

Successful prolonged cardiopulmonary resuscitation in a Siamese cat: A case of post-cardiac arrest concerns

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Abstract

A 4.5-year-old male neutered Siamese cat was admitted to referral hospital for medical work-up of chronic anorexia and emesis. No severe abnormalities were detected on routine blood work; however thickened gastric wall and small intestines were noted on abdominal ultrasonography. The cat was premedicated with buprenorphine followed by a diazepam-propofol co-induction. Endotracheal intubation was performed, and anaesthesia was maintained using isoflurane in oxygen for endoscopic investigation of the thickened gastric wall and biopsies retrieval. During the anaesthetic recovery, the animal went into cardiopulmonary arrest. Successful ROSC (return of spontaneous circulation) was achieved at approximate 40 minutes (prolonged) post-arrest, and the animal had a survived event (ROSC > 20 minutes). However, failure to identify the inciting cause of the CPA and laxity in the post-cardiac arrest care resulted in rearrest of the cat, 77 hours after the initial ROSC with no success of a second ROSC.

BACKGROUND

Cardiopulmonary arrest (CPA) is one of the most crucial of emergencies faced by a veterinarian that requires rapid response and competency, of the individual rescuer or team, when performing effective cardiopulmonary resuscitation (CPR). The American College of Veterinary Emergency and Critical Care and the Veterinary Emergency and Critical Care Society undertook an initiative to evaluate the current evidence of veterinary-related CPR and compiled evidence-based guidelines for veterinarians. The initiative is named The Reassessment Campaign on Veterinary Resuscitation (RECOVER), and more information and the current guidelines can be found on their website (www.acvecc-recover.org). The CPR effort is made up of two, sometimes overlapping, phases. First, basic life support is started, ideally within minutes of suspected CPA, followed by advanced life support (ALS).¹

The purpose of CPR is to support the animal until its own autonomic functions restart, and when this occurs it is called a return of spontaneous circulation (ROSC). The sooner ROSC is achieved then less permanent central nervous system dysfunction occurs.²⁻⁴ However, it is unknown when the animal will achieve ROSC. In a study by McIntyre et al, the median (range) duration of CPR for all cats and dogs with and without ROSC was five (0.5–40) and 13 (two–40) minutes, respectively.⁵ In that same study, the median (range) duration of CPR for cats with and without ROSC was seven (0.5–30) and 15 (five–40) minutes, respectively. Furthermore, the median (range) duration of CPR in all patients that survived to discharge was three (0.5–15) minutes. Whereas Kass and Haskins reported that the mean (\pm standard

deviation) duration for CPR in cats was 18 (\pm 15) minutes, and the longest record CPR event was 60 minutes.⁶

Anecdotally, a prescribed time limit of 20 minutes has been assigned in which ROSC should be achieved. Reason being that if ROSC has not been achieved by this time, a worse outcome (neurological and myocardial dysfunction) is expected, the cost of resuscitation gets too expensive or inability of a facility to manage the post-cardiac arrest (PCA) period. In 1989, Van Hoeyweghen et al⁷ showed that under certain conditions (pulseless electrical activity, asystole, agonal breathing) more time should be allocated for continuation of CPR especially if efficient external chest compressions are being performed.

Rapid identification of CPA improves the chances of achieving ROSC, neurological function and survivability to discharge.⁸ However, ROSC depends on the inciting cause of CPA, whether it be traumatic or illness related. Prompt identification and reversal of the cause may lead to good functional outcome and survivability.³ Kass and Haskins⁶ reported that patients which arrested from non-drug or anaesthetics reactions had a higher risk of rearrest within three days than those which arrested due to drug or anaesthetic reactions. Failure to correct the underlying pathology either during CPR or PCA period will lead to unfavourable outcome, and thus rearrest is imminent.

This case report demonstrates the successful ROSC in a cat after prolonged CPR was performed. However, pitfalls in the PCA period were experienced which lead to the rearrest of the cat.

