (Zinacef; GlaxoSmithKline) n=8, imipenem (Primaxin; Merck Sharp & Dohme Ltd),
71 n=1). Briefly, a right fifth intercostal thoracotomy was performed. The pericardium
72 was opened and pericardial basket sutures placed. Venous drainage was achieved
73 through two right angle cannulas placed in the cranial and caudal vena cavae through

74 purse-string sutures in the adjacent right atrial myocardium. The arterial limb of the
75 circuit was completed with arterial cannula in the right external carotid.
76 Cardiopulmonary bypass was initiated and the dogs were cooled to an oesophageal
77 temperature of 28° C. Rummel tourniquets of umbilical tape were used to form a seal
78 around the intracaval part of the venous cannulas and the azygous rummel was
79 tightened to stop flow through the azygous vein. An 18g cardioplegia cannula was
80 inserted into the aortic root through a horizontal mattress suture of 5-0 polypropylene
81 (Prolene; Ethicon) with expanded polytetrafluoroethylene (ePTFE) pledgets.
82 Following aortic cross-clamping, cold (4° C) cardioplegia solution (Cardioplegia
83 infusion; Martindale), combined with blood from the bypass circuit, was infused into
84 the aortic root. Cardioplegia was delivered at 20 minute intervals or whenever
85 mechanical cardiac muscular activity was observed.

86 The right atrial incision was made along a line parallel with the atrioventricular
87 groove and equidistant from it and the dorsal pericardial reflection of the right atrium.
88 Stay sutures of 3-0 polyglactin 910 (Vicryl; Ethicon) were placed around the atrial
89 incision to maintain exposure of the tricuspid valve orifice. The tricuspid valve was
90 inspected and the septal leaflet excised. Interrupted mattress sutures of 2-0 braided
91 polyester with 7mm x 3mm PTFE pledgets (Ti-Cron Davis and Geck) were placed
92 around the tricuspid annulus such that the edges of the pledgets were closely
93 approximated on the ventricular side of the annulus, (Figure 1). The mural valve
94 leaflet was “gathered” or “reefed” to preserve chordal attachments but prevent the
95 valve leaflet impinging on the artificial valve. Once all the sutures had been placed
96 around the annulus, a valve “sizer” was gently inserted into the annulus so that the
97 correct valve size could be selected. The pre-placed sutures were then passed through
98 the suturing ring of the correctly sized artificial valve at even space intervals, and tied,

99 (Figure 2). The valve holding apparatus was removed, the heart was de-aired by
100 allowing it to fill with blood from the azygous vein. The atriotomy incision was
101 closed using 4-0 polypropylene (Prolene; Ethicon) with ePTFE pledgets in a
102 continuous mattress suture oversewn by a simple continuous suture. Two suture
103 strands were used, one starting from each end of the atriotomy and the sutures were
104 tied in the middle of the incision following a final de-airing of the atrium.

105 During atriotomy closure the dogs were warmed to an oesophageal temperature of
106 37°C. At the end of atriotomy closure, the aortic cross clamp was removed and the
107 myocardium allowed to re-perfuse. If normal sinus rhythm did not resume,
108 ventricular fibrillation was managed by direct internal electrical defibrillation (20 –
109 50J) and asystole was managed by the placement of temporary epicardial pacing leads
110 (Ethicon temporary pacing leads (2-0)), and pacing begun at 100 beats per minute.

111 The dogs were weaned from bypass, a thoracostomy tube was placed and the
112 thoracotomy closed in a routine fashion. Ventilatory support was provided using a
113 mechanical ventilator that provided inspiratory pressure support (2 to 8 cm H₂O)
114 along with supplemental oxygen. The level of ventilatory support and supplemental
115 oxygen required was determined by results of arterial blood gas analysis. The dogs
116 were recovered from anaesthesia in the intensive care unit where their therapy was
117 adjusted according to perceived needs based on changes in arterial blood gas
118 measurements, blood pressure, urine production and fluid retrieved from the chest
119 drain. Once the thoracostomy tube had been removed and all post-operative bleeding
120 had stopped, heparin (100 U kg⁻¹ SC q 8h) was administered in all but one dog (which
121 received aspirin alone). Warfarin (0.1 mg kg⁻¹ PO q 24hrs), was initiated the day after
122 heparin was started and was continued for three months after valve implantation.
123 Heparin therapy was discontinued three days after initiation of warfarin treatment at

