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Echocardiographic Examination of 16 Eastern Grey Kangaroos (*Macropus giganteus*) and One Red Kangaroo (*Osphranter rufus*)

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Habitat loss, road trauma, predation, disease, and natural disasters impact the health and survival of the family Macropodidae, including kangaroos. Cardiac disease has been reported, including hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), nutritional myodegeneration, valvular pathology, cardiovascular parasites, toxoplasmosis, and toxicities. Human research has evaluated macropod pericardium and aortic valves as possible bioprostheses. The goals of this prospective anatomic study were to echocardiographically evaluate opportunistically presented kangaroos: 14 clinically normal eastern grey kangaroos (EGK—*Macropus giganteus*), two sick EGKs, and one sick red kangaroo (RK, *Osphranter rufus*). Similar techniques as described in the dog were used. Standard B-mode images, M-mode mensuration, and Doppler measurements were attained; values were descriptively compared with published normal values. The clinically normal animals' M-mode values were similar to the closest weight-related kangaroo values. Most of the animals showed thicker-than-expected left ventricular and interventricular septal walls, and relative wall thickness (RWT) of 0.5 and 0.6; this may be the norm for macropods, but since an RWT>0.45 may indicate human HCM, this should be considered in the kangaroo. The sick animals were euthanized. Necropsy revealed highly suspect HCM in one EGK, and myxomatous mitral valve degeneration with suspect DCM in the other EGK and RK. In conclusion, there are weight-related similarities between previously published kangaroo values. More work is required on a larger number of weight and age cohorts of kangaroos. Subclinical HCM may be present in apparently normal animals. Findings can be used during clinical health assessments and for further research into macropod cardiac conditions.

1 | Introduction

The Macropodidae family of marsupials encompasses several groups, including the kangaroo, wallaby, and pademelon. The family is made up of diverse species occupying a range of habitats, with the smallest musky-rat kangaroo (*Hypsiprymnodon moschatus*; body mass (BM): 500 g) inhabiting rainforests and the largest red kangaroo (*Osphranter rufus;* BM: 90 kg) inhabiting arid desert regions [1]. They are endemic to Australia and New Guinea, and many are threatened or endangered, with six species declared extinct. They are under threat from habitat destruction, pollution, feral carnivores, feral and introduced competitors, road accidents, and climate change [2].

Various cardiac diseases in the macropod have been described [3], with hypertrophic cardiomyopathy (HCM) being one of the more common conditions. Ten of 14 Australian rock kangaroos (*O. robustus*) died unexpectedly under anesthesia,

secondary to ventricular fibrillation. The electrical abnormalities and left ventricular hypertrophy found at postmortem, were concluded to be most likely due to HCM. It was suggested that the kangaroo may be used as a model for HCM studies in humans [4]. Hypertrophic cardiomyopathy was also diagnosed postmortem in two of three captive adult Bennett's wallabies (*Macropus rufogriseus rufogriseus*) [5]. Sugishita et al. [6] used the echocardiographic-derived relative wall thickness ratio in diastole (RWT = (LVFWd+IVSd)/LVIDd) in seven apparently clinically normal red kangaroos (RK, *O. rufus*) and determined they likely had HCM, since the value was similar to those in humans with HCM, where a RWT greater than 0.44 or 0.45 is deemed to be above normal [7].

Hypertrophic cardiomyopathy was successfully managed for eleven years in a Matschie's tree kangaroo (*Dendrolagus matchiei*) exhibiting cardiovascular and respiratory distress. Comparative serial echocardiographic evaluations of four apparently normal conspecific tree kangaroos were made [8].

Tree kangaroos (genus *Dendrolagus*) have been reported to have a high incidence of cardiomyopathies and valvular abnormalities, likely the cause of morbidity and mortality [9], and have been associated with increased post-anesthetic deaths [10]. Dilated cardiomyopathy in an antilopine kangaroo (*M. antilopinus*) [11] and degenerative cardiomyopathy in an eastern grey kangaroo (EGK—*M. giganteus*) have also been documented [12].

Cardiovascular nematodes, such as *Durikainema* and *Breinlia* species, have been reported in various macropods, causing cardiovascular conditions and serositis, often severe enough to cause constrictive pericarditis [13].

Fatal toxoplasmosis in macropods may include cardiomegaly with myocardial hemorrhage, pale streaks, myocardial necrotic foci, pericarditis, and mononuclear cardiac cell infiltration [13–15]

Toxicities such as sodium fluoroacetate (compound 1080) and mebendazole have been reported [13].

Species-spanning pathology between humans and, in the case of HCM, the kangaroo, and other mammals has been reported [16], and a one-health initiative also using HCM as an example was proposed for human and veterinary collaboration [17].

Investigation on the use of kangaroo aortic valves for use as bioprosthetic material in humans, due to the kangaroo's thinner valves and their more compact collagen structure compared with porcine valves, has been reported [18]. The absence of a right coronary cusp muscular shelf in kangaroos, which pigs have, is similar to that in humans [19]. Currently, the use of kangaroo pericardium for the manufacture of bioprosthetic valves in humans is under investigation [19].

There is a paucity of echocardiographic information in the kangaroo, with one study utilizing echocardiography in seven apparently normal kangaroos, thought to have subclinical HCM due to high RWT values [6], and another of a tree kangaroo with suspected HCM [8].