CASE PRESENTATION

Pegasus, a 4.9 kg, 4.5-year-old male neutered Siamese cat, was referred for an internal medicine consultation at Valley Farm Animal Hospital, South Africa. The chief complaint was chronic emesis, despite ongoing symptomatic treatment from the referral veterinary clinic. On presentation, the cat was lethargic and had a body condition score of 4 of 9 (little subcutaneous adipose tissue; WSAVA Cat Body Condition Scores) with thoracic kyphosis, mild skin tenting, oculonasal mucoid discharge (frequent life-long problem), severe halitosis, pink with slight icteric tinge on mucus membranes, elevated heart rate (HR) of 210 beats per minute, rectal temperature of 39.0°C and painful on cranial abdominal palpation. A packed cell volume (microhaematocrit) of 52% was measured. A biochemical profile (IDEXX Catalyst, IDEXX Lab Inc., USA) revealed a mild hypokalaemia (3.2 mmol/L; reference interval 3.5-5.8 mmol/L) and mild hyperlactataemia (3.95 mmol/L; reference interval 0.6-2.5 mmol/L), with normonatraemia and normochloroemia. The urine analysis revealed a specific gravity of 1.006, and a pH of 5, protein 1+, glucose 1+ and bilirubin 2+ on the urine dipstick (Combur⁹Test, Cobas, Roche). Abdominal ultrasonography revealed a thickened gastric wall. Presumptive diagnosis of gastritis or some form of inflammatory bowel disease. Owner consented to treatment of hypokalaemia and gastritis with lactated Ringer's solution (LRS) (Fresenius Kabi Manufacturing), maropitant (1 mg/kg once a day; Cerenia, Zoetis), metronidazole (15 mg/kg, bid for 7-day course; Tricazole, Fresenius Kabi Manufacturing) and oral potassium supplementation (for 5 days; Kaligel, Kyron Laboratories). Owner insisted on a gastroscopy, and thus we advised to place an oesophagostomy feeding tube under the same general anaesthesia (GA).

The medicine team used a routine GA protocol familiar to them and continued with the procedures. The cat was premedicated intravenously (IV) with buprenorphine (0.015 mg/kg;

Temgesic, Indivior) 30 minutes prior to IV co-induction of diazepam (0.2 mg/kg; Pax, Pharmacare) and propofol (2 mg/kg total dose titrated to clinical effect; Fresenius Propoven, Fresenius Kabi Manufacturing). The trachea was intubated using a cuffed polyvinyl chloride tube (4.0 mm internal diameter; Kruuse) and was maintained on isoflurane in 100% oxygen via a precision vaporiser (Isotec Tec3, GE Healthcare). The anaesthetic gas mixture was delivered via a Mapleson D non-rebreathing system. Endoscopy of the oesophagus, stomach and duodenum with biopsies of the latter two regions and the oesophagostomy tube placement were successfully completed. Upon endoscopic evaluation of distal oesophagus, suspicion of a sliding hiatal hernia was proposed. Monitoring during the endoscopic evaluation comprised of cardiopulmonary auscultation and femoral pulse palpation only. The cat was moved to the intensive care unit (ICU) for recovery from the GA. The cat's trachea was extubated when the medial palpebral reflex had returned, but within two minutes, the cat became cyanotic. The ICU technicians removed the cat from the enclosure and placed it on a workbench and called for the anaesthesiologists.

LEARNING POINTS/TAKE-HOME MESSAGES

- Prolonged CPR with return to full consciousness is achievable in cats.
- Capnography monitoring during CPR aids in decision making where changes in technique or addition of other interventions can be applied without delay. An end-tidal carbon dioxide greater than 20 mm Hg should be targeted to improve the chance of return of spontaneous circulation.
- CPA aetiologies must be identified and either corrected or managed to mitigate possible rearrest.
- Dummy-assisted refresher training should be done at least every six months to ensure best-effort team driven CPR interventions.

