Cardiomiopatía fenotipo dilatado

Dr. Alberto R. Meder
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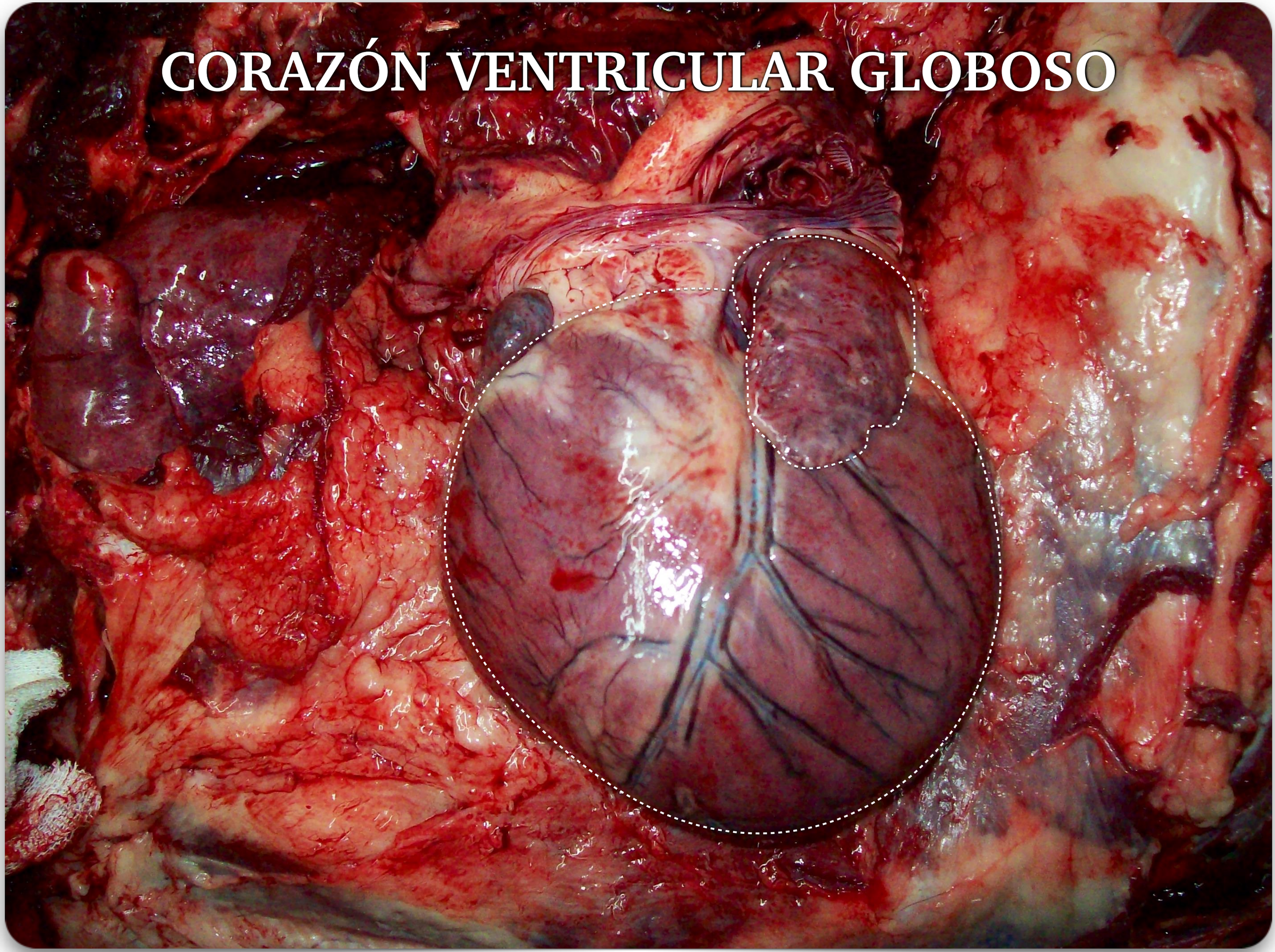
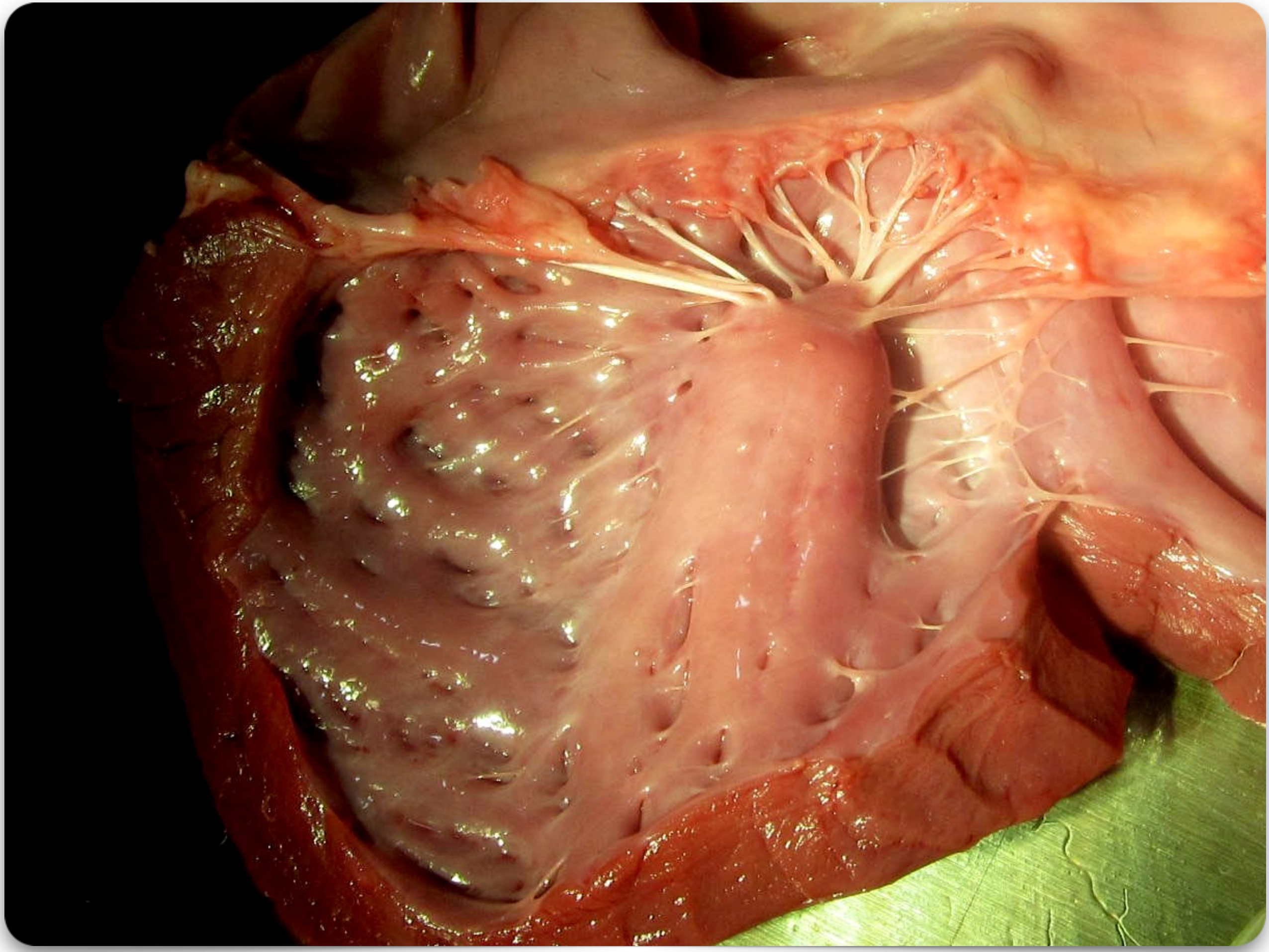
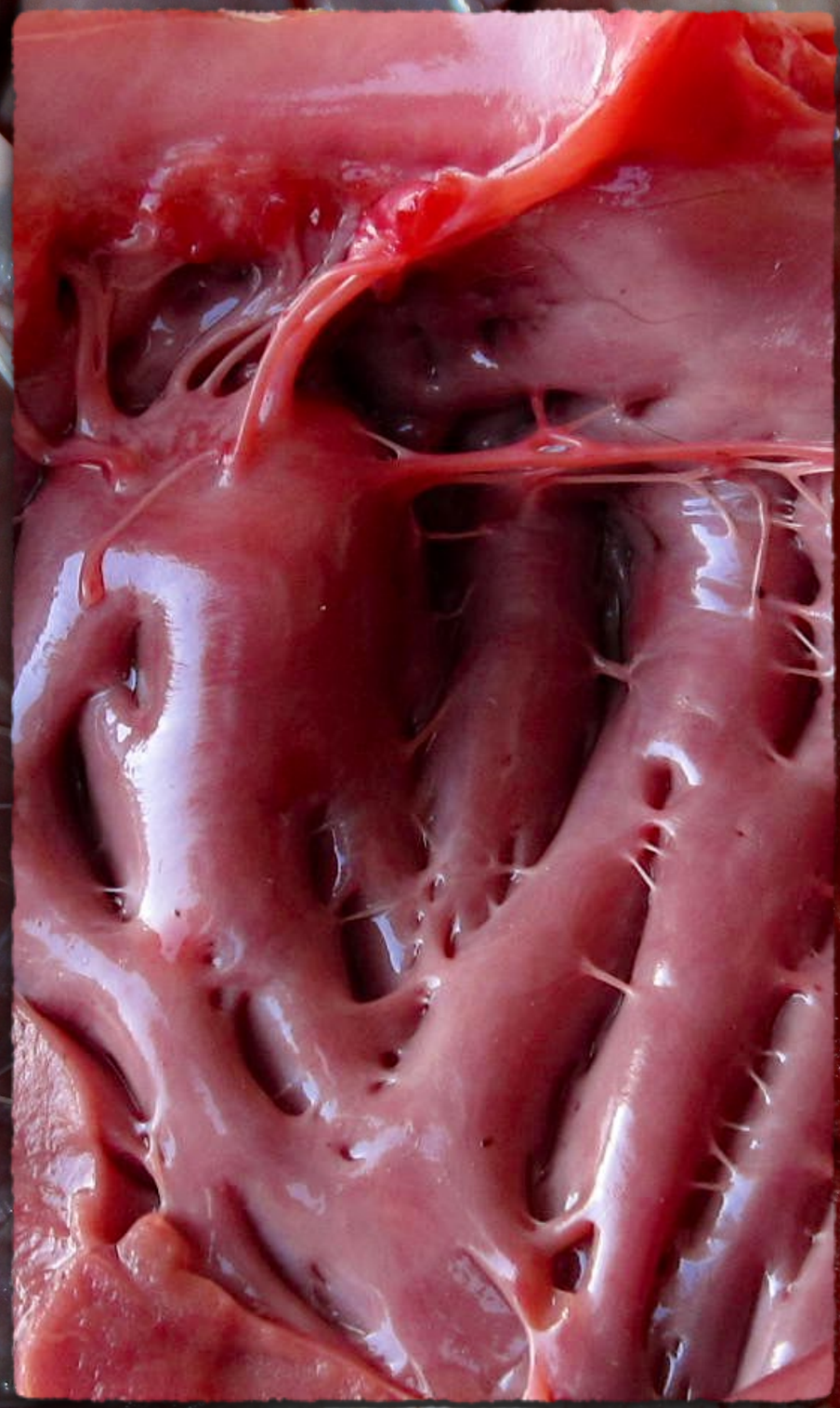
FOR.VET | ESPACIO DE FORMACION INTEGRAL VETERINARIA