The current study aimed to prospectively acquire echocardiographic information to lay an initial descriptive foundation to address this lack of knowledge, thereby hoping to stimulate further research in this field. The goals of this study were to echocardiographically evaluate the hearts of opportunistically presented kangaroos, whether healthy or not, provide basic mensuration values, and to descriptively compare these to a reported set of apparently healthy weight-correlated kangaroo values. Pathological conditions found would also be reported.

2 | Materials and Methods

2.1 | Selection and Description of Subjects

This prospective anatomic study was carried out under the NSW National Parks and Wildlife Service Scientific Licence SL102465 and approved by the Charles Sturt University (CSU) Animal Care and Ethics Committee (A20247 & A20303). All persons involved in the study were vaccinated for Q fever.

Any live kangaroos of either sex, any age, whether healthy or not, and from the NSW Riverina area, were included in this study over 36 months during 2021–2024: 13 from a local wildlife care facility, one wild, and three from a local zoo. No sample size calculations were conducted. One of the unwell animals was presented from the wild, one from the local zoo and one from the rehabilitation center—they were recumbent, in poor condition, and were euthanized and examined postmortally and histologically by a registered wildlife veterinarian (author J. C.) and a final year pathology resident, respectively, under supervision of a Diplomate of the American College of Veterinary Pathology (Supporting Information S1). There were no exclusion criteria.

2.2 | Data Recording and Analysis

Kangaroos were captured and examined quietly, avoiding ambient temperature extremes. Two captive kangaroos were darted by a registered veterinarian (author J. C., with 35 years of wildlife experience) using a Pneu-Dart Model 196 tranquilliser firearm and disposable gel darts (Pneu-Dart Inc., Williamsport, PA, USA). The animal's weight was estimated, and it was then anaesthetised by the wildlife veterinarian (intramuscular injection of approximately 8 mg/kg Zoletil 100 (Virbac Australia Pty Ltd, Milpera, NSW, Australia)). A blind wild kangaroo (K4) was assessed from a distance, quietly captured with a blanket, transferred into a cloth "roo" bag, and sedated (approximately 1.0 mg/kg diazepam (Diazepam injection, Troy Laboratories Pty Ltd, Glendenning, NSW, Australia)) intramuscularly [20].

The clinically normal kangaroos were sedated for clinical examination, and blood samples for a full blood count, biochemistry, and cardiac troponins were collected, as well as feces (parasite egg count and identification, not reported in this manuscript) as part of the pre-release evaluation. The carer encouraged the kangaroo into the "roo" bag, and the animal was weighed (AgBoss, Albury, NSW, Australia). An intramuscular injection of diazepam (0.5–2 mg/kg) was given and repeated as required. Vital signs were monitored closely. A physical examination included

TABLE 1 | Comparative M-mode and B-mode (for LA, Ao, and PA) echocardiographic findings of the current 14 clinically normal eastern grey kangaroos (*Macropus giganteus*), and those of published normal kangaroo [6] data (grey shaded); measurements in mm

Number	Signalment	Weight, kg	IVS, d	IVS, s	LVFW, d	LVFW, s	LVID, d	LVID, s	RWT, d	FS	Ao	LA	LA:Ao, (1.05–07)	PA
K15	Juvenile M	8.3	7.1	10.1	7.3	10.81	23.5	13.5	0.61	0.43	12.6	12.9	1.02	13
ţ	NW	5.5	7		7		22	15	0.64	0.32				
K25	Young adult F	11.5	7.2	11.2	9.1	15.5	28	14.6	0.58	0.48	13.1	15.2	1.16	
K29	Young adult M	11.6	6.4	9.4	7.1	10.6	24.2	15.4	0.56	0.36	13.9	17.4	1.14	
K26	Young adult M	12.8	8	13.2	9	15.6	28.8	15.7	0.59	0.46	15.2	15.9	1.04	
K27	Young adult M	13.5	8	14.7	10.7	16.7	35.6	17.3	0.53	0.51	17.3	17.8	1.02	
K13	Young adult F	14	8.7	11.1	7	8.6	28.1	19.2	0.56	0.32	16.4	17.4	1.06	15.9
K28	Young adult M	14.5	7.5	10	8.8	11.1	29.6	17	0.55	0.43	14.8	17.9	1.2	
K24	Young adult M	14.7	10.4	9.7	11.6	13.7	29.8	20.8	0.74	0.3	17.1	21.2	1.24	15.1
ţ	NW	15	7		9		35	24	0.46	0.31				
K6	Young adult M	16	9.13	9.38	10.1	11.7	31.9	22.1	0.6	0.31	16.8	20.6	1.2	15.1
K14	Young adult F	16	7.9	12	9.4	13.4	30	16	0.58	0.47	19.3	19.5	1.01	17.9
ţ	NW	15	7		9		35	24	0.46	0.31				
K5	Adult F	22	10.5	11.6	6.1	9.8	31	21.7	0.54	0.30	16.9	19.5	1.15	15
K7	Adult M	22	12.1	14.2	10.4	13.7	38.3	24.1	0.59	0.37	22.4	26.5	1.18	17.2
K21	Adult F	22	9.1	10.3	12.2	14.9	38.9	28.9	0.55	0.26	22	22.5	1.02	17.9
ţ	NW	23	10		12		36	24	0.61	0.33				
Z3	Adult M	57	14.9	22.2	11.4	19	45.6	26.3	0.58	0.42	32.6	30.10	0.92	26.9
ţ	NW	48	15		15		42	31	0.71	0.26				

Note: Taken from ţ Sugishita et al. [6]. The FS and RWT values of Sugishita et al. [6] were calculated using the values provided in the article.

