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CASE REPORT

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# Successful resuscitation and neurological monitoring of a dog with out-of-hospital cardiopulmonary arrest due to pentobarbital overdose

**Claudia Iannucci Dr med vet, DACVECC, DECVECC[1] | Sabrina N. Hoehne Dr med vet, DACVECC, DECVECC[2] | Vishal D. Murthy DVM, DACVIM[2] | Guillaume Dutil Dr med vet[3] | Arianna Maiolini DVM, PhD, DECVN[3]**

[1]Department of Clinical Veterinary Medicine, Division of Small Animal Emergency and Critical Care, Vetsuisse Faculty, University of Zurich, Zurich, Switzerland

[2]Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Washington State University, Pullman, Washington, USA

[3]Department of Clinical Veterinary Medicine, Division of Neurology, Vetsuisse Faculty, University of Bern, Bern, Switzerland

**Correspondence**
Sabrina N. Hoehne, Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Washington State University, 205 Ott Rd, Pullman, WA 99164, USA.
Email: sabrina.hoehne@wsu.edu

## Abstract

**Objective:** To describe the clinical signs, electroencephalographic (EEG) findings, treatment, and outcome in a dog after successful resuscitation from out-of-hospital cardiopulmonary arrest (OHCA) induced by pentobarbital intoxication.

**Case Summary:** A 10-year-old, male intact Jack Russell Terrier was referred for management of refractory status epilepticus and presented dead on arrival. After 7 minutes of cardiopulmonary resuscitation, return of spontaneous circulation was achieved, but the dog remained comatose, apneic, and lacked brainstem reflexes on neurological examination 6 hours following resuscitation. Magnetic resonance imaging showed polioencephalomalacia consistent with prolonged epileptiform activity, and EEG was initially concerning for electrocerebral inactivity. Following supportive care that included short-term mechanical ventilation, the dog made a full recovery and was discharged from the hospital alive 7 days postresuscitation. It was later revealed that the dog had been administered an unknown amount of pentobarbital during transportation, which likely contributed to the OHCA, clinical, and EEG findings.

**New Information Provided:** This is the first report to describe the full recovery and hospital discharge of a dog suffering OHCA and the first description of EEG findings in a clinical veterinary patient following cardiopulmonary arrest and successful resuscitation. Factors likely contributing to successful patient outcome and potential benefits and limitations of EEG in monitoring postcardiac arrest patients are discussed.

KEYWORDS
brainstem auditory-evoked response, electroencephalography, out-of-hospital cardiopulmonary arrest, pentobarbital intoxication, survival to hospital discharge

**Abbreviations:** BAER, brainstem auditory-evoked response; CPA, cardiopulmonary arrest; CRI, constant rate infusion; ECI, electrocerebral inactivity; EEG, electroencephalography; MRI, magnetic resonance imaging; OHCA, out-of-hospital cardiopulmonary arrest; OU, oculus uterque (both eyes); PEEP, positive end-expiratory pressure; RI, reference interval; ROSC, return of spontaneous circulation; SE, status epilepticus.

Sabrina N. Hoehne and Vishal D. Murthy contributed equally to this work.

## 1 | INTRODUCTION

Despite recent advances in the field of small animal CPR, outcomes of dogs suffering cardiopulmonary arrest (CPA) remain poor.[1] Fewer than 10% of dogs survive and, to date, there are no reports of dogs experiencing out-of-hospital cardiopulmonary arrest (OHCA) and being discharged from the hospital alive.[1] The proportion of dogs that achieve return of spontaneous circulation (ROSC) following CPR is substantially higher, but almost two thirds of initially successfully resuscitated dogs are subsequently euthanized due to poor prognosis, cost associated with intensive post-CPA care, or a combination thereof.[2]

Predictors of patient outcome for post-CPA prognostication are understudied in veterinary medicine and, because of the lack of patients surviving to hospital discharge, have not been established for dogs suffering OHCA. Nevertheless, a better understanding of factors associated with positive patient outcomes would be helpful to aid in client communication and decision-making regarding post-CPA care. In human medicine, electroencephalography (EEG) is widely used for monitoring and prognostication in post-CPA patients, with nonconvulsive epileptiform activity, burst-suppression pattern, low-voltage activity, and electrocerebral inactivity (ECI) each being associated with a poor outcome.[3–6] EEG remains underutilized in veterinary medicine and is primarily used to confirm epileptic seizures.[7] While not previously reported, it is possible that EEG also may help monitor neurological status and direct therapeutic plans in veterinary post-CPA patients.