CORAZÓN VENTRICULAR GLOBOSO





VENTRÍCULO IZQUIERDO DILATADO



APLANAMIENTO PARED INTERNA Y PAPILARES

CARDIOMEGALIA ATRIAL IZQUIERDA

PAREDES DELGADAS-LISAS-NO CONTRACTILES

PUNTO CLAVE DE LA PATOLOGÍA

- Es una enfermedad primaria del músculo cardíaco
- Secundaria a las siguientes causas:
  - Tóxica (Doxorrubricina)
  - Nutricional (Carnitina/Taurina)
  - Endocrina (Hipotiroidismo)
  - Infecciosa (Tripanosomiasis)

ETIOLOGÍA

Los mocitos ventriculares izquierdos (derechos) van sufriendo un proceso de muerte celular (GENES), que genera el reemplazo por tejido conectivo o adiposo, los cuales disminuyen la contractilidad miocárdica de forma progresiva e irreversible, generando deficiencia sistólica ventricular primaria

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RESEÑA

Caninos - Raza pura (SRD), adultos/gerontes, más en machos (60/40)

- Wess G, Schulze A, Butz V, et al. Prevalence of dilated cardiomyopathy in Doberman Pinschers in various age groups. J Vet Intern Med 2010;24:533–538
- O'Grady MR, O'Sullivan ML. Dilated cardiomyopathy: an update. Vet Clin North Am Small Anim Pract 2004;34: 1187–1207
- Calvert CA, Jacobs G, Pickus CW, et al. Results of ambulatory electrocardiography in overtly healthy Doberman Pinschers with echocardiographic abnormalities. J Am Vet Med Assoc 2000;217:1328–1332.

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J Vet Intern Med 2009;23:1014–1019

Decreased Triadin and Increased Calstabin2 Expression in Great Danes with Dilated Cardiomyopathy

M.A. Oyama, S.V. Chittur, and C.A. Reynolds

Background: Dilated cardiomyopathy (DCM) is a common cardiac disease of Great Dane dogs, yet very little is known about the underlying molecular abnormalities that contribute to disease.

Objective: Discover a set of genes that are differentially expressed in Great Dane dogs with DCM as a way to identify candidate genes for further study as well as to better understand the molecular abnormalities that underlie the disease.

Animals: Three Great Dane dogs with end-stage DCM and 3 large breed control dogs.

Methods: Prospective study. Transcriptional activity of 42,869 canine DNA sequences was determined with a canine-specific oligonucleotide microarray. Genome expression patterns of left ventricular tissue samples from affected Great Dane dogs were evaluated by measuring the relative amount of complementary RNA hybridization to the microarray probes and comparing it with expression from large breed dogs with noncardiac disease.

Results: Three hundred and twenty-three transcripts were differentially expressed (≥2-fold change). The transcript with the greatest degree of upregulation (+61.3-fold) was calstabin2 (FKBP12.6), whereas the transcript with the greatest degree of downregulation (−9.07-fold) was triadin. Calstabin2 and triadin are both regulatory components of the cardiac ryanodine receptor (RyR2) and are critical to normal intracellular Ca^{2+} release and excitation-contraction coupling.