Abbreviations: Ao, aorta diameter; d, diastole; F, female; FS, fractional shortening; IVS, interventricular septum; LA, left atrial width at level of aortic sinus; LVFW, left ventricular free wall; LVID, left ventricular internal diameter; M, male; NW, nearest weight to kangaroo groups; RWT, relative wall thickness; s, systole.

TABLE 2 | Comparative M-mode echocardiographic findings of two sick eastern grey kangaroos (*Macropus giganteus*) (K4, K16), one red kangaroo (*Osphranter rufus*) (Z2), and those of published normal kangaroo [6] data (grey shaded); measurements in mm

Number	Signalment	Weight, kg	IVS, d	IVS, s	LVFW, d	LVFW, s	LVID, d	LVID, s	RWT, d	FS	Ao	LA	La:Ao Ref: (1.05-07)	PA
K16	Young adult F – ERM, suspect HCM	8.5	7.6	10.4	13.2	20.6	20.3	10.2	1.02	0.5	16.8	18.1	1.08	15.6
ţ	NW	11	9		9		31	24	0.59	0.23				
Z2	Adult F – suspect DCM	26.5	10.0	8.37	7.2	8.7	44.9	34.8	0.38	0.23	17.7	22.6	1.01	18.2
K4	Adult F – ERM, suspect volume overload	26.5	11.4	15.5	9.23	15.8	43.5	26.5	0.47	0.39	22.2	22	0.99	16.2
ţ	NW	23	10		12		36	24	0.61	0.33				

Note: Taken from ţ Sugishita et al. [6]. The FS and RWT values of Sugishita et al. [6] were calculated using the values provided in the article.

Abbreviations: Ao, aorta diameter; d, diastole; ERM, exertional rhabdomyolysis; F, female; FS, fractional shortening; IVS, interventricular septum; LA, left atrial width at level of aortic sinus; LVFW, left ventricular free wall; LVID, left ventricular internal diameter; M, male; NW, nearest weight to kangaroo groups; RWT, relative wall thickness; s, systole.

morphometrics (tail length, tail base circumference, pes length, head length, and weight), BCS (1–5, where 5 is obese), thoracic auscultation, rectal temperature, hydration status, oral cavity, skin examination, and visible injuries, if present. The animals were placed on a padded table, kept warm, their eyes covered, and noise was kept to a minimum. Two animals from the zoo were scanned outside on the ground due to logistical reasons.

Echocardiography was performed on all 17 animals; 15 were evaluated by a European College of Veterinary Diagnostic Imaging–registered radiologist (A. C.) and two by a second-year diagnostic imaging resident (A. B.), under the supervision of the radiologist.

Most of the examination was done with the kangaroos in right lateral recumbency, utilizing published dog and cat echocardiographic transducer positions and techniques [21]. The phased array transducer (4–7 MHz, GE Logic e, Radincon X-ray, South Creek Road Dee Why, NSW, Australia) was initially placed in the right third intercostal space moving it cranially or caudally one intercostal space to optimize the image, also using the point of maximal intensity for optimal transducer placement; if necessary, the animal was placed in left lateral recumbency particularly for the subcostal apical view. Seventy percent ethanol was used as a coupling agent, with no clipping of hair required. No electrocardiographic (ECG) coupling was used.

B-mode evaluation for gross pathology was performed, along with standard M-mode and Doppler evaluation as described for echocardiography in dogs [21]. Fractional shortening (FS) = ((LVIDd-LVIDs)/LVIDd) and RWT ((RWT = (LVFWd+IVSd)/LVIDd)) were determined. All measurements were made using the RadiANT DICOM viewer (Medixant, Poznan, Poland) by the European College of Veterinary Diagnostic Imaging diplomate.

The mensuration data were descriptively compared with previously published healthy kangaroo weight-compared results [6]. Any pathology present was described.

Due to the low number of animals and the variability in weights and their estimated ages, this was a descriptive study only, with no statistical analyses performed.

3 | Results

Fourteen of the 17 kangaroos were deemed clinically normal (eleven from the rehabilitation center; three from the zoo), and three were sick; of the latter, two were recumbent with poor BCS and two clinically blind; all three were euthanized directly after clinical and ultrasonographic assessment. The signalment, history, and necropsy information (if relevant) for the 17 animals are given in Supporting Information S1, the M-mode and Doppler values for the clinically normal animals in Tables 1 and 2, respectively, and for the sick animals in Tables 3 and 4, respectively.