124 which stage aspirin was started and continued for the remainder of the dogs life. The
125 dose of warfarin was adjusted according to changes in the measured prothrombin time
126 and subsequent calculation of the international normalized ratio (INR) with the goal
127 of maintaining the INR between 2.5 and 3.5. The INR was calculated 72 hours after
128 initiating warfarin and checked weekly to 4-6 weeks depending on the result of the
129 INR at each check.

130 Dogs remained in the ICU for a minimum of five days. Unless complications were
131 encountered, echocardiography was repeated at 48 hours post-operatively and on
132 alternate days thereafter for the remainder of their hospitalisation.

133

134 **Results**

135 Nine dogs met the inclusion criteria with their owners electing surgery. A variety of
136 breeds were represented, with Labrador Retrievers (n=3) being the most common,
137 followed by Golden Retrievers (n=2) and one each of Dogue de Bordeaux, Rhodesian
138 Ridgeback, Rottweiler and Bassett Hound, (Table 1).

139 Six males, (two neutered) and three females, (all entire) were treated. Median age at
140 surgery was 13 months (range 7-61 months). Median weight was 26.5kg (range 9.7-
141 59 kg), (Table 1). Six dogs had a history of CHF prior to surgery and three had atrial
142 fibrillation. In one dog, electrical cardioconversion was attempted prior to surgery but
143 was unsuccessful. A variety of clinical signs were present including exercise
144 intolerance, polyuria/polydipsia, distended abdomen, lethargy, stunted growth,
145 dyspnoea and cachexia. All dogs apart from one were receiving a combination of
146 medications prior to surgery; furosemide (Frusedale; Dechra, Frusemid; Millpledge,

147 Frusol; Rosemont), (n=6), pimobendan (Vetmedin; Boehringer Ingelheim), (n=3),
148 enalapril (Enacard; Merial), (n=8), digoxin (Lanoxin; Asper Pharma trading), (n=3),
149 spironolactone (Prilactone; Ceva), (n=1).

150 The grade of heart murmur was recorded in 6 dogs pre-operatively, and was a grade
151 V/VI in five and III/VI in one. On echocardiographic examination no dogs had
152 evidence of apical valve displacement and all dogs had tethering of the septal valve
153 leaflet to the septal wall. The free wall leaflets varied in appearance, ranging from thin
154 and tethered to very thick, with variable chordae tendinae attachments. Eight dogs had
155 tricuspid regurgitation, and the remaining dog had tricuspid stenosis.

156 Cross clamp and total bypass times were available in 8 dogs with a median of 65
157 minutes (range 45-90) and 98.5 minutes (range 65-120) respectively. One dog had a
158 patent foramen ovale closed during the procedure. Eight bovine pericardial valves
159 were used (27-33mm sizes), (Perimount Plus; Carpentier-Edwards) and one 25 mm
160 porcine aortic valve prosthesis (Baxter).

161 One intra-operative complication occurred: a tear in the aorta at the insertion site of
162 the cardioplegia cannula which was successfully repaired with sutures. All dogs
163 survived surgery but six dogs experienced complications during hospitalisation, and
164 four of these were fatal. Of the minor complications, one dog developed partial
165 tongue necrosis, minor wound dehiscence and a supraventricular tachycardia, all of
166 which resolved. The other dog developed a pneumothorax after thoracostomy tube
167 removal which was successfully managed by a period of continuous pleural drainage.
168 This dog also developed a large right atrial thrombus but remained stable with a good
169 cardiac output and was discharged 29 days post-operatively. Of the dogs experiencing
170 fatal complications, one dog developed acute central nervous system (CNS) signs on