The case described here documents the successful resuscitation and survival to hospital discharge of a dog following OHCA suspected to have been induced by pentobarbital overdose. It is furthermore the first report of the clinical use of EEG for neurological assessment in the post-CPA phase of a dog.

## 2 | CASE PRESENTATION

A 10-year-old, male intact Jack Russel Terrier, weighing 10 kg, was referred to the Emergency Service of the University of Bern Small Animal Hospital for refractory status epilepticus (SE). The dog first experienced cluster seizures 6 months earlier and based on normal CBC, biochemistry panel, and thoracic and abdominal radiographs, a presumptive diagnosis of idiopathic epilepsy (epilepsy of unknown cause) was made and phenobarbital therapy (1.5 mg/kg, PO, q 12 h) initiated.[8] Twenty hours prior to referral, the dog was presented to its primary care veterinarian for cluster seizures that progressed to SE. CBC and biochemistry panel were unremarkable, except for mild hyperglycemia (8.5 mmol/L [153 mg/dL]; reference interval [RI]: 3.3–6.1 mmol/L [60–110 mg/dL]). Benzodiazepine and anticonvulsant therapy (unknown medications and dosages) were initiated but did not terminate the SE, and the dog was referred for further management. Transportation was arranged through a chauffeur service, and the dog was connected to a free dripping, unlabeled bag of IV fluids during the 2-hour car ride.

On presentation to the emergency department, the dog was nonresponsive and apneic and had cyanotic mucous membranes, and cardiac sounds were absent on auscultation. CPR was initiated according to the Reassessment Campaign on Veterinary Resuscitation (RECOVER) guidelines.[9] Chest compressions with rescuers rotating every 2 minutes were performed at 120/min, and manual ventilation with an $FiO_2$ of 1.0 through orotracheal intubation was provided at 10/min. Capnometry[a] and ECG monitoring were initiated. Cardiac arrest rhythms were analyzed during intercompression pauses and showed asystole throughout CPR efforts. The only end-tidal carbon dioxide measurement obtained was 0 mm Hg. The transport fluid bag was disconnected from the dog's IV catheter, and epinephrine[b] (0.01 mg/kg) and atropine[c] (0.1 mg/kg, IV) were administered once.

During the fourth cycle of basic life support, an increase in end-tidal carbon dioxide to 33 mm Hg and strong femoral pulses consistent with ROSC were noted. Chest compressions were discontinued, and ECG revealed a sinus tachycardia of 180/min. Lingual oxygen saturation was 88%, and Doppler blood pressure was 70 mm Hg. Manual ventilation was continued, and 15 mL/kg (IV) of isotonic crystalloids[d] was administered, which restored normotension. An abbreviated neurological examination revealed the dog to be comatose with bilateral, nonresponsive mydriasis and absent brainstem reflexes. Modified Glasgow Coma Scale score was 3, and though not validated for monitoring of the post-CPA patient, was used to track neurological status over time (Table 1). The head was elevated 30° degrees, and mannitol[e] (1 g/kg, IV over 20 minutes) was administered to treat possible intracranial hypertension post-CPA and prolonged seizure activity. Rectal temperature was 37°C (98.6°F). Active cooling measures were not applied, but patient orders included no active warming unless shivering was noted. An arterial blood sample was obtained and revealed a pH of 7.24 (RI: 7.32–7.44), $PaO_2$ of 382 mm Hg (RI: 85–95 mm Hg), $PaCO_2$ of 39.1 mm Hg (RI: 36–40 mm Hg), and bicarbonate concentration of 17.0 mmol/L (RI: 19–24 mmol/L). Results of a CBC and biochemical analyses are summarized in Tables 2 and 3.

Despite cardiovascular stabilization, apnea and absent brainstem reflexes persisted, necessitating continuation of manual ventilation. Mechanical ventilation[f] was initiated 1.5 hours post-ROSC, using a pressure-controlled mode with a positive end-expiratory pressure (PEEP) of 5 cm $H_2O$, pressure above PEEP between 5 and 7 cm $H_2O$ to target tidal volumes between 6 and 10 mL/kg, $FiO_2$ of 0.8, and respiratory rate of 12–20/min. No sedative or anesthetic agents were required to maintain intubation. Two hours post-ROSC, magnetic resonance imaging (MRI) of the brain was pursued and revealed an area of cortical thinning and widened sulci within the left parietal and occipital lobes. The white matter showed T1W hypointense and T2W hyperintense signal and incomplete suppression in FLAIR. The cingulate gyrus appeared hyperintense on T2W and FLAIR and hypointense on T1W images. The left lateral ventricle was enlarged. Mild contrast enhancement of the meninges overlying the affected areas of the left parietal and occipital lobes was noted. A CSF sample collected from the cerebellomedullary cistern revealed normal protein concentrations (0.26 g/L [2.6 g/dL]; RI: 0.08–0.3 g/L [0.8–3.0 g/dL]) and total nucleated cell count (0.7 cells/$\mu$L; RI: 0–5 cells/$\mu$L; differential cell count: 19% lymphocytes,