Animals deemed clinically normal had no clinical signs (other than two with mild metatarsophalangeal synovial effusion). They had a normal appetite and no obvious heart murmurs. Of these 14 EGKs, ten were juveniles/young adults and four were adults. Two

TABLE 3 | Pulsed wave Doppler echocardiographic findings of fourteen clinically normal eastern grey kangaroos (*Macropus giganteus*); velocity in cm/s for mitral, tricuspid, pulmonary, and aortic valves

Number	Signalment	Weight, kg	PA	PVR	PV PPG	MV E peak	MV A peak	MVR	TV E peak	TV A peak	AoV	Ao PPG
K15	Juvenile M	8.3	69	—	1.9	67	33	—	30	13	98	—
K25	Young adult F	11.5	—	—	—	63.5	42.1	—	43.9	31.1	—	—
K29	Young adult M	11.6	110.8	—	—	74	43	—	50	39	141	—
K26	Young adult M	12.8	103.2	—	—	56.8	41.2	—	47.8	64.8	85.7	—
K27	Young adult M	13.5	128.2	—	—	66.6	33.8	—	—	—	88.1	—
K13	Young adult F	14	60	—	0.6	49.5	34.3	—	56.3	35.5	105	—
K28	Young adult M	14.5	92.2	—	—	64.7	37.7	—	—	—	93.9	—
K24	Young adult M	14.7	105	—	4.4	38	50	53	—	—	72	2.09
K6	Young adult F	16	51	—	—	41.7	27	—	42.9	23.3	60.2	—
K14	Young adult	16	130	—	—	48	38	—	—	—	110	—
K5	Adult	22	100.1	84	—	47	38	—	—	—	76.2	—
K7	Adult	22	84.3	—	—	61.5	35.5	—	58.7	46.6	—	—
K21	Adult	22	100.1	—	2.8	47	38	—	—	—	113	5.13
Z3	Adult	57	100.5	105.5	—	72.5	45	—	58	35	105	—
	Average	18.3	95	94.8	2.4	57	38.3	53	48.5	36	95.7	3.6
	SD	11.5	22.8	10.8	1.4	11.3	5.5	0	9	14.4	20.6	1.5

Abbreviations: AoV, aortic valve; MV, mitral valve; MVR, mitral valve regurgitation; PA, pulmonary artery; PPG, peak pressure gradient (mmHg); PVR, pulmonary valve regurgitation; TV, tricuspid valve.

clinically unwell EGKs were evaluated (one young adult and one adult), and one sick RK adult.

The weight of the clinically normal single juvenile (estimated under 10 months) was 8.3 kg, the median weight of the nine normal young adults (approximately 10–20 months) was 14 kg, and that of the four normal adults (over approximately 20 months) was 39.6 kg (Table 1). The weight of the single sick young adult was 8.5 kg, and the two sick adults weighed 26.5 kg each (one EGK and one RK). The sick kangaroos (regardless of age) had poorer body condition scores (BCS: 2–3/5), while the healthy kangaroos had BCS of 3–4/5. The ages of all the animals ranged from 8.5 months to 8–10 years, and were estimated using specific body measurements from a published reference [22]. Images were obtained with the kangaroo in right and left lateral recumbency. For left apical view images, the transducer was generally placed on top of the left thorax. For right parasternal view images, images were obtained both using the standard method (transducer on dependent side), with the patient's shoulders elevated with a towel, and with the transducer on the right thorax when the patient was in left lateral recumbency. The lateral recumbent position (both right and left) was particularly useful for the subcostal apical view, resulting in excellent quality images.

M-mode images and La:Ao were the most challenging to obtain, with subtle transducer movements resulting in optimal images (i.e., obtaining a clear M-mode without papillary muscle coming into view, and an optimal view of the atrium). Placing the transducer on the nondependent side of the thorax was the preferred method, and good images were obtained, likely the optimal method in the field, precluding the necessity of a table with a cut-out and ease of pouring alcohol on the nondependent part of the thorax.

Measurements were taken from leading edge to leading edge. If possible, three measurements were made and averaged at the most representative image of the cardiac cycles Table 1.

No B-mode echocardiographic abnormalities were found in thirteen of the fourteen clinically normal animals (Table 1), except that all had RWTs of 0.45 to 0.64 (weight-matched values: 0.46 to 0.71[6]) (Table 1). K5 and Z3 both showed mild incidental pulmonary valve insufficiency on Doppler evaluation, with no secondary echocardiographic changes. K24 showed a mild mitral valve regurgitation.

The sick EGK young adult female (K16, 8.5 kg, BCS 2/5) had been presented in recumbency and hypothermic (34.3°C); clinical pathology was suggestive of exertional (capture) rhabdomyolysis (see Supporting Information S1). Echocardiographically, she was seen to have what appeared to be a markedly thickened left ventricular free wall in comparison with a reported 11 kg normal kangaroo [6], associated with left ventricular chamber diminution, (Figure 1) a RWT of 1.02, with an averaged fractional shortening of 0.5 (Table 2); the endocardium appeared more hyperechoic than normal. See Figure 2 for a comparative normal short-axis view (K21). There was mild mitral valve regurgitation. She was euthanized with an overdose of barbiturates (Lethabarb Euthanasia injection (325 mg/mL pentobarbitone) IV; Virbac Australia Pty Ltd, Milpera, NSW, Australia). Necropsy revealed a markedly thickened left ventricular and interventricular

TABLE 4 | Pulsed wave Doppler echocardiographic findings of two sick eastern grey kangaroos (*Macropus giganteus*) (K4, K16) and one red kangaroo (*Osphranter rufus*) (Z2); velocity in cm/s for mitral, tricuspid, pulmonary, and aortic valves.