Conclusion and clinical importance: Great Dane dogs with DCM demonstrate abnormal calstabin2 and triadin expression. These changes likely affect Ca^{2+} flux within cardiac cells and may contribute to the pathophysiology of disease. Microarray-based analysis identifies calstabin2, triadin, and RyR2 function as targets of future study.

Key words: Canine; Microarray; Ryanodine.

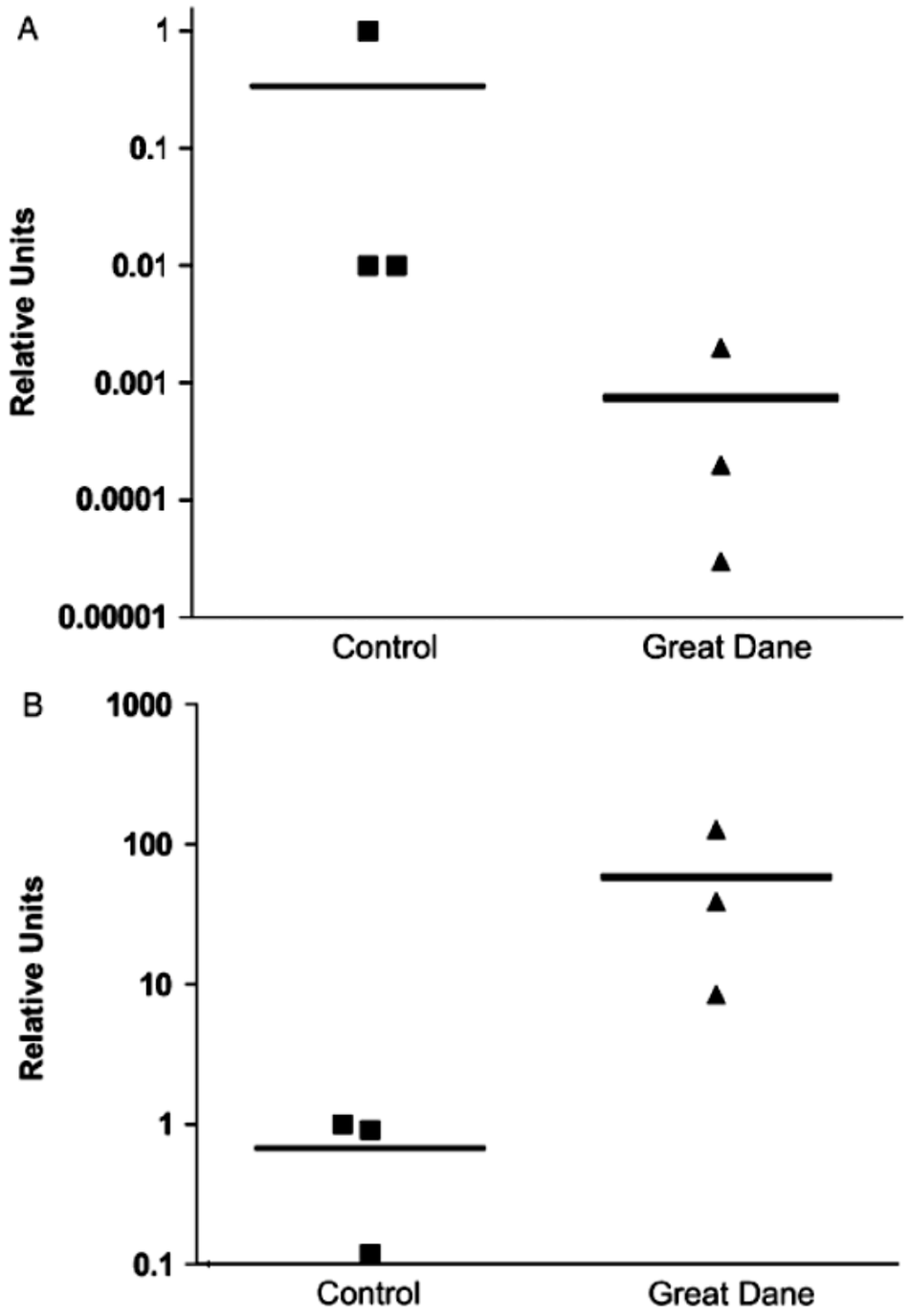

Fig 1. Relative expression of (**A**) triadin and (**B**) calstabin2 transcripts determined by a real-time reverse transcriptase-quantitative polymerase chain reaction assay in 3 Great Danes with end-stage dilated cardiomyopathy and 3 controls. Note the logarithmic scale used for the *Y*-axis. Bar represents mean value.

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Research Article

Multiple Genetic Associations with Irish Wolfhound Dilated Cardiomyopathy

Siobhan Simpson,[1] Mark D. Dunning,[1] Serena Brownlie,[1] Janika Patel,[1] Megan Godden,[1] Malcolm Cobb,[1] Nigel P. Mongan,[1,2] and Catrin S. Rutland[1]

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[2]*Department of Pharmacology, Weill Cornell Medical College, 1300 York Avenue, New York, NY 10065, USA*

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Table 7: Risk of developing DCM/AF for each genotype at loci identified as significantly associated with DCM/AF.

Genotype	Risk	Total genotype	Risk relative to population risk	95% CI
Chr1 CC	0.81	54	1.08	0.91 to 1.28
Chr1 CT	0.68	22	na	na
Chr1 TT	0.5	4	na	na
Chr1 CT/TT	0.65	26	0.86	0.64 to 1.17
Chr21 AA	0.61	18	0.81	0.55 to 1.19
Chr21 GA	0.77	39	na	na
Chr21 GG	0.82	33	na	na
Chr21 GA/GG	0.79	72	1.04	0.89 to 1.23
Chr37 AA	0.85	27	1.12	0.93 to 1.36
Chr37 GA	0.70	23	na	na
Chr37 GG	0.73	44	na	na
Chr37 GA/GG	0.72	67	0.95	0.78 to 1.14

The risk of developing disease for each genotype, the total number of individuals with each genotype, the relative risk of each genotype compared to the population risk, and associated 95% confidence intervals. Where dominant or recessive modes of penetrance were established or less than 5 total individuals had a genotype, genotypes were pooled and these are also presented. Chr1 refers to the SNP on chromosome 1, Chr21 the SNP on chromosome 21, and Chr37 the SNP on chromosome 37 as identified in Philipp et al. [23] and Table 1.

El riesgo de desarrollar CMD / FA se asocia a la combinación de múltiples genes

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DOI 10.1007/s00439-012-1158-2

ORIGINAL INVESTIGATION

A splice site mutation in a gene encoding for PDK4, a mitochondrial protein, is associated with the development of dilated cardiomyopathy in the Doberman pinscher

Kathryn M. Meurs · Sunshine Lahmers · Bruce W. Keene · Stephen N. White · Mark A. Oyama · Evan Mauceli · Kerstin Lindblad-Toh

Received: 4 August 2011 / Accepted: 8 March 2012 / Published online: 25 March 2012