171 the morning of planned hospital discharge (day five post-operatively). He
172 subsequently became acutely hypotensive with low output heart failure after an
173 uneventful initial recovery, presumed to be due to a thrombus on the valve. The cause
174 of the neurological signs was thought likely due to a transient hypoxia either due to
175 low output heart failure or a pulmonary embolus. This dog was treated with a
176 thrombolytic agent (Tenecteplase (TNKase®; Genentech)) at a dose according to the
177 recommendations in people, but developed profuse haemorrhagic diarrhoea and was
178 euthanatised. Post mortem evaluation confirmed the presence of thrombus on this
179 dog's valve as a cause of acute valve failure (Figure 3). This dog was the only case
180 that had a porcine aortic valve implanted, and was also the only dog to receive just
181 aspirin rather than heparin and warfarin as well. The second dog developed
182 hypotension, hypoxia and oliguria approximately 12 hours post-operatively. Despite
183 aggressive supportive care this dog continued to deteriorate and was euthanatised at
184 approximately 20 hours post-operatively. The third dog initially recovered well but
185 remained in the hospital while warfarin treatment was stabilised. He became pyrexia
186 on the 8th post-operative day and on day 11, a positive blood culture confirmed
187 highly resistant strains of *Enterobacter cloacae* and *Escherichia coli*. This dog
188 experienced a suspected brain stem haemorrhage, with the loss of brain stem auditory
189 evoked responses on day 14 and was euthanatised. The fourth dog also made a good
190 recovery initially but became pyrexia on the fourth post-operative day and died from a
191 cardiorespiratory arrest. Again, a multi-resistant *Enterobacter cloacae* and
192 *Acinetobacter baumannii* were cultured from ante mortem blood samples.

193 Five dogs were discharged from the hospital. One dog collapsed after a minor fall at
194 home and was returned to the hospital seven days after discharge, and was dead on
195 arrival. At post mortem examination this dog had a large volume intrathoracic

196 haemorrhage, likely due to minor trauma in conjunction with the anti-coagulant
197 medications. Despite this fatal haemorrhage, this dog had thrombus covering his
198 valve, (Figure 3). Of the four remaining dogs, one dog had a low volume pleural
199 effusion at three months post-operatively at which stage he was started on furosemide.
200 He remained well for the following month, and at 4 months post-operatively he had
201 no evidence of a thrombus or micro clots, and had only mild tricuspid regurgitation.
202 At 8 months post-operatively he was presented in congestive heart failure and atrial
203 fibrillation; a very large thrombus was found on the valve causing valvular stenosis
204 and the dog was euthanatised. The second dog had an echocardiogram performed four
205 months post-operatively, which showed improved right ventricular function and a
206 reduction in his heart murmur from a grade IV/VI to a grade I/VI, but was
207 euthanatised due to metastatic osteosarcoma at 246 days after surgery. Revision
208 surgery was attempted in the third dog 12 months post-operatively, but she was
209 euthanatised on the table when it became clear that explanting the valve would be
210 impossible due to extensive inflammatory tissue engulfing the prosthesis.
211 Inflammatory pannus was confirmed histologically at post mortem examination
212 (Figure 4). The final dog collapsed and died 1277 days post-operatively whilst
213 exercising. A post mortem examination was declined but three months prior to this a
214 repeat echocardiogram of the valve showed no abnormalities, (Table 1).

215 **Discussion**

216 In the group of dogs undergoing cardiopulmonary bypass for tricuspid valve
217 replacement in the study reported here, only 5/9 dogs survived to discharge. Of the
218 five dogs that died in the short term, three died because of problems associated with
219 coagulation (thrombus formation, n=1), or anticoagulation (fatal hemorrhage, n=2).

220 Two dogs developed pyrexia with positive blood cultures, and it is assumed they were
221 septicaemic, several days after apparently uneventful recovery. Of the four dogs that
222 survived in the long term, two died as a result of stenosis of the valve with the
223 presence of fibrous tissue (inflammatory pannus/organized thrombus) confirmed
224 histopathologically, the cause of one death was unknown and one death (euthanasia
225 because of osteosarcoma) was unrelated to cardiac disease.