TREATMENT

The cat was unconscious, and the technicians reintubated the trachea and provided 100% oxygen via a neonatal Ambu-bag. The anaesthesiologists confirmed CPA, within five minutes of detecting cyanosis, based on heart auscultation and absence of peripheral pulses and CPR commenced immediately. During manual ventilation it became evident that there were no chest excursions visible as compared to the volume being delivered by the Ambu-bag. The overpressure valve of the neonatal Ambu-bag engaged thus indicating a low or non-compliant system. Furthermore, no audible leak around the endotracheal tube (ETT) cuff was detected, which led to the presumption of blockage of the ETT lumen. The ETT was exchanged which revealed copious amounts of mostly white foamy fluid that had some brown tinging. The gasses delivered by the Ambu-bag could not move past the foamy fluid. The cat was immediately inverted (suspended by the pelvic girdle, held upside-down with head vertically below the thorax) and approximately 30 mL of fluid poured out of the ETT. The ETT lumen and possible mainstem bronchi were suctioned prior to reintubation with a clean ETT. Upon delivering a breath with the Ambu-bag, chest excursions were observed. All these airway interventions were done without ceasing single-handed cardiac compressions. Electrocardiogram (ECG) and capnography were used to monitor the cat. Multiple doses of adrenaline (0.01 mg/kg at three-minute intervals; Kyron Prescription) and a single bolus of atropine (0.05 mg/kg, IV; Bayer) were administered. External chest compressions were maintained between 80–120 compressions per minute with manual ventilation delivered at six–10 breaths per minute using bag mask device. An end-tidal carbon dioxide (PE'CO₂) of 15–17 mm Hg was achieved and thus CPR continued. At approximately 38 minutes of CPR,

ROSC was achieved with a HR of 60 beats per minute and a spike in PE'CO₂ (31 mm Hg). Pegasus was placed on pressure-controlled ventilation (Carestation 650, GE Healthcare) aiming for 8 mL/kg tidal volume with positive end-expiratory pressure of 4 cmH₂O and peak inspiratory pressure of 12 cmH₂O. The oesophageal temperature was 33.5°C and was allowed to slowly increase (<1°C/hour) by using active heating by a forced air warming device (BairHugger, 3 M). The cat's PE'CO₂ was increasing but oxyhaemoglobin saturations (SpO₂) remained low, between 70–80%. A single bolus of mannitol (800 mg/kg; Fresenius Kabi Manufacturing), furosemide (2 mg/kg; Lasix, MSD Animal Health) and aminophylline (4 mg/kg, IV; SABAX Aminophylline, Adcock Ingram) were administered postresuscitation. The HR remained low (60 beats per minute) and atropine (0.05 mg/kg, IV; Bayer) was administered once again. An adrenaline constant rate infusion of 0.01 mg/kg/hour for four hours was started and then dropped to 0.001 mg/kg/hour. Intravenous fluids were LRS spiked with 40 mEq of potassium chloride and administered at a rate of twice maintenance. Venous blood was collected 10 minutes after ROSC and analysed immediately and revealed moderate hyperlactataemia (6.0 mmol/L; reference interval 0.6–2.5 mmol/L), mild hypokalaemia (3.0 mmol/L; reference interval 3.5–5.8 mmol/L), mild hyponatraemia (145 mmol/L; reference interval 150–165 mmol/L), mild hypochloraemia (106 mmol/L; reference interval 112–129 mmol/L), mild hyperglycaemia (11.4 mmol/L; reference interval 4.1–8.8 mmol/L); mild hypoalbuminaemia (19 g/L; reference interval 22–40 g/L) and raised ALT (300 U/L; reference interval 12–130 U/L) and total bilirubin (44 µmol/L; reference interval 0–15 µmol/L). Glycopyrrolate (0.01 mg/kg, IV; Robinul, Aspen Pharmacare) was subsequently administered when the HR dropped yet again to around 60 beats per minute. A single bolus of dexamethasone (0.5 mg/kg, IV; Kortico, Bayer) and hydroxyethyl starch (5 mL/kg, IV; Voluven 6%, Fresenius Kabi Manufacturing) were administered. Eyes were lubricated every hour until spontaneous blinking. The cat was weaned off the ventilator and extubated when the gag reflex returned and moved to the ICU for further PCA care six hours post-CPA.