Number	Signalment	Weight, kg	PA	PVR	PV PPG mmHg	MV E peak	MV A peak	MVR	TV E peak	TV A peak	TVR	Ao	AoVR	Ao PPG mmHg
K16 – suspect HCM	Young adult	8.5	69	—	18.8	39.3	13.2	52	29	23	—	73	—	2.1
Z2 – suspect DCM	Adult	26.5	100.1	Mild	31.6	70.2	46.5	—	—	—	47	82.4	—	2.7
K4 – suspect volume overload, ERM	Adult	26.5	92	—	33.9	60.9	27.9	428	—	—	—	90.5	mild	—

Abbreviations: Ao, aorta; AoVR, aortic valve regurgitation; DCM, dilated cardiomyopathy; ERM, exertional rhabdomyolysis; HCM, hypertrophic cardiomyopathy; MV, mitral valve; MVR, mitral valve regurgitation; PA, pulmonary artery; PPG, peak pressure gradient; PVR, pulmonary valve regurgitation; TV, tricuspid valve.

septal thickening, with myocardial fibrosis (Figure 1; Supporting Information S1).

The sick captive RK adult female (Z2, 26.5 kg, BCS 2.5–3/5) was submitted due to weight loss and exercise intolerance. (Supporting Information S1). The RK was included, although a different species, since her size and weight were similar to those of another sick adult (K4).

Echocardiographically, the interventricular septum and left ventricular free wall thicknesses were lower or on the low side of normal to the closest weight-compared normal kangaroo value (23 kg), with a relatively low fractional shortening (0.23). The left ventricular internal diameter of this 26.5 kg animal was wider or on the very high side of normal in diastole and systole (44.9 and 34.8 mm, respectively) than the closest weight-matched and lighter kangaroo (23 kg) values (36 and 24 mm, respectively); the RWT was 0.38 (Table 2). There was mild mitral valve regurgitation (Figure 3) and mild, insignificant, pulmonary valve regurgitation. See the Doppler velocities in Table 4 [23]. A presumptive diagnosis of dilated cardiomyopathy and myxomatous mitral valve degeneration was made. She was euthanized with an overdose of barbiturates. Gross postmortem examination revealed endocardiosis leading to mitral insufficiency. Histopathology was not performed due to a technical fault. See Supporting Information S1 for more necropsy information.

The sick EGK adult female (K4, 26.5 kg) was recumbent and in an extremely poor BCS of 2/5 (Supporting Information S1). The internal diameter of the left ventricle in diastole and systole was 43.5 and 26.5 mm, respectively. These values were larger than the closest weight-matched kangaroo (23 kg; systole: 24 mm, diastole: 36 mm); the RWT was 0.47. There was mild mitral valve regurgitation and subjective left atrial enlargement, and mild, insignificant pulmonary valve regurgitation. The Doppler velocities are given in Table 4. A presumptive diagnosis of myxomatous mitral valve degeneration and volume overload due to chronic anemia was made, with mild mitral and insignificant, mild aortic valve insufficiencies (Figure 4). She was euthanized with an overdose of barbiturates. Postmortem examination revealed exertional myocardial rhabdomyolysis and nodular myxomatous calcified endocardiosis. See Supporting Information S1 for more necropsy information.

4 | Discussion

This study aimed to document the echocardiographic findings in an opportunistic group of kangaroos of various ages, weights, and wellness, to compare the findings with published kangaroo values, and to report on any pathology present.

The gross anatomy of the western grey kangaroo (*M. fuliginosus*) heart has been reported, with some variations from the better-known human and dog heart anatomy [19]. It has also been reported that the heart of Macropods could be up to a third larger than that of size-matched eutherian mammals [24–27]. Determining radiological vertebral heart scores in the macropod may shed more light on comparative heart sizes of eutherian mammals.

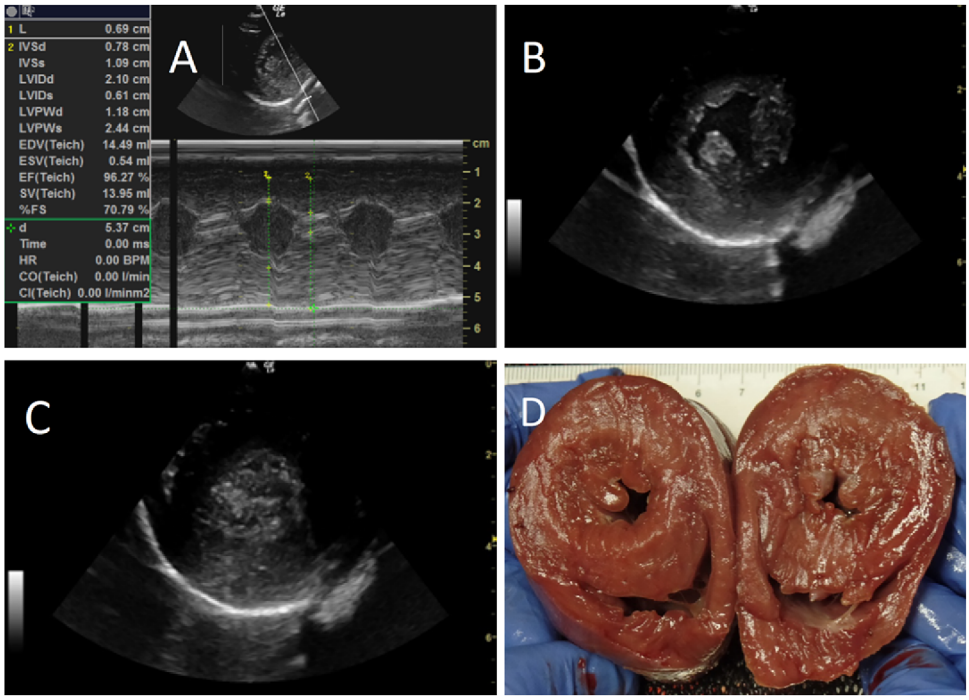

FIGURE 1 | Right parasternal short-axis view at the level of papillary muscles of a young adult female EGK (K16) M-mode (A) and B-mode, just below the mitral valve in diastole (B) and systole (C), illustrating marked left ventricular free wall and interventricular septal thickening, and decreased left ventricular chamber size. Transverse section through the heart illustrates the marked myocardial thickening (D) EGK, eastern grey kangaroo.