226 There is only one other report in the veterinary literature describing tricuspid valve
227 replacement in dogs (Arai et al. 2011). The mortality rate in the study reported here
228 was higher in the short term (n=5/9) when compared to Arai et al. 2011 (n=2/12). The
229 reason for this difference is unknown; the surgical technique including cannulation
230 methods are identical between both centers, indeed, the surgery, perfusion and post
231 operative care team from Colorado State University performed the first tricuspid
232 valve replacement at the Royal Veterinary College (RVC), alongside the RVC team.
233 These nine dogs, along with 12 dogs that underwent open patch grafting of the right
234 ventricular outflow tract to treat pulmonic stenosis and double chambered right
235 ventricle (unpublished data), represent the first 21 dogs operated on at the RVC under
236 cardiopulmonary bypass and so it would be reasonable to expect a higher incidence of
237 technical failures initially, but similarly, it would be expected that these would reduce
238 as familiarity with the techniques developed.

239 Most of the deaths in the dogs in our study were related to problems with blood
240 clotting (inadequate haemostasis and thrombogenic complications), despite our
241 attempts to use the anti-coagulation therapy previously reported, which consisted of
242 heparin and warfarin once post-operative bleeding had ceased. The only difference
243 between the protocol used in the study reported here and that reported by Arai et al.

244 (2011), was that warfarin therapy was started the day following heparin initiation in
245 our population, compared with the second post-operative day in the study reported by
246 Arai et al. (2011). One of the dogs in our study only received antiplatelet therapy
247 (aspirin) following immediate post-operative heparin therapy, based on the
248 recommendation of an experienced human cardiac surgeon; and this was the dog that
249 died as a result of acute valve failure secondary to thrombus formation. Although only
250 one case, it would appear that aspirin alone is not an effective strategy in dogs, despite
251 its success in humans. This was also the only dog in our paper to have a porcine aortic
252 valve implanted. One of Arai et al.'s (2011) conclusions was that inflammatory
253 pannus was more likely with implantation of a bovine pericardial valve (2/4
254 developed this in their cases), as opposed to a porcine aortic valve (0/5 developed
255 this), however because the only case that received a porcine valve was also the only
256 case treated with aspirin alone, the finding of tricuspid valve thrombus on post
257 mortem should be interpreted cautiously. In contrast with our findings, humans appear
258 to have a relatively low risk of death or embolic complications in the first three
259 months following surgery for aortic valve bioprosthesis replacement (Brennan et al.
260 2012). This study showed that the combination of aspirin and warfarin relative to
261 aspirin alone had a lower adjusted risk of death and embolic events, however this
262 group of patients had a higher risk of bleeding (Brennan et al. 2012). A meta-analysis
263 from 2001 on humans with prosthetic heart valves, concluded that adding low dose
264 aspirin to warfarin decreases the risk of embolism or death, with a slightly increased
265 risk of bleeding, and concluded that there was a favorable risk to benefit ratio with
266 this protocol (Massel & Little 2001). Even in human medicine, controlling the balance
267 of the coagulation cascade post-operatively is clearly still a challenge, however, on

268 the evidence of the dogs reported here, much work is needed before we can
269 recommend the use of valves that require even short-term anticoagulation in dogs.

270 The reason tissue valves were chosen as the prosthesis for these dogs was because
271 human patients with tissue valves do not require life-long anticoagulant therapy once
272 the exposed elements of the valve are coated with native endothelium, (Bloomfield
273 2002). In addition, Orton et al. (2005) concluded that long term anticoagulant therapy
274 was difficult to monitor and control in a report of a series of dogs that underwent
275 mitral valve replacement using a bi-leaflet mechanical valve; with thrombus-related
276 valve failure seen as a frequent event (Orton et al. 2005). Again, it is not clear why
277 our results differ from those of Arai et al. (2011) as the variation in anticoagulant
278 therapy (with the exception of one dog) is minor and would have been more likely to
279 reduce coagulation related problems. The group at Colorado State have published
280 several reports on the use of warfarin in dogs, (Arai et al. 2011, Monnet & Morgan
281 2005, Orton et al. 2005), so we conclude that some of the complications we saw
282 associated with anticoagulation were due to our relative inexperience, but also that
283 this remains problematic even in the hands of those more experienced in its use.