Pegasus was subsequently placed in an ICU pet incubator (Aeolus Incubator, USA) with nasal oxygen support. During the PCA period, the respiratory rate ranged between 42 and 60 breaths per minute, HR between 160 and 210 beats per minute and SpO₂ maintained above 90%. Non-invasive blood pressure (high definition oscillometric) was also recorded with a mean arterial pressure ranging between 58 and 90 mm Hg. Within eight hours PCA, Pegasus was able to maintain his own body temperature, but had neurological deficits which included swaying of the head and blindness. Buprenorphine (0.01 mg/kg, IV) was administered every six hours for any post-CPR pain. The adrenaline infusion was stopped at 18 hours PCA. Pegasus was fed via the oesophagostomy tube with a high calorie meal (Recovery Liquid, Royal Canin) and electrolyte solution (Pro-lyte, Kyron). Two days PCA, only ALT and total bilirubin were mildly elevated, other variables were within normal limits, and the level of monitoring was decreased and intravenous fluids stopped, but Pegasus remained in the ICU pet incubator.

OUTCOME AND FOLLOW-UP

On the third day PCA (approximately 72 hours later), Pegasus was lethargic, hypothermic (35.2°C) with pale mucous membranes and brown foul-smelling fluid present in the mouth. Pegasus rapidly decompensated and rearrested 77 hours after the first ROSC, and the second CPR was unsuccessful.

DISCUSSION

CPR, being one of the most commonly encountered emergencies in a veterinary field (in and out of hospital setting), has numerous aspects that need to be considered and addressed. Veterinary CPR has evolved quite substantially in the last couple of years, and evidence-based guidelines have been developed to assist in increasing the rate of survived event and rate to discharge.^{1, 9, 10} The major considerations, concerns and pitfalls are highlighted, looking especially at prolonged CPR, causes of CPA, equipment utilised, why rearrests occur, and the PCA care period is discussed.

Successful ROSC leading to a survived event (ROSC longer than 20 minutes) was achieved following prolonged CPR in this case, however, unsuccessful survivability to discharge. Identification of aetiologies for the CPA was overlooked, and in combination with relaxing PCA monitoring could have led to the rearrest.

Current published literature on CPR and the survivability in domestic felines, show no improvement in ROSC rate or survival to discharge since 1992.^{2, 5, 11} However, prolonged CPR, described as CPR lasting longer than 20 minutes,¹⁰ with successful return to full consciousness could result in the improvement of these statistics. In human literature, the rate of ROSC was >90% when patients were resuscitated for less than 10 minutes but around 50% if resuscitation occurred for 30 minutes or more.¹² In another article, statistics revealed that CPR of 20 minutes had a lower survival rate compared to 20–30 minutes and >30 minutes in humans in an out of hospital setting.¹³ Prolonged CPR can lead to better survival rates if the rescuers adhere to the evidence-based CPR guidelines.¹⁴ If the generation of adequate pulses from effective chest compression is achieved and a PE'CO₂ of >15 mm Hg is reached, this could favour the ROSC even after an extended period.¹⁵

Capnography has gained favour as an essential monitoring tool during CPR not only to confirm tracheal intubation but also to govern CPR efforts. This monitoring modality is a non-invasive method to ascertain the partial pressure of CO₂ in the alveoli (end-tidal CO₂; PE'CO₂) which is a close approximation of the partial pressure of CO₂ in pulmonary arterial system. The PE'CO₂ is affected by both pulmonary perfusion and alveolar ventilation.¹ During CPR, low PE'CO₂ could represent inadequate alveolar ventilation or ineffective chest compressions to generate adequate forward blood flow. Performing effective chest compression and delivering adequate manual breaths, the PE'CO₂ level could be raised above or maintained around 15 mm Hg. Although Hofmeister et al reported a 90% successful resuscitation of cats when the PE'CO₂ was >20 mm Hg but only 44% successful resuscitation of cats with a PE'CO₂ < 20 mm Hg.² Due to this high probability of success reported in the literature, the decision to continue CPR in Pegasus for a prolonged period resulted in successful ROSC (seen with a rise in PE'CO₂). A sudden spike in PE'CO₂ (>31 mm Hg) corresponds to an increase in pulmonary blood flow due to ROSC.¹ However, prolonged CPR relies on other ALS modalities such as ECG and, blood gas analysis, to ensure adequate management of physiological derangements and possible worsening of organ injury. All this increases the cost of resuscitation.