**TABLE 1** Modified Glasgow Coma Scale score over time following return of spontaneous circulation (ROSC) in a dog experiencing out-of-hospital cardiopulmonary arrest and undergoing CPR.

| Modified Glasgow Coma Scale | Immediately post-ROSC | 12 hours post-ROSC | 24 hours post-ROSC | 36 hours post-ROSC | 48 hours post-ROSC | 60 hours post-ROSC | 72 hours post-ROSC |
|---|---|---|---|---|---|---|---|
| Motor activity | 1 | 1 | 4 | 4 | 5 | 5 | 6 |
| Brainstem reflexes | 1 | 3 | 4 | 5 | 5 | 6 | 6 |
| Level of consciousness | 1 | 2 | 3 | 5 | 5 | 5 | 6 |
| Total score | 3 | 6 | 11 | 14 | 15 | 16 | 18 |

Abbreviation: ROSC, return of spontaneous circulation.

**TABLE 2** Complete blood count results over time in a dog experiencing out-of-hospital cardiopulmonary arrest and undergoing CPR following return of spontaneous circulation (ROSC).

| Parameter | 1 hour post-ROSC | 24 hours post-ROSC | 72 hours post-ROSC | Institutional reference interval |
|---|---|---|---|---|
| HCT (%) | 52 | 44 | 41 | 39–57 |
| Total hemoglobin (g/L) | 172 | 146 | 142 | 138–204 |
| Erythrocytes (×1012/L) | 7.20 | 6.29 | 6.02 | 5.7–9.1 |
| Thrombocytes (×109/L) | 172 | 181 | 215 | 150–400 |
| Leukocytes (×109/L) | 11.82 | 10.47 | 9.27 | 6–12.0 |
| Neutrophils segmented (×109/L) | 10.34 | 9.16 + **toxic changes** | 7.37 | 3.0–11.5 |
| Band neutrophils (×109/L) | **0.53** | **0.42** | 0.05 | 0.0–0.3 |
| Lymphocytes (×109/L) | 0.53 | 0.68 | 1.21 | 1.0–4.8 |
| Monocytes (×109/L) | 0.35 | 0.21 | 0.51 | 0.15–1.35 |
| Eosinophils (×109/L) | 0.00 | 0.00 | 0.14 | 0.1–1.25 |
| Basophils (×109/L) | 0.00 | 0.00 | 0.00 | 0.0–0.04 |

*Note*: Bolded values indicate values outside of the institutional reference interval.
Abbreviation: ROSC, return of spontaneous circulation.

**TABLE 3** Results of serum biochemical testing over time in a dog experiencing out-of-hospital cardiopulmonary arrest and undergoing CPR following return of spontaneous circulation (ROSC).

| Parameter | 1 hour post-ROSC | 24 hours post-ROSC | 72 hours post-ROSC | Institutional reference interval |
|---|---|---|---|---|
| Sodium (mmol/L / mEq/L) | **162** | 152 | 142 | 142–154 |
| Chloride (mmol/L / mEq/L) | **127** | **122** | 104 | 106–118 |
| Potassium (mmol/L / mEq/L) | **3.52** | 4.05 | 4.32 | 3.95–5.40 |
| Phosphorus (mmol/L) (mg/dL) | **3.14 (9.72)** | – | 1.35 (4.18) | 0.91–1.90 (2.82–5.88) |
| Total calcium (mmol/L) (mg/dL) | **2.32 (9.28)** | – | **2.27 (9.08)** | 2.42–2.85 (9.68–11.4) |
| Glucose (mmol/L) (mg/dL) | **4.13 (74.41)** | 5.43 (96.22) | – | 4.16–6.69 (74.95–120.54) |
| Total protein (g/L) (g/dL) | **51.0 (5.1)** | – | – | 55–73 (5.5–7.3) |
| Albumin (g/L) (g/dL) | 29.9 (2.99) | 25.1 (2.51) | 24.2 (2.42) | 20–41 (2.0–4.1) |
| Blood urea nitrogen (mmol/L) (mg/dL) | 8.03 (22.49) | – | 4.83 (13.53) | 3.30–10.83 (9.24–30.34) |
| Creatinine (μmol/L) (mg/dL) | 95 (1.07) | 67 (0.76) | 77 (0.87) | 52–117 (0.59–1.32) |
| Bilirubin (μmol/L) (mg/dL) | **8.3 (0.49)** | **4.1 (0.24)** | – | 0.5–3.9 (0.03–0.23) |
| Alanine aminotransferase (IU) (μkat/L) | **478 (7.98)** | – | – | 26–126 (0.43–2.1) |
| Aspartate aminotransferase (IU) (μkat/L) | **228 (3.81)** | – | – | 22–76 (0.37–1.27) |
| Creatinine kinase (IU) (μkat/L) | **1131 (18.89)** | – | – | 64–400 (1.07–6.68) |
| Canine C-reactive protein (mg/L) (nmol/L) | **79.7 (759.06)** | **128.6 (1224.79)** | **11.4 (108.57)** | <10.7 (<101.91) |