284 Two dogs in the study reported here died of septic complications, one dog four days
285 after surgery and the other dog ten days after surgery. Both of these dogs had
286 recovered uneventfully initially, having received cefuroxime (Zinacef;
287 GlaxoSmithKline), during the perioperative period. In both dogs, multi resistant
288 enterobacteriaceae were involved in the infection. It is assumed, therefore, that these
289 were nosocomial infections that gained access to the body through either the
290 intravenous access sites, chest drain or urinary catheter. Whilst we endeavored not to
291 leave such catheters in longer than necessary, the critical nature of the first 24 -48

292 hours recovery period necessitates intensive monitoring and such “instrumentation” is
293 essential. Clearly, in any busy hospital, it is advisable to remove any instrumentation
294 as soon as it is reasonable to do so, to eliminate or reduce the risk of ascending
295 infection. Imipenem was used in the case subsequently to these two cases for 48
296 hours, based on the above dogs’ culture and sensitivity and the presumption that these
297 were hospital acquired. We have subsequently reverted back to the protocol of using
298 cefuroxime and now de-instrument dogs sooner if they are stable.

299 The reasons for the poorer outcome in the study reported here remain unclear. With
300 so many variables (surgery team, anaesthesia team, cross clamp time, bypass time,
301 total surgery time, valve type used, weight, etc) that could affect outcome, a larger
302 number of dogs undergoing this procedure would have to be studied. Based on the
303 results reported here, we have to conclude that at least in our hands, bioprosthetic
304 tricuspid valve replacement in dogs has poor results with a high short term mortality
305 rate and a short survival time postoperatively.

306 No conflicts of interest have been declared

307

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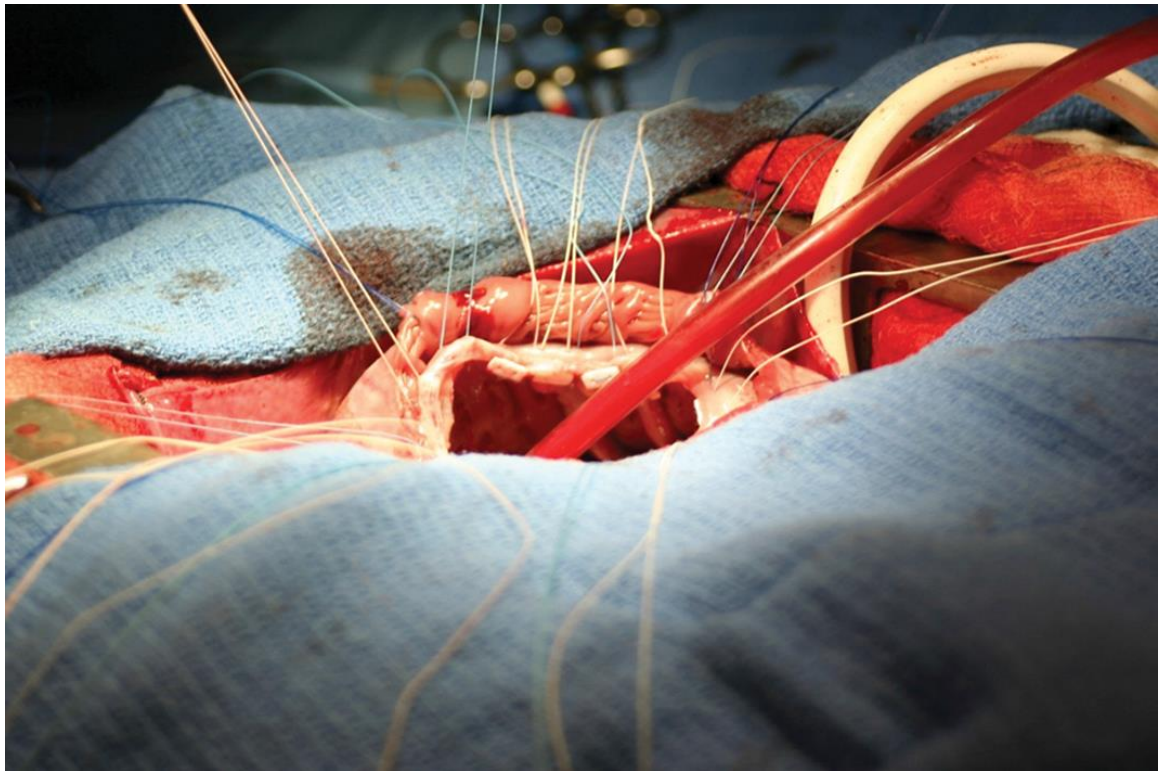
351 Table 1: Signalment and survival details