Other pitfalls which can present with prolonged CPR, especially in a small rescuer team, is the increase in CPR cycles and rescuer fatigue, possible drug shortage and/or worsened physiological derangements. Also, if the persistent prearrest pathology or illness is too advanced or, the inciting cause of the CPA is not addressed or unresponsiveness to ALS, then achieving ROSC has a lower success rate. Furthermore, loss of clinical skills associated with

performing CPR can result in poorer outcomes and lower success rates to achieve ROSC. Thus, refresher training should be completed every six months to maintain cognitive and clinical skills associated with CPR. These refresher training sessions must incorporate recent evidence-based knowledge and allow simulation training with immediate feedback to improve psychomotor skills.¹ However, effective CPR will be futile if the inciting cause or at-risk patients are not identified.

Identifying patients at risk of CPA is the ideal approach to safeguard and minimise the potential for CPA. The mnemonic '5 Ts and 5 Hs' have assisted in the identification of at-risk patients and thus decrease the likelihood of CPA.⁸ The '5 Ts' represents toxins, tension pneumothorax, thromboembolism, tamponade (cardiac), trauma, whereas the '5 Hs' represents hypovolaemia/haemorrhage, hypoxia/hypoventilation, hydrogen ions (acidosis), hyper-/hypokalaemia and hypoglycaemia. Hofmeister et al reported hypoxaemia (36%), shock (18%), anaemia (13%), arrhythmia (8%), MODS (6%), traumatic brain injury (5%), anaphylaxis (1%) or other (21%) are causes of CPA.² Precipitating pathologies will most likely persist during ROSC and could influence the PCA care and prognosis.⁹ The inciting cause in both CPA events in this case was never determined.

Pegasus presented with numerous prearrest pathologies that is chronic weight loss, chronic oculonasal discharge, chronic emesis, icterus and severe halitosis. However, no investigation into these prearrest clinical findings was performed as the owner was insistent on only a gastroscopy. The chronic nasal discharge (most probably from viral infection as a kitten) could have compromised the respiratory system and during the GA recovery period led to upper airway obstruction followed by respiratory arrest. Chronic emesis and possible regurgitation or reflux from gastroscopy under GA could have resulted in aspiration pneumonia (indicated by the brown tinge foul-smelling fluid) which precipitated the CPA.

The Siamese breed is overrepresented with regard to lower airway disease,¹⁶ and no investigation into the pulmonary system (i.e. thoracic radiographs) was performed. Fluid aspiration, emesis, endotracheal intubation/extubation and oesophagostomy tube placement all could have resulted in an increased vagal tone. This increased vagal tone might have resulted in bradyarrhythmia and possible sinoatrial arrest during the GA recovery period. Under the influence of high vagal tone, excessive upper respiratory secretions might have resulted in a lower respiratory tract obstruction. Chronic intermittent anorexia which could have resulted in hepatic lipidosis and hepatic insufficiency as indicated by the icterus and bilirubin on urine analysis. A further hypoxic insult to the hepatic tissue during CPR might have contributed to the rearrest three days later.

No investigation (echocardiography) was performed into any Siamese predisposed cardiovascular pathologies that is endomyocardial restrictive cardiomyopathy and hypertrophic cardiomyopathy (HCM)^{17, 18} which could have contributed to both CPA events. Undiagnosed or ill-managed HCM could have resulted in the rearrest during the PCA period. The hypoxaemia with resultant hypoxic insult to the thickened myocardium during CPR might have been the leading factor in the recurrence of CPA. Speculations can be made, as to when chest compression (cardiac pump technique) was being delivered, thromboemboli from an intracardiac thrombus seeded to various organs contributing to ischaemic injury and multiple organ dysfunction (gastrointestinal tract, kidney, brain, lungs) and subsequent rearrest.¹⁹

Siamese cats are predisposed to megaesophagus,²⁰ although rare, which might have been the abnormality seen on endoscopy and not necessarily a sliding hiatal hernia. A more thorough history might have distinguished passive regurgitation from active emesis. Other diseases which Siamese cats are predisposed are diabetes mellitus, insulinoma and amyloidosis,²¹ and these too were not investigated.