*Note*: Bolded values indicate values outside of the institutional reference interval.
Abbreviation: ROSC, return of spontaneous circulation.

40% monocytes). The observed MRI changes were believed to represent edema and polioencephalomalacia, likely secondary to prolonged epileptiform activity. Based on these findings, a diagnosis of idiopathic epilepsy (epilepsy of unknown cause) was made.[7,8]

Six hours post-ROSC, apnea and absent brainstem reflexes persisted. An EEG was observed for 30 minutes, recorded with a commercial electrodiagnostic recording system[g] and subdermal electrodes[h] positioned as previously described.[10] Electrode layout was as follows: Fp1/Fp2, left and right prefrontal (frontopolar); F3/F4, left and right frontal; P3/P4, left and right parietal; O1/O2, left and right occipital; T3/T4, left and right temporal; Fz, midline frontal; Cz, central midline (vertex); Oz, midline occipital. A ground electrode was placed in the dorsal cervical region. Impedance prior to recording did not exceed 5 kΩ. The EEG was recorded with low- and high-frequency filters of 5 and 70 Hz, respectively, with an amplifier sensitivity of 10 $\mu$V/mm, and paper speed of 30 mm/s. The EEG tracings revealed a flatline with intermittent artifacts that were concomitant with prescribed positive pressure respiratory rate (Figure 1A). No evidence of nonconvulsive SE was identified. The owner was informed that, despite the current clinical and EEG findings, more conclusive prognostication might not be possible until 72 hours post-ROSC, and it was decided to continue care overnight.

Upon updating the referring veterinarian, it was revealed that the dog had received pentobarbital 5 mg/kg (IV) once and then been maintained on a pentobarbital constant rate infusion (CRI, 0.5–2.0 mg/kg/h) overnight prior to referral. Pentobarbital was furthermore added to the bag of isotonic crystalloids that were administered during transport. In light of this, a serum sample was collected approximately 7 hours following presentation and revealed a serum pentobarbital concentration of 16.7 mg/L. Extracorporeal toxin removal was discussed with the client but was declined due to cost. IV lipid emulsion was not administered due to concerns for nonspecific serum anticonvulsant depletion and risk of recurrent SE. Brainstem auditory-evoked response (BAER) testing was pursued to further evaluate brainstem function and revealed intact, normal waveforms bilaterally. The dog was admitted to the ICU for continued mechanical ventilation, supportive care, and clinical and neurological monitoring. Medications included Plasmalyte[4] supplemented with 30 mmol/L potassium chloride[i] (2 mL/kg/h, IV) and maropitant[j] (1 mg/kg, IV, q 24 h).

Eight hours post-ROSC, the dog's pupils remained nonresponsive to light, though changed from bilaterally mydriatic to miotic. Spontaneous attempts at inspiration were noted, and ventilator support was changed to pressure-controlled synchronized intermittent mandatory ventilation with PEEP of 4 cm $H_2O$, pressure above PEEP 6 cm $H_2O$ for assisted breaths, $FiO_2$ of 0.65, and respiratory rate of 10–12/min. Ten hours post-ROSC, spontaneous respiratory rate increased >20/min, and bilaterally intact palpebral reflexes and responsive miotic pupils were seen. Ventilation mode was changed to continuous spontaneous ventilation with continuous positive airway pressure of 2–4 cm $H_2O$. Eleven hours post-ROSC, the dog could be weaned from the ventilator but, due to an absent gag reflex, remained intubated on flow-by oxygen (2 L/min). Levetiracetam[k] therapy (loading dose 60 mg/kg, IV, followed by 20 mg/kg, IV, q 8 h) was initiated. Two hours after ventilator weaning, focal facial muscle twitching was noted, and a single dose of phenobarbital[l] (2 mg/kg, IV) was administered to no effect. A midazolam[m] CRI (0.2 mg/kg loading dose, followed by 0.25 mg/kg/h, IV) was started, and the twitching resolved but recurred 2 hours later. A midazolam bolus (0.2 mg/kg, IV) was repeated, the CRI increased to 0.45 mg/kg/h (IV), and phenobarbital administration scheduled as 3 mg/kg (IV, q 12 h), successfully resolving the facial twitches.

Twenty hours post-ROSC, the dog could be extubated and maintained normoxemia on flow-by oxygen via face mask. Its clinical condition remained stable, except for a body temperature of 39.1°C (102.4°F) and corneal ulcers in both eyes (OU). A progressive left shift and increase in canine C-reactive protein were documented on recheck bloodwork (Tables 2 and 3). Thoracic radiographs and repeat EEG were declined due to the overall improving condition of the dog. Ampicillin–sulbactam[n] (30 mg/kg, IV, q 8 h) was empirically initiated for suspected aspiration pneumonia, and vitamin A[o] and tobrexidine eye ointment[p] (1 cm strip, OU, q 4 h) were added to the treatment regimen. Thirty hours post-ROSC, 1 self-limiting generalized seizure was noted, and the midazolam CRI was increased to 0.6 mg/kg/h (IV).

Forty-eight hours post-ROSC, the dog's mentation improved markedly, and it subsequently began to eat and drink voluntarily. The midazolam CRI was reduced to 0.3 mg/kg/h (IV), and a complete neurological examination revealed mild obtundation and disorientation, generalized ataxia, proprioceptive deficits in all limbs, and an absent menace response in the right eye. The remainder of the neurological examination was unremarkable. Over the next 3 days, the dog's general and neurological condition continued to improve. The midazolam CRI was discontinued on the fourth day of hospitalization, and no further seizures were observed. The dog was discharged from the hospital 7 days post-ROSC on phenobarbital[q] (3 mg/kg, PO, q 12 h), levetiracetam[r] (25 mg/kg, PO, q 8 h), amoxicillin–clavulanic acid[s] (12.5 mg/kg, PO, q 12 h), tobrexidine eye drops[t] (1 drop, OU, q 6 h), and vitamin A eye ointment[15] (1 strip, OU, q 8 h). One week following discharge, a recheck examination at the primary care veterinarian revealed the dog to have normal mentation and behavior without any further seizures. The corneal ulcerations and leukocytosis were resolved, and ocular treatments and amoxicillin–clavulanic acid were discontinued.

Thirteen months after initial presentation, the dog was presented to the Neurology Service at the University of Bern for evaluation of increased seizure frequency to once a week. The dog's medication regimen consisted of levetiracetam (20 mg/kg, PO, q 8 h) and, due to owner misunderstanding, phenobarbital was administered only once since discharge. Neurological examination revealed no abnormalities, and repeat MRI showed static to slightly progressive left parietooccipital polioencephalomalacia with no new intracranial lesions. Treatment recommendations were revised to include phenobarbital (2.5 mg/kg, PO, q 12 h) and levetiracetam (20 mg/kg, PO, q 8 h). No further follow-up information was available at the time of manuscript preparation.

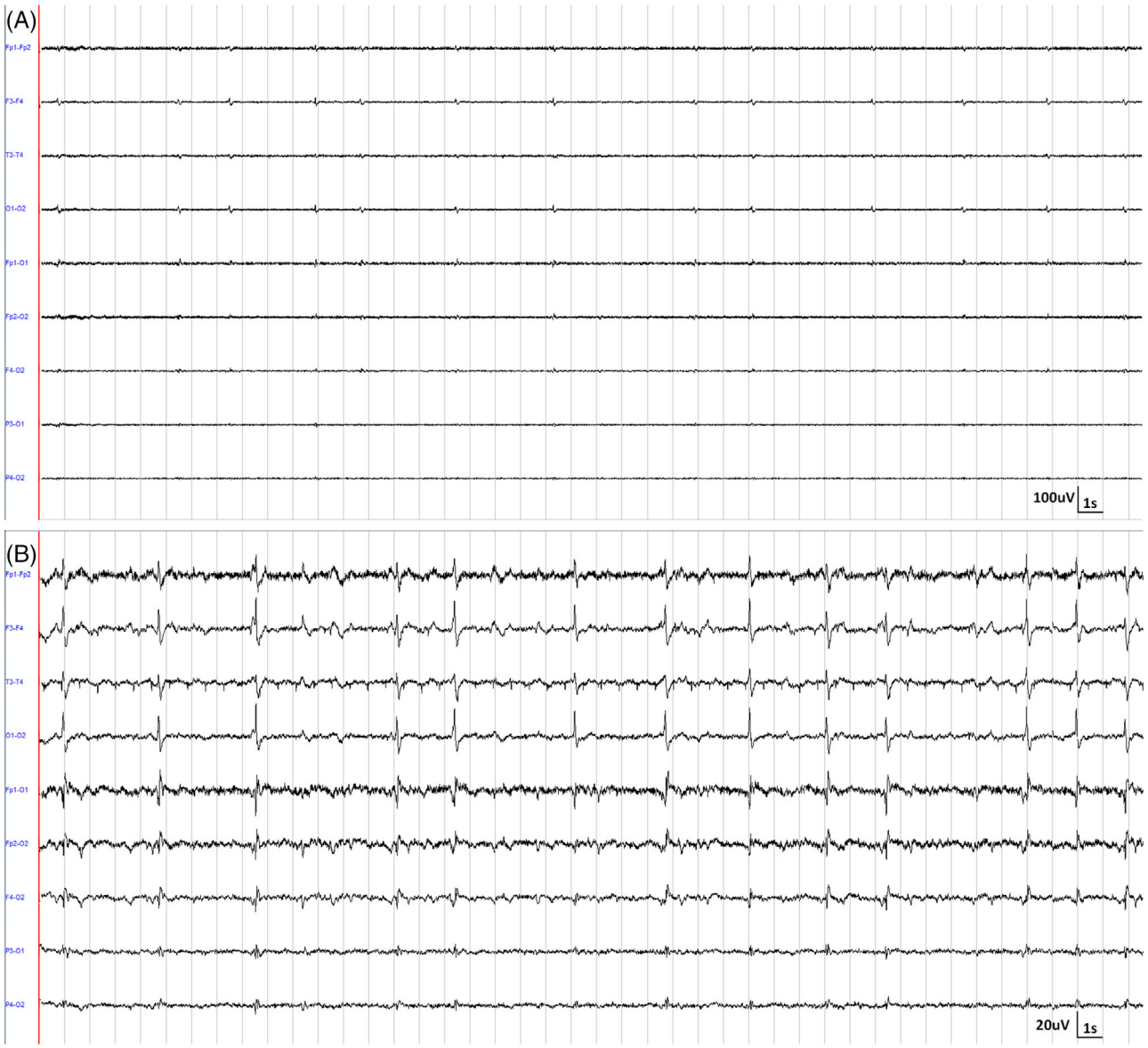


**FIGURE 1** (A) Bipolar montage of electroencephalographic (EEG) tracings obtained in a dog suffering cardiopulmonary arrest 6 hours post-return of spontaneous circulation, at original recording settings (sensitivity 10 $\mu$V/mm, low-frequency filter 5 Hz, high-frequency filter 70 Hz) and paper speed of 1 cm/s. Apparent diffuse flatline with artifacts at 20/min, suspected to be respiratory in origin, is seen. (B) Bipolar montage of EEG tracing post hoc displayed per criteria for electrocerebral inactivity (ECI) diagnosis (sensitivity 2 $\mu$V/mm, low-frequency filter 0.5 Hz, high-frequency filter 30 Hz) and paper speed of 1 cm/s. Diffuse low-voltage background beta activity is seen with interspersed artifacts at 20/min, suspected to be respiratory in origin.

## 3 | DISCUSSION

This report describes the successful recovery and hospital discharge of a dog suffering OHCA due to pentobarbital overdose. Additionally, it is the first reported use of EEG in the post-CPA clinical canine patient and highlights its potential in gaining a more comprehensive neurological patient assessment as well as pitfalls associated with test timing and the underlying cause of CPA.

Several studies investigating veterinary CPR outcomes report that no dogs or cats suffering OHCA survived to hospital discharge, and factors associated with increased odds of survival after OHCA remain unknown.[1,11,12] CPA occurring while under general anesthesia, due to relative or absolute drug overdose, witnessed CPA, and IV access already established at the time of CPA are the most commonly reported factors associated with increased rates of ROSC and survival following in-hospital CPA.[1,11,12] Even though not specifically established for dogs experiencing OHCA, it appears likely that the dog described in this report had a positive outcome because CPA occurred secondary to drug overdose and that the presence of an IV catheter allowed undistracted initiation of basic life support and prompt administration of vasopressor therapy.