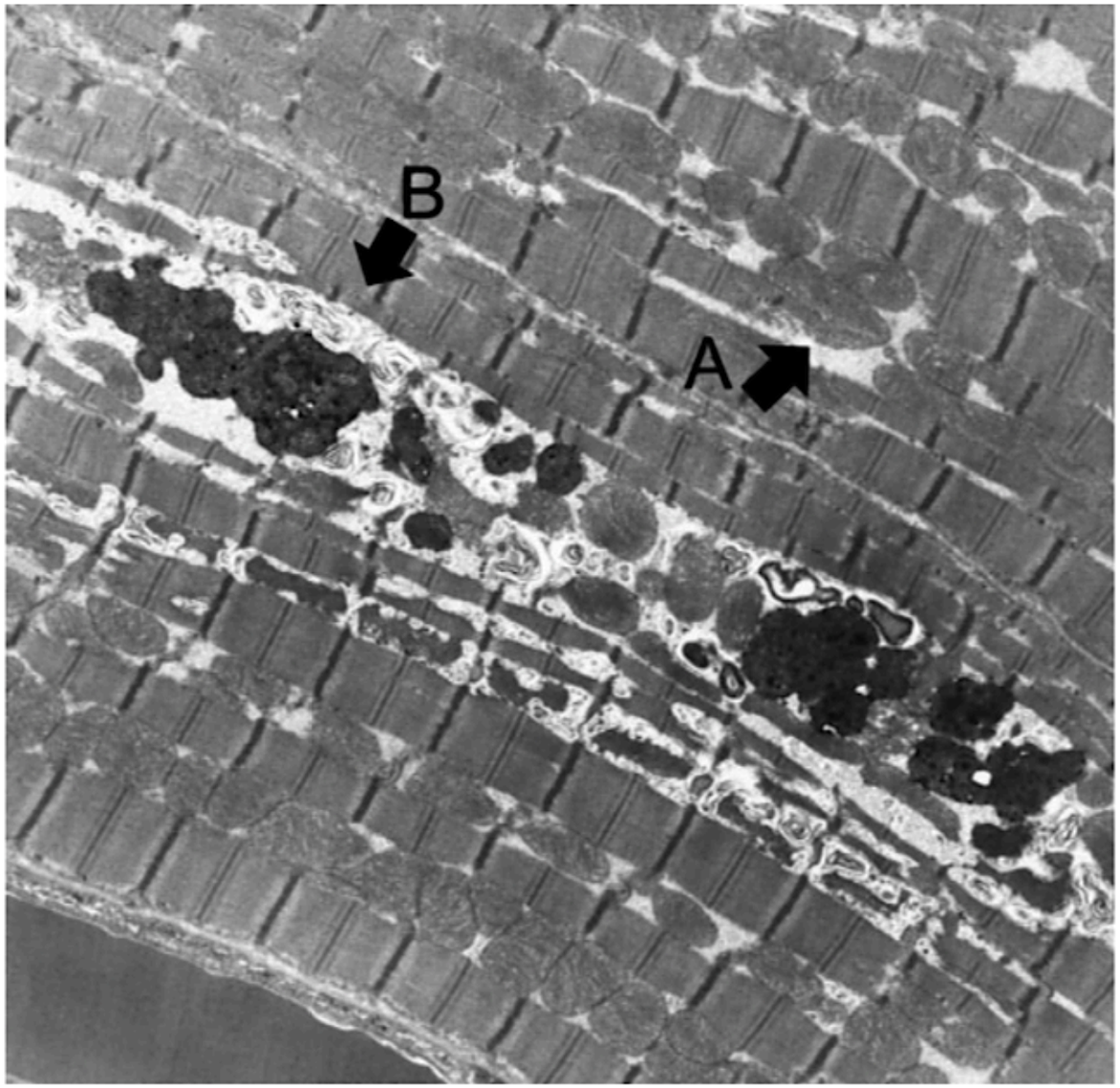

Fig. 4 Electron microscopy of myocardium from affected dogs demonstrated disorganization of the Z line, mild to moderate T tubule and sarcoplasmic reticulum dilation, marked pleomorphic mitochondrial alterations with megamitochondria (*A*), scattered mitochondria with whorling (*B*) and vacuolization disruption and mild aggregates of lipofuscin granules

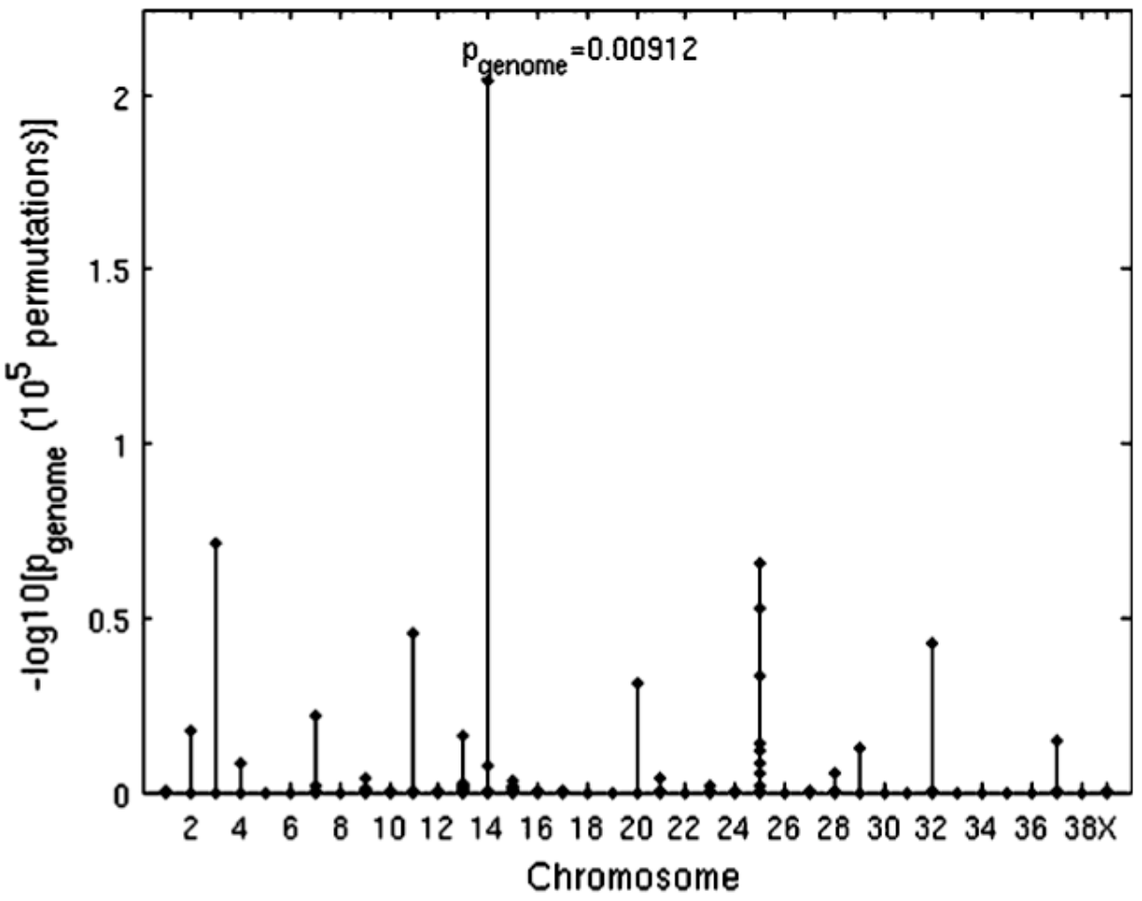

Fig. 1 Genome-wide association mapping of canine dilated cardiomyopathy. Genome-wide association analysis identified an area of statistical significance on canine chromosome 14 ($p_{raw} = 9.999e{-}05$ corrected for genome-wide significance, $p_{genome} < 0.009$ based on 100,000 permutations)

Área de significación estadística en el cromosoma 14 canino. La secuenciación del ADN identificó una deleción de 16 pares de bases en el sitio de empalme de 50 donantes del intrón 10 del gen de la piruvato deshidrogenasa quinasa 4 en perros afectados (p>0,0001).