Possible hypovolaemia and dehydration (unresolved from prearrest clinical findings) might have potentiated the rearrest. Fluid management during the CPR and PCA is an essential component, as fluid administration is key in the stabilisation of haemodynamics (improve venous return and thus cardiac output), assisting with therapeutic hypothermia and treatment of systemic inflammatory response syndrome (SIRS).^{2, 22} However, large fluid volumes will decrease coronary perfusion pressure. Fluid administration (of crystalloid and colloids) was not only vital in the correction of already present dehydration and hypovolaemia, but also due to the administration of mannitol (decrease cerebral oedema) and furosemide (pulmonary oedema) which both promotes diuresis and more fluid loss.

Apart from fluid therapy in the case, a brief mention of the other administered drug is warranted. Adrenaline is the first line drug at the referral hospital as the cost implication of administering vasopressin is not feasible. However, vasopressin has not shown to be beneficial over the administration of adrenaline.¹ During the PCA care, the administration of glycopyrrolate appeared to have treated the vagal-induced bradycardia. The reasoning for administering glycopyrrolate over atropine, was due to its inability to cross the blood-brain-barrier and thus does not cause mydriasis. The administration of dexamethasone is still part of the referral hospital's CPR protocol, although it has been phased out due no beneficial effect demonstrated in the literature.^{1, 8, 23} However, due to possible chronic illness-related corticosteroid insufficiency from chronic emesis and weight loss, the administration of a corticosteroid was thought to be beneficial although hydrocortisone would have been the ideal drug of choice.

Oxygen supplementation continued during the PCA period, while the cat remained in the ICU incubator. However, inadequate environmental monitoring (humidity, temperature) within the ICU incubator was suspected to have contributed to the dehydration of the cat, as the fluid therapy during PCA care was that of one-time maintenance for only two days. Nutritional support, which is of key importance during PCA care,²⁴ was provided via the oesophagostomy tube; however the caloric intake was inadequate for the animal. More astute monitoring during the PCA period could have revealed possible indications for recurrence of CPA. Immediately after ROSC, attention should be directed at prevention of recurrence of CPA and limitation of organ injury that is reperfusion injury and ischaemia, PCA brain injury and myocardial dysfunction and persistent precipitating pathological processes.⁹

PCA syndrome shares a lot of similarities with severe sepsis and multiple organ dysfunction syndrome. Treatment of the underlying disease processes, prognostication and rehabilitation are emphasised in later care. Boller and Fletcher described two paradigms of care for the PCA period:⁹ the pathophysiological process that occur post-resuscitation and¹⁰ a shift in treatment prioritisation as time after ROSC progresses. Vigilant PCA monitoring started to deteriorate under the assumption the animal became more stable, which could also have been the downfall. The time of rearresting was at the change of personnel shift, which could have been a contributing factor. No PCA care time frame is published, although clinicians refer to 72 hours PCA by which major clinical manifestations would have peaked. Physiological derangements and cellular death from inflammatory processes, reperfusion failure and

reoxygenation injury during PCA, can lead to multiple organ dysfunction up to three days postresuscitation.²³ However, clinicians should utilise an individualised patient approach and treatment of organ injury, as they evolve. This individualised monitoring and treatment planning can lead to greater survivability compared to a blanket treatment for all derangements encountered.

If pre-existing pathologies are not investigated and managed, there will be a high probability for the recurrence of CPA. However, this is somewhat different if CPA was due anaesthetic or drug-related involvement. Gilroy et al stated that anaesthetised cats have 36% survival rate compared to ICU ill cats that have a 0% survival rate,²⁵ although the incidence of CPA during anaesthesia or sedation has been reported by Brodbelt et al to be 0.24% for cats.²⁶ Rearrests were reported in Wingfield and Van Pelt²⁷ and McIntyre et al⁵ at 37.5% for cats and 30% in cats and dogs, respectively. Euthanasia was a key factor in cases of rearrest.

Limitations with this case were inadequate investigation into possible breed predisposed pathologies. Outdated CPR interventional knowledge was applied with regard to certain drugs. No access to a benchtop blood gas analyser because it was in for repairs. No postmortem was performed.

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