Pentobarbital is a potent short-acting barbiturate that finds potential use as an anesthetic, anticonvulsant, and euthanasia agent in veterinary medicine.[13] Due to substantial side effects of severe respiratory depression and systemic hypotension, its primary use is humane euthanasia in animals.[13] Inadvertent intoxication is rare and has mostly been reported following consumption of carcasses euthanized with pentobarbital.[14] Depending on the ingested dose, signs may vary from sedation, generalized ataxia, and coma to death. While intoxicated dogs have been reported to recover from a coma, there are no reports of successful resuscitation and long-term survival following death from pentobarbital intoxication.[14]

As pentobarbital is rarely used as an anticonvulsant these days, unless specified in the treatment history, it may not be considered as a potential toxicant. The $LD_{50}$ of pentobarbital in dogs is reported to be 40–60 mg/kg (IV) and in people is 35 mg/L.[13–15] In this case, a serum pentobarbital concentration of 16.7 mg/L was obtained approximately 7 hours following pentobarbital discontinuation. Assuming zero-order kinetics and a hepatic metabolism rate of 15% per hour in dogs, the serum pentobarbital concentration at presentation was estimated to be approximately 44.4 mg/L, which exceeds the human lethal dose and was likely the cause of CPA.[14,15] Additional evidence includes the lack of other systemic disease, the lack of brainstem herniation on MRI, and the clinical course of recovery with supportive care, which is in accordance with previous reports of pentobarbital intoxication in dogs and people.[14,15] Lastly, the EEG findings also support the suspicion of pentobarbital being the cause of CPA. Pentobarbital has been shown to suppress EEG activity, including induction of ECI in dogs, although at higher doses than those measured in this case.[16] Given the prolonged period of unchecked pentobarbital administration during transport and preferential distribution to the brain, the peak brain pentobarbital concentration is likely to have been significantly higher than the estimated serum concentration, which resulted in the CNS depression culminating in CPA. Decontamination techniques like IV lipid emulsion administration and hemodialysis have been reported to be effective in barbiturate intoxication in both human and veterinary patients.[17,18] While both were initially considered in this case, hemodialysis was declined due to cost. IV lipid emulsion therapy was not pursued due to the risk of recurrent refractory seizures through the nonselective removal of lipophilic anticonvulsants and previous reports of prolonged narcoleptic effects of pentobarbital.[19]

Following brain ischemia and resuscitation, it can be challenging to assess the full extent of neurological injury based on neurological examination alone. Adjunctive multimodal diagnostic approaches, including EEG and BAER, may be useful in providing a more detailed and comprehensive assessment of the CNS for neurological monitoring and should be considered more often in veterinary patients. The World Brain Death Project and the American Heart Association advocate a similar multimodal approach to neurological monitoring of the post-CPA patient and recommend that neurological testing and decisions regarding withdrawal of life support only be performed after 24–72 hours post-ROSC, respectively, as there is no method to reliably identify patients with a poor functional outcome at earlier timepoints.[20–23] Although no data on neurological outcomes of canine CPA survivors are available, veterinary CPR guidelines in accordance with human guidelines currently suggest care for at least 72 hours post-ROSC prior to attempting conclusive prognostication.[24]

Despite these recommendations, there is some evidence in people that specific EEG patterns, such as ECI, have prognostic significance even <24 hours post-ROSC.[25,26] At 6 hours post-ROSC, as performed in our case, ECI in people would be suggestive of brain death and associated with a probability of a poor outcome in >50% of patients.[25]

While at initial recording and interpretation, the EEG findings were concerning for ECI, not all criteria for ECI diagnosis established by the American Clinical Neurophysiology Society were met.[27] These include (1) a complete complement of scalp electrodes; (2) interelectrode impedances >100 and <10,000 Ω; (3) testing of the integrity of the recording system; (4) electrode pairs at least 10 cm apart; (5) sensitivity of 2 $\mu$V/mm for at least 30 minutes; (6) high-frequency filter ≥30 Hz, low-frequency filter ≤1 Hz; (7) clarification of artifacts; (8) lack of reactivity to intense stimuli; (9) recordings performed by qualified technologists; (10) repeated EEG recordings parameters; and (11) documentation of medications and physiological variables.[27] At initial recording, criteria 1–3 were met, but given the patient's small skull size, interelectrode distances of 10 cm were not feasible. Criteria 8–10 were followed, and criterion 11 could not be met at the time due to the lack of knowledge of pentobarbital administration. In order to meet criteria 5–7, a post hoc review of the EEG with sensitivity of 2 $\mu$V/mm, low filter 0.5 Hz, and high filter 30 Hz settings was performed, revealing diffuse low-voltage beta waves (Figure 1B) similar to those reported in post-CPA anoxic brain injury.[6] While these changes could reflect transient hypoxic injury, they are considered more likely to also reflect the pentobarbital overdose, given the patient's clinical progression.

In the dog described in this study, the EEG was performed before the history of pentobarbital administration was available. While pentobarbital intoxication negates the prognostic significance of the documented EEG findings, they remained helpful to exclude nonconvulsive SE as a cause for the patient's clinical signs and in formulating an appropriate patient treatment plan.

This case illustrates the successful resuscitation and survival to hospital discharge of a dog following OHCA. It highlights the challenges of neurological monitoring and illustrates the inaccuracies of testing brainstem function through clinical examination alone. While additional testing such as concurrent EEG and BAER can aid in patient monitoring and provide a more detailed neurological assessment, prognostication should ideally be delayed for 24–72 hours post-ROSC. While confounded by pentobarbital administration in this case, EEG may have utility in patient monitoring and prognostication post-ROSC, but the ideal timing in veterinary patients has yet to be prospectively evaluated. In the meantime, single EEG recordings within the first 72 hours post-ROSC should be interpreted with significant caution, especially in cases of OHCA where toxicant exposure may be unknown, and concurrent EEG and BAER testing may be preferable.

## CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

## ORCID

*Claudia Iannucci Dr med vet, DACVECC, DECVECC* https://orcid.org/0000-0002-6976-7627

*Sabrina N. Hoehne Dr med vet, DACVECC, DECVECC* https://orcid.org/0000-0002-0884-6268

*Vishal D. Murthy DVM, DACVIM* https://orcid.org/0000-0002-2879-9593

## ENDNOTES

[a] Zoll M series CCT including LoFlo $ETCO_2$ module, Zoll Medical Corporation, Chelmsford, MA

[b] Adrenalin Sintetica, 1 mg/mL, Sintetica SA, Mendrisio, Switzerland

[c] Atropinsulfat, 1 mg/mL, Bichsel, Laboratorium Dr. G. Bichsel AG, Unterseen, Switzerland

[d] Plama-Lyte A, Baxter AG, Opfikon, Switzerland

[e] Mannitol 20%, Bichsel, Laboratorium Dr. G. Bichsel AG, Unterseen, Switzerland

[f] Maquet Servo-i, Siemens Schweiz AG, Rheinfelden, Switzerland

[g] Lifelines Trackit Mk3 ambulatory EEG recorder, Lifelines Neuro Company, LLC, Louisville, KY

[h] Natus Neurology, length 14 mm, diameter 0.38 mm for temporal electrodes; length 12 mm, diameter 0.4 mm for the remainder, Natus Medical Inc, Pleasanton

[i] Kaliumchlorid (potassium chloride) 15%, Bichsel, Laboratorium Dr. G. Bichsel AG, Unterseen, Switzerland

[j] Cerenia, Zoetis Schweiz GmbH, Delémont, Switzerland

[k] Keppra, 100 mg/mL (5 mg/5 mL), UCB-Pharma SA, Bulle, Switzerland

[l] Phenobarbital 10%, 100 mg/mL, Laboratorium Dr. G. Bichsel AG, Interlaken, Switzerland

[m] Midazolam Sintetica, 50 mg/10 mL-5 mg/mL-Sintetica SA, Mendrisio, Switzerland

[n] Ampicillin plus sulbactam eberth, 1 g/0.5 g, Dr. Friedrich Eberth Arzneimittel GmbH, Ursensollen, Germany

[o] Vitamin A, Bausch & Lomb Swiss AG, Zug, Switzerland

[p] Tobrex, 3.5 g, Novartis PharmaSchweiz AG, Risch, Switzerland

[q] Aphenylbarbit, 15 mg, Streuli Pharma AG, Uznach, Switzerland

[r] Keppra, 100 mg, UCB-Pharma SA, Bulle, Switzerland

[s] Clavaseptin, 250 mg, Vetoquinol GmbH, Ismaning, Germany

[t] Tobrex, 3 mg/mL (5 mL), Novartis PharmaSchweiz AG, Risch, Switzerland

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