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FASE ASINTOMÁTICA OCULTA
(variable / años)
(empezamos el chequeo a los 3 años)

&

FASE CLÍNICA ESTABLECIDA
(< 15% más de 1 año)

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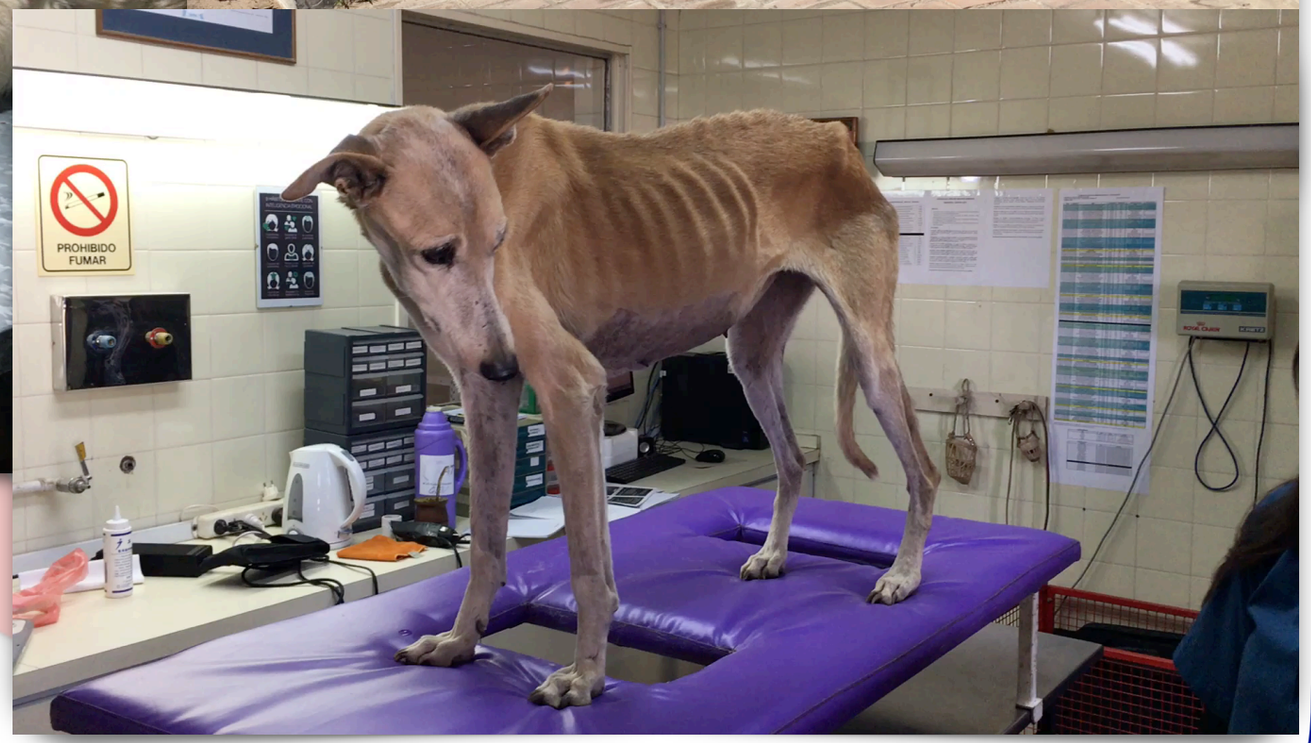
ANAMNESIS

En general los propietarios no se dan cuenta del proceso patológico y recurren a la consulta con dudas sobre:

- Distensión abdominal por derrame
- Debilidad / letargia / decaimiento



- Pérdida de peso / caquexia
- Síncopes
- Intolerancia al ejercicio

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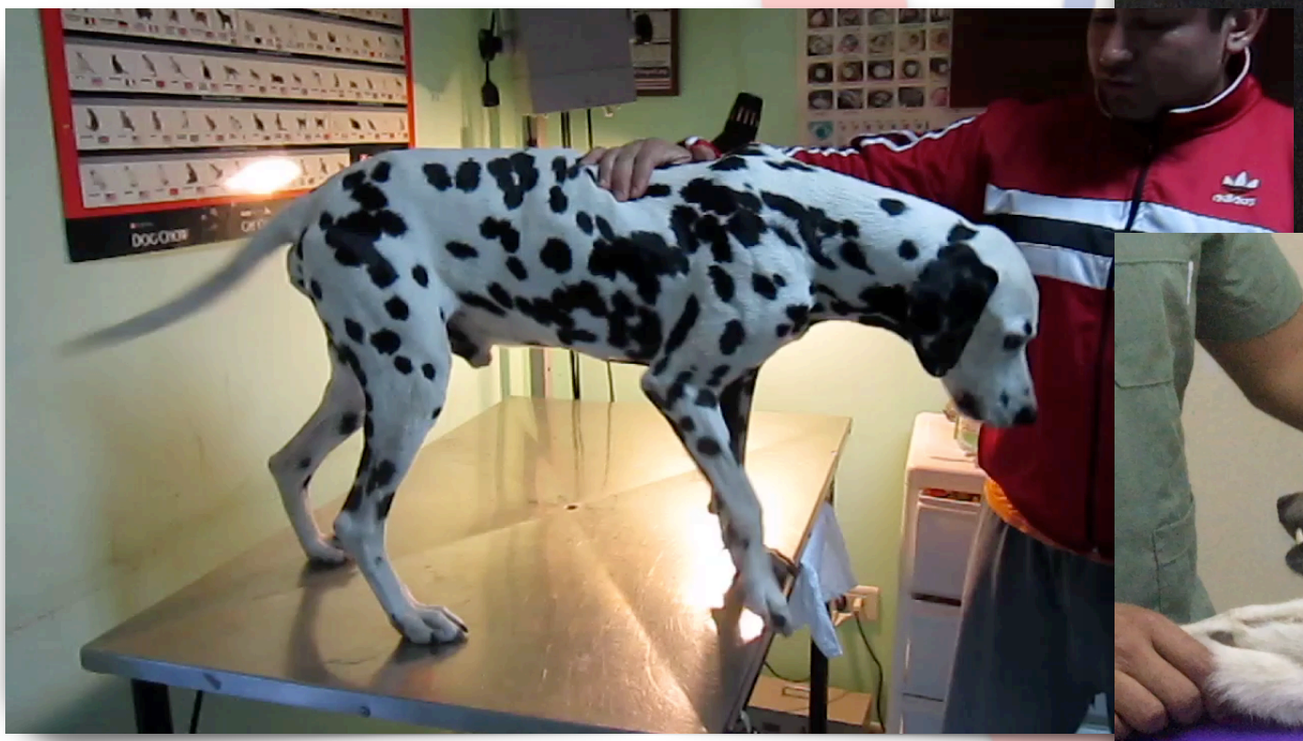
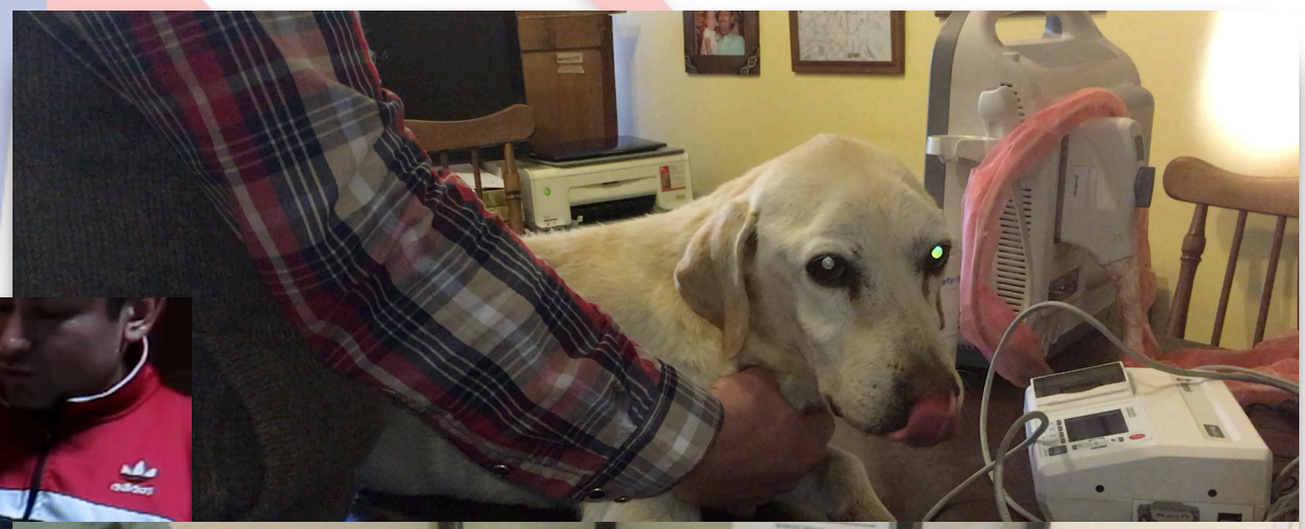
EXAMEN FÍSICO