Case	Breed	Age at surgery (months)	Body weight at surgery (kg)	Survival (days)
1	Rhodesian ridgeback	61	52	246
2	Rottweiler	7	19.5	5
3	Labrador retriever	12	25	370
4	Bassett hound	36	18.5	240
5	Labrador retriever	13	28	1277
6	Golden retriever	10	23	1
7	Labrador retriever	36	34	14
8	Dogue de Bordeaux	13	59	4
9	Golden retriever	13	30	?

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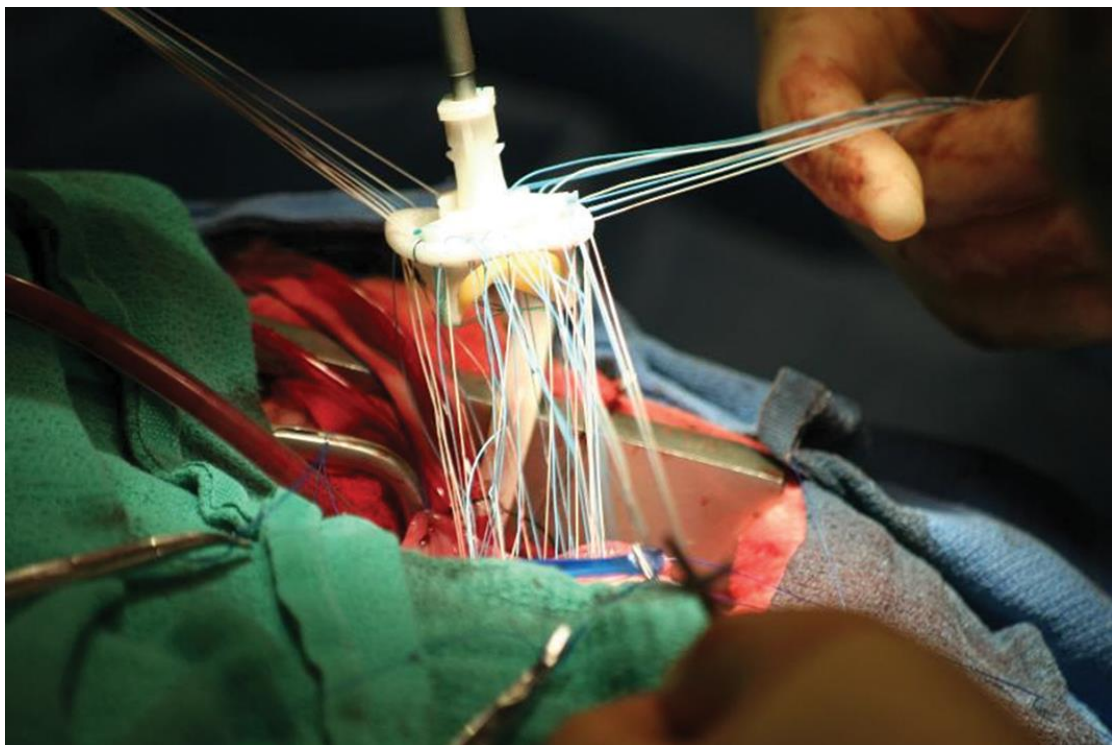
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354 Figure 1: Sutures of 2-0 TiCron placed in the tricuspid annulus with pledgets on the
355 ventricular side



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357 Figure 2: Prosthetic valve mounted on handle, after preplaced sutures have been
358 passed through suturing ring



359

360

361 Figure 3: Post-mortem picture of thrombus on valve (day four postoperatively, case 8)



362