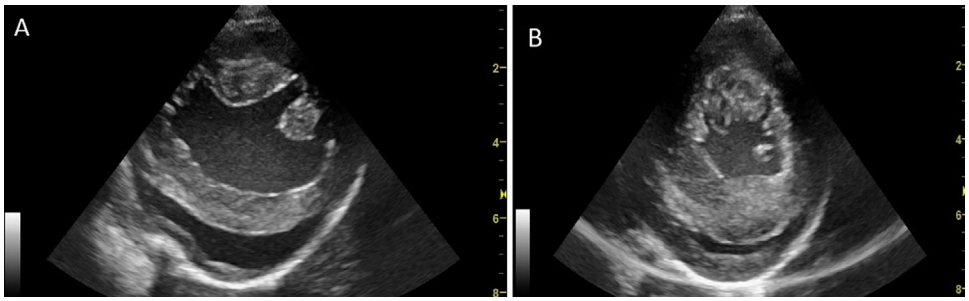

FIGURE 2 | Right parasternal short axis view at the level of papillary muscles of a normal young adult female eastern grey kangaroo (K21) B-mode, just below the mitral valve in diastole (A) and systole (B).

In a published kangaroo echocardiographic project [6], all seven sedated apparently healthy adult rock kangaroos (*Osphranter robustus*), were considered to have normal clinical parameters, but had RWTs of between 0.46 and 0.71 (the report's values were reciprocal to recent reporting formats, the values being a mean of 0.58 (reciprocal: 1.7), with a range of 0.53 to 0.71 (reciprocal: 1.9–1.4)). In the current study, our values were similar, ranging between 0.53 and 0.74. Sugishita et al. [6] hypothesized that this apparent hypertrophy may be normal for macropods, due to strenuous exercise such as jumping, or in fact due to hypertrophy associated with HCM. Human HCM diagnosis leans heavily on the clinical and echocardiographic assessment, both for obstructive and nonobstructive HCM [28], where HCM historically is considered to be present with an RWT greater than 0.45. Definitive conclusive evidence of HCM requires a full clinical and echocardiographic evaluation. Also, if simultaneous volume and pressure overload is present, left ventricular hypertrophy can occur without significant changes in RWT [7]. It may be that kangaroos generally have a thicker myocardium, or it may be an indication of the presence of subclinical HCM, which warrants further investigation. Two of the sick kangaroos (Z2 and K4, both with mild MVI and one with suspected DCM, and the other volume overload likely due to chronic anemia) had an RWT of 0.38 and 0.47, respectively; the suspect HCM kangaroo (K16) showed a RWT of 1.02—the latter possibly indicative of the condition. This juvenile EGK (K16) had generalized left ventricular concentric hypertrophy (Figure 1) consistent with hypertrophic cardiomyopathy, with no histopathological evidence of necrosis or fibrosis. She was an orphan in rehabilitation care, so it was unlikely that her myocardial thickening was as result of Takotsubo-like cardiomyopathy associated with capture myopathy as reported in a Red-necked Wallaby (*M. rufogriseus)* [29]; other differentials include transient myocardial thickening or myocarditis, pseudohypertrophy [30] due to volume depletion/dehydration, systemic hypertension, outflow tract obstructions, or neoplasia. Transient ventricular hypertrophy has been reported in cats and should also

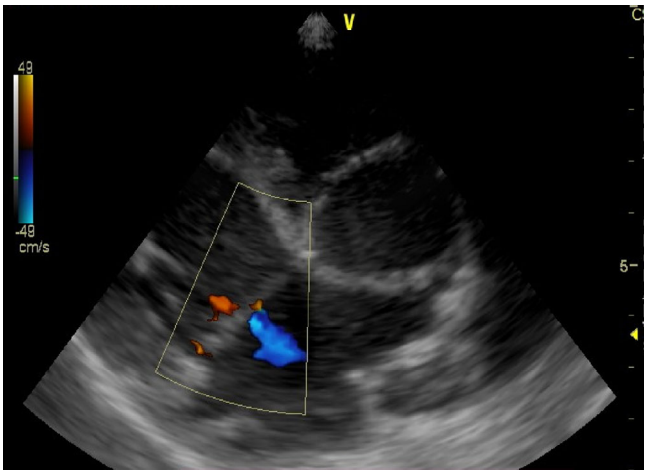

FIGURE 3 | Apically angled right parasternal long axis view color flow Doppler of the female red kangaroo (Z2, suspect dilated cardiomyopathy) with very mild mitral valve insufficiency.

be considered as a differential diagnosis [31]. It is unknown if cardiomyopathies could be due to increased exertion, particularly if an underlying cause such as HCM, dilated cardiomyopathy, or nutritional myocardial degeneration is present. Myocardial thickening and intracardiac thrombi have also been reported in cats with wildfire thermal injuries [32]. Bushfires are common and a severe problem in Australia; determining the presence or absence of this condition in macropods requires further investigation. All these conditions need further investigation, particularly in relation to stress induced by capture, handling, and captivity [5, 33].