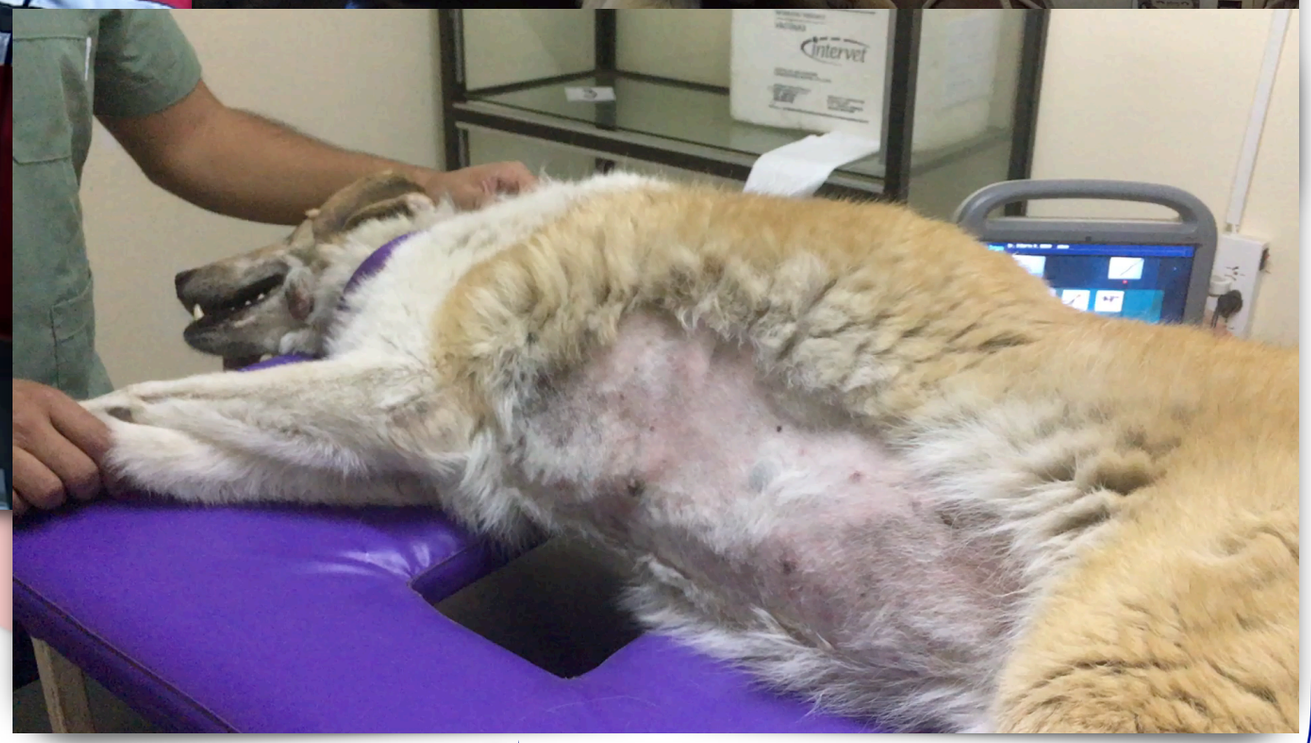
- Los signos clínicos pueden ser causa de consulta o hallazgo del examen físico cuando el paciente es explorado:

  - Tos suave / húmeda / débil
  - Disnea mixta / taquipneica





- Disnea inspiratoria restrictiva por colecta pleural cardiogénica



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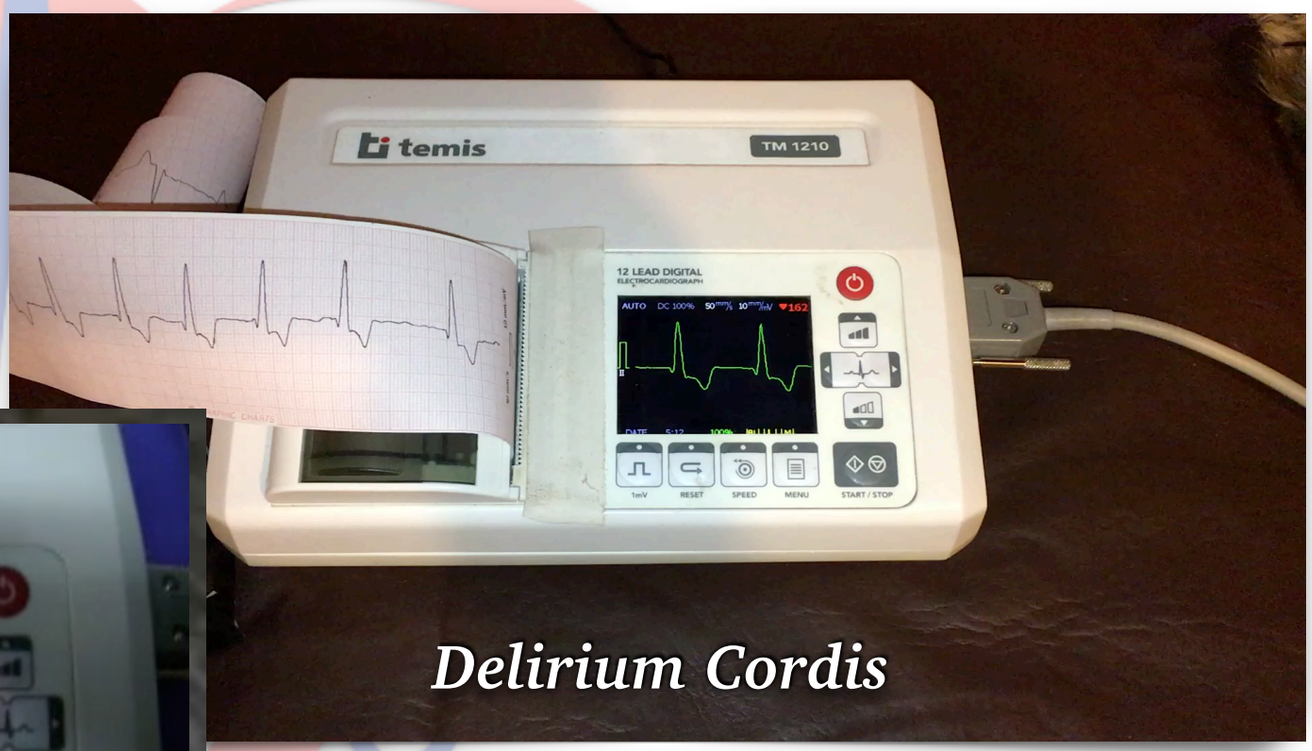
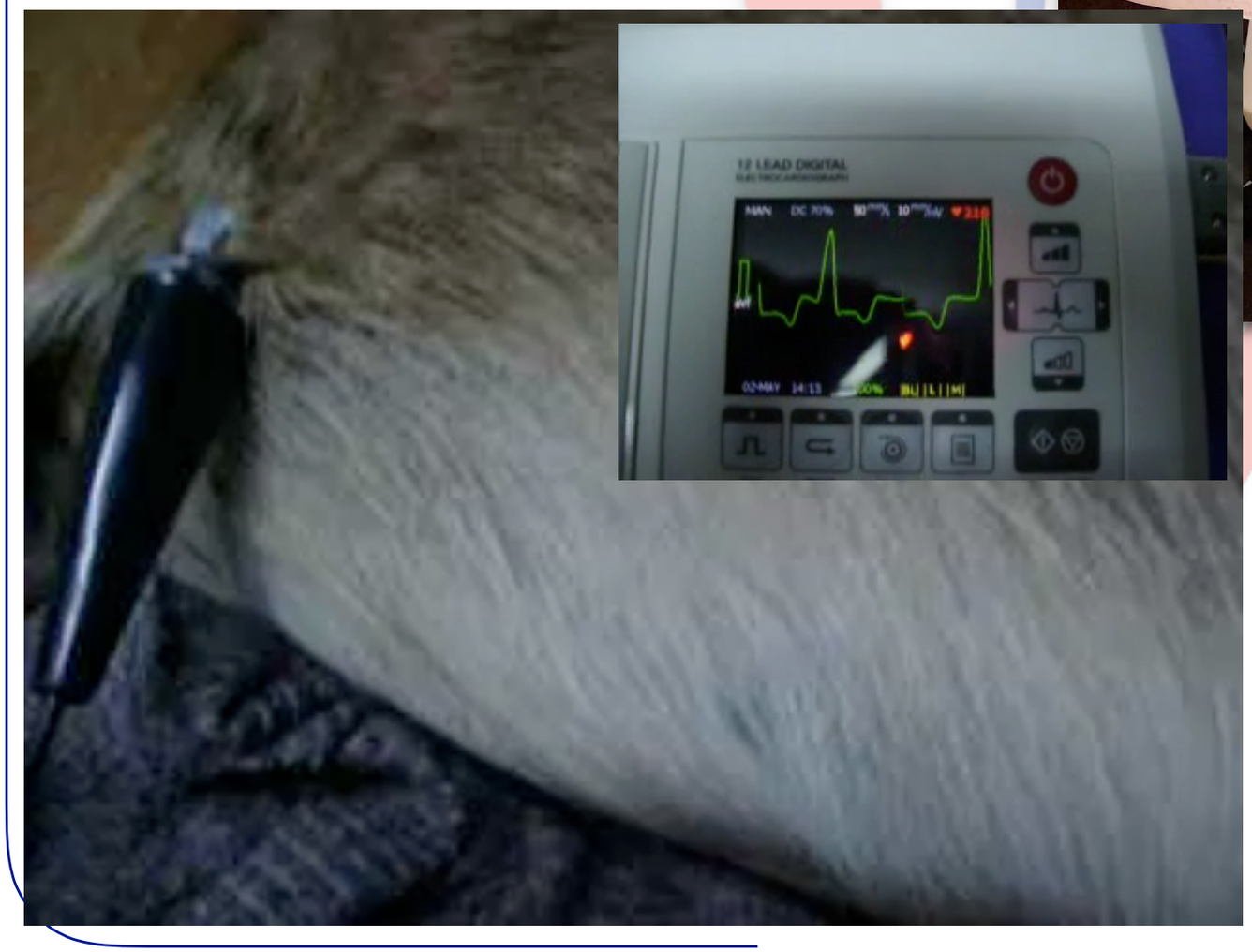
EXAMEN FÍSICO

- Las arritmias no son observadas por el propietario pero se detectan, en general, fácilmente en la auscultación:

  - Fabricación Atrial (> 2.5 FC basal)



Delirium Cordis



- Pulso: deficitario/débil/asincrónico
- TLLC: aumentado / mayor a 2”
- Mucosas: rosadas / rosadas pálidas
- Choque precordial: suave
- **SOPLO** ¿qué opinan?
- **FRÉMITO** ¿qué opinan?

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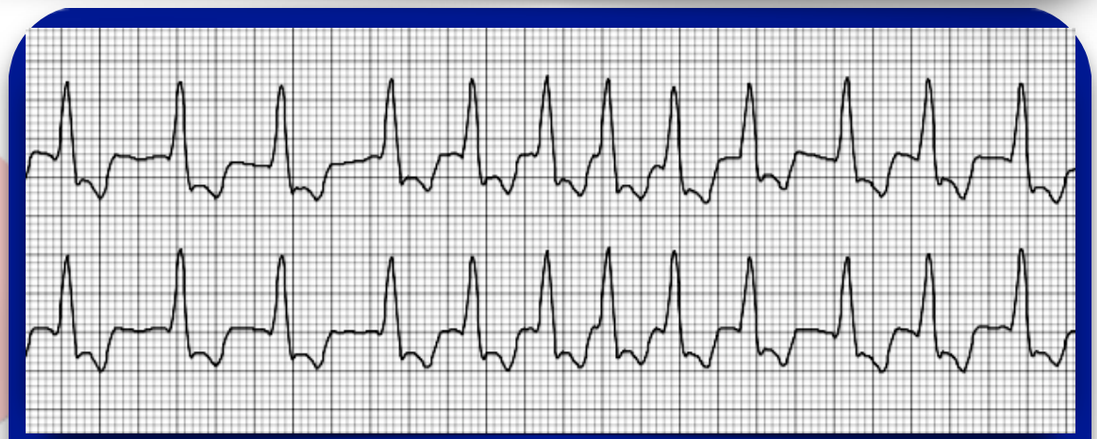
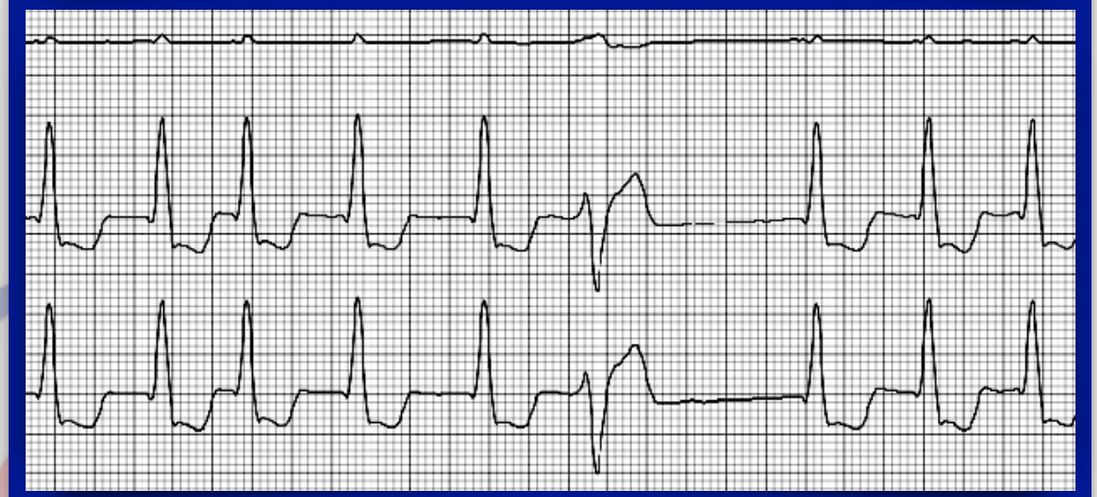
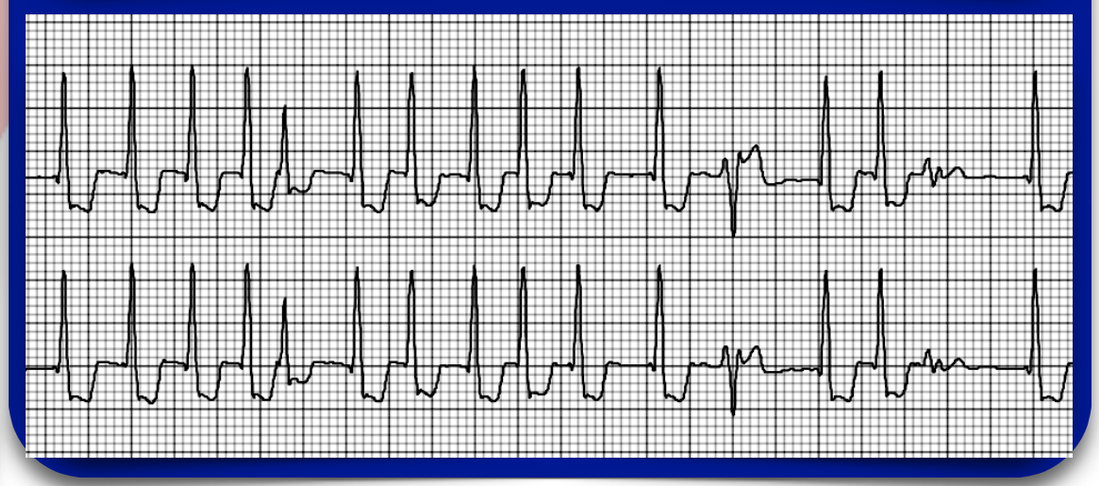
COMPLEMENTARIOS

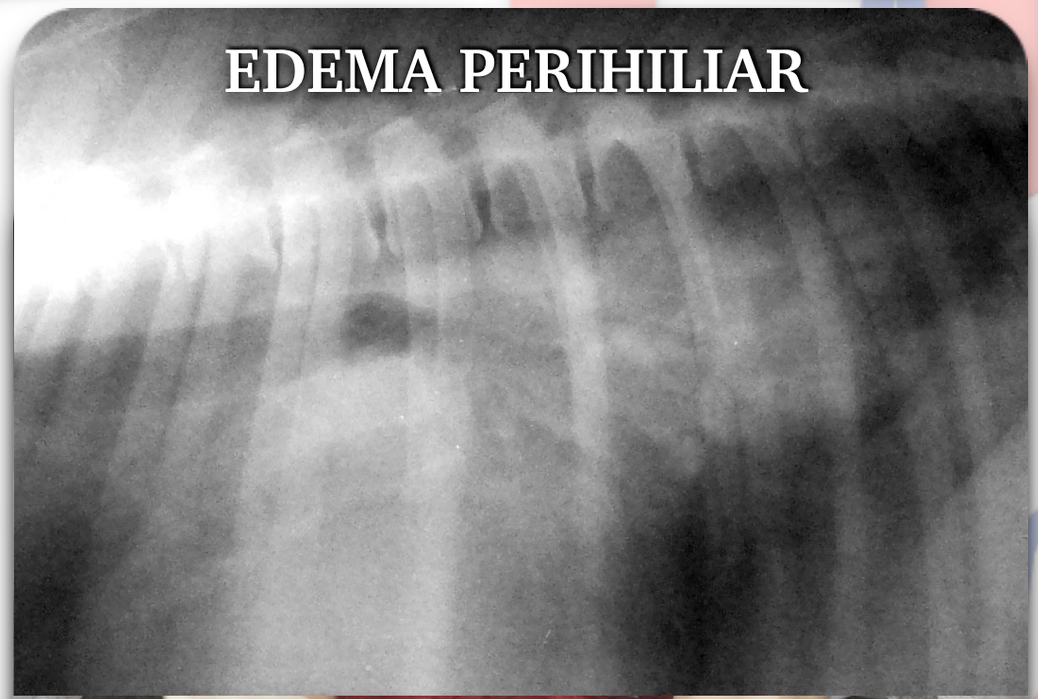
ELECTROCARDIOGRAMA

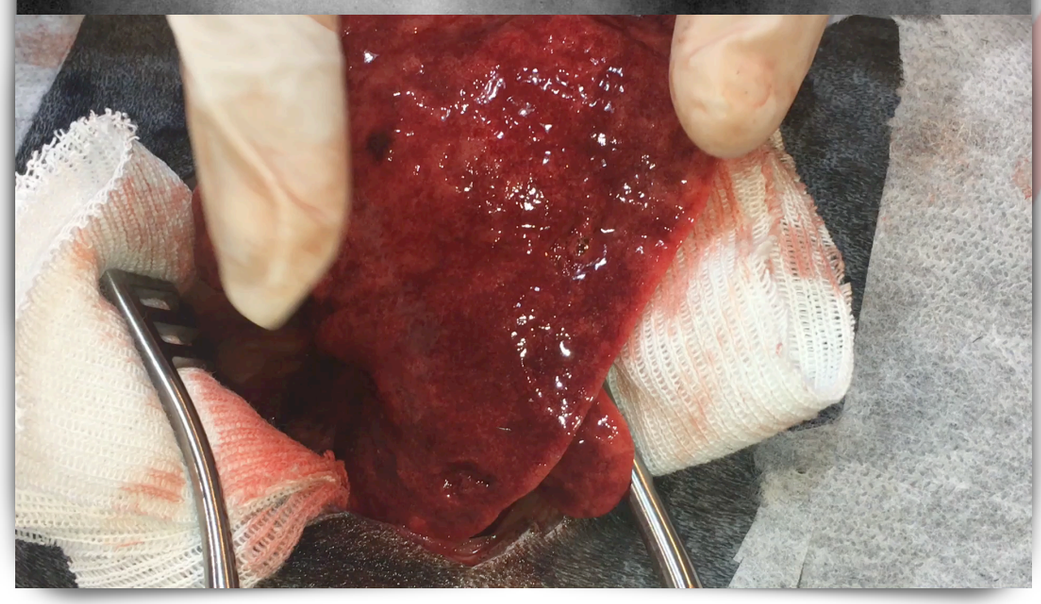






RADIOLOGÍA





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