Transducer placement for right parasternal views gave good images with the patient in right lateral recumbency, with the cranial thorax slightly elevated with towels. For right parasternal views, left lateral recumbency also gave excellent images with the transducer placed on top; this may actually be the preferred method since alcohol is more easily placed on the nonrecumbent side and a cut-out table top is unlikely to be available in the field. This left lateral recumbency was also utilized by Sugishita et al. [6]. A few different positions to find optimal transducer placement for Doppler evaluation were tried, particularly when scanning was done outside in nonoptimal conditions. This may explain the variation in Doppler findings, although some variation has also been reported in the lowest Doppler reference ranges in dogs [34]. The kangaroo Doppler flow values were generally less than weight-related published dog values [23, 35, 36] and those of humans in general (not weight-related) [37], although making these comparisons is likely not valid or useful.

Little has been published on other marsupial echocardiology: a single nonpublished comment on echocardiographic suspect left ventricular hypertrophy in wombats [6], and mild aortic insufficiency and ectasia in a ring-tailed possum (*Pseudocheirus peregrinus*) [38]. The wombat echocardiography authors comment that this slow-moving marsupial's apparent cardiac hypertrophy may indicate that strenuous activity is likely not the cause of this anomaly. Further research in marsupial cardiology is indicated.

From what we have learned from this work, we suggest that a clinical and echocardiographic re-evaluation be performed within 2–3 months if the animal shows a clinically significant cardiac problem or if the animal is seen to deteriorate clinically, where therapy or humane euthanasia may be instigated. The caveat here is that most kangaroos are wild, and it is unlikely that they will be re-evaluated unless they are in a zoo or rehabilitation setting.

The most important limitation of this study was the relatively low number of subjects evaluated and the large variation in age, size, and weight, precluding statistical analyses. Since this study was opportunistic and only these few animals were presented within the study time, this limitation was expected.

The lack of ECG coupling during the examination is also a limitation, but it was not available for this study. More advanced future echocardiographic studies would require an ECG. The absence of precisely weight-matched comparative mensuration values is also a limitation, but unavoidable due to no other values having been reported.

Performing echocardiography in sedated patients, as was required in this study, is another important limitation. Minimal work has been published on the effect of chemical sedation of kangaroos. Sedatives may result in cardiovascular changes, such as decreased cardiac output and blood pressure. Reports of 26 western grey kangaroos (*M. fuliginosus ocydromus*) sedated with intramuscular tiletamine-zolazepam [39], and two kangaroos sedated with acepromazine and xylazine, respectively [40], have been published, but no cardiovascular parameters were reported. No echocardiographic parameters showed significant changes in dogs subjected to different induction protocols, including diazepam; however, a diazepam and ketamine combination resulted in decreased blood pressure [41]. Diazepam did not alter heart rate or mean arterial pressure in dogs but did increase cardiac output at higher doses (2.5 mg/kg) [42]. The necessity for using sedation is self-evident when working with wild or semi-tame wild animals, and it may have affected the echocardiographic parameters obtained in this study.

Since these animals are unlikely to be evaluated in the wild, it is important to observe captive animals for abnormalities such as lethargy and inappetence, but since it has been described that animals have been found to have died spontaneously with no preceding clinical signs [5, 6], it could be challenging. Captive animals and those destined for release postrehabilitation are usually seen daily and evaluated clinically on a regular basis; this would be an optimal time for specific cardiac evaluation, such as auscultation, ECG, echocardiography, cardiac troponins, and other biochemical parameters. Prospective further research could be done at this time, utilizing this information. Although limited in number, case reports have described the response to therapy, that is, a single Matschie's tree kangaroo with HCM from age 3–11.5 years [8]. It is also important to note that one is aware of possible anesthetic risks/deaths in kangaroos with cardiomyopathies [6].

Finally, knowledge of kangaroo echocardiography could be of benefit to man, as components of the kangaroo heart could potentially replace traditional pig or cow heart valves in human

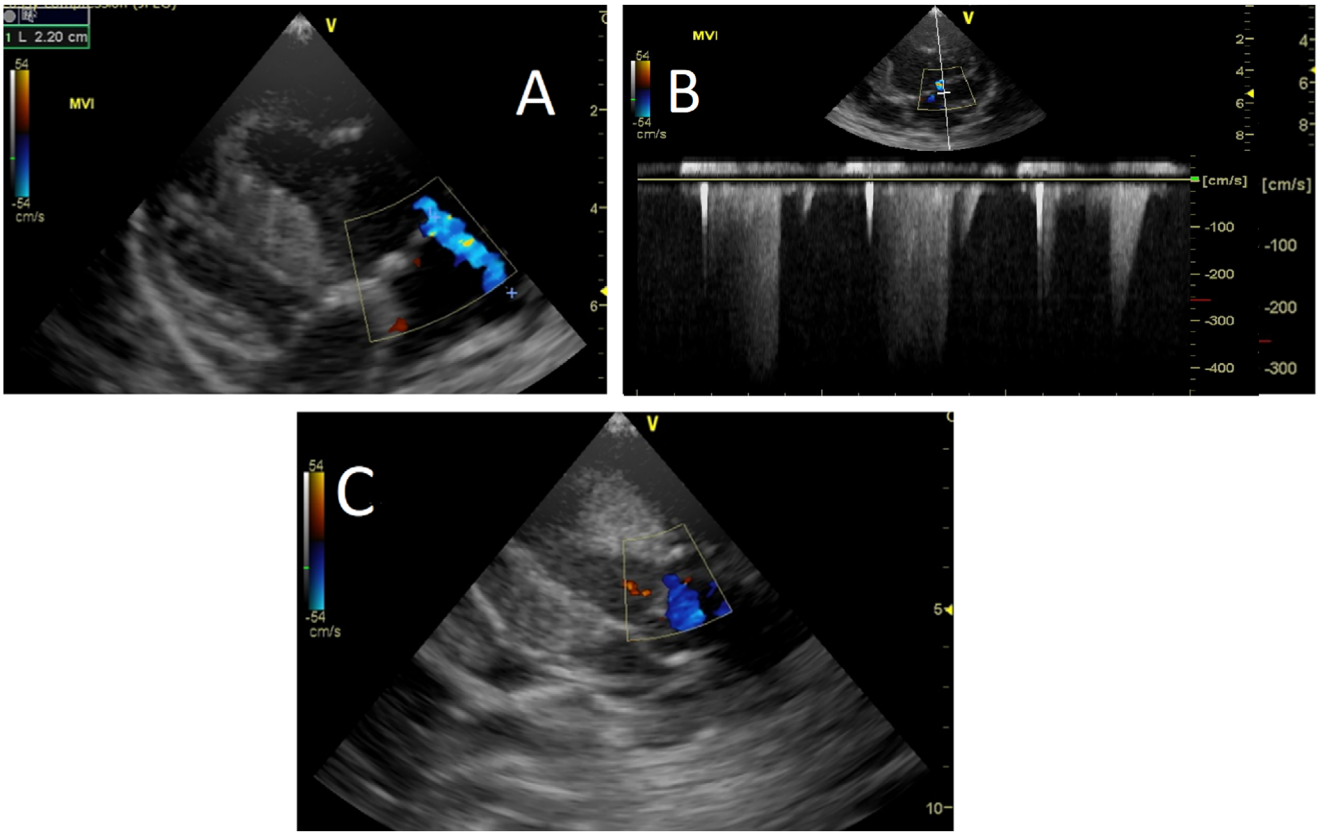

FIGURE 4 | Left parasternal apical four-chamber view color flow Doppler of a female EGK kangaroo (K4, suspect volume overload due to chronic anemia) with mild mitral valve insufficiency (A), pulsed wave Doppler image illustrating the turbulent flow (B), and mild aortic regurgitation in a right parasternal long axis view (C). EGK, eastern grey kangaroo.

heart valve replacement [18, 19]. Bioprosthetic valves made from the kangaroo pericardium have a similar durability and function to pig valves, but are still in early stages of research and development [19]. The upright stance of kangaroos, being similar to that of humans, may be an additional advantage. The sustainability and ethical implications of using kangaroo valves in human medicine, however, are a concern. More work is also required to determine whether kangaroos can act as experimental models for studying the mechanisms of HCM in humans [6].

In conclusion, the M-mode values in clinically normal kangaroos are similar to weight-matched previously reported kangaroo values, providing presumptive references for echocardiography in the kangaroo. Relative wall thickness evaluation may also be of use. Possible hypertrophic and dilated cardiomyopathy were concerning conditions found in two of the three sick kangaroos scanned, and it is questioned whether the normal animals may have had subclinical HCM or if the macropod ventricle is normally relatively thick. More work is required to determine normal values in different kangaroo species, age and weight categories, the prevalence and cause of cardiac disease in the macropod population, and whether certain macropod cardiac parameters can be utilized in the study of some cardiac conditions in humans.

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(b) Acquisition of data: Carstens, Birckhead

(c) Analysis and interpretation of data: Carstens, Birckhead

Category 2

(a) Drafting the article: Carstens

(b) Revising article for intellectual content: Carstens, Connolly, Dutton, Stannard, Barkman, Birckhead

Category 3

(a) Final approval of the completed article: Carstens, Connolly, Dutton, Stannard, Barkman, Birckhead

Category 4

(a) Agreement to be accountable for all aspects of the work, ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved: Carstens, Connolly, Dutton, Stannard, Barkman, Birckhead

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Conflicts of Interest

The authors declare no conflicts of interest.

Previous Presentation or Publication Disclosure

Poster presentation: A.Carstens, G.Dutton, H.Stannard, A.Birckhead, W.Barkman, J.Connolly. Electrocardiographic examination of 16 eastern grey kangaroos (*Macropus giganteus*) and a red kangaroo (*Osphranter rufus*). The 73rd Annual International Conference of the Wildlife Disease Association in Victoria, British Columbia, Canada, July 27-August 1, 2025.

Reporting Checklist Disclosure

No EQUATOR network checklist or other reporting checklist was used.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Supplement 1: Signalment and information on the 16 eastern grey kangaroos (*Macropus giganteus*) and the one red kangaroo (*Osphranter rufus*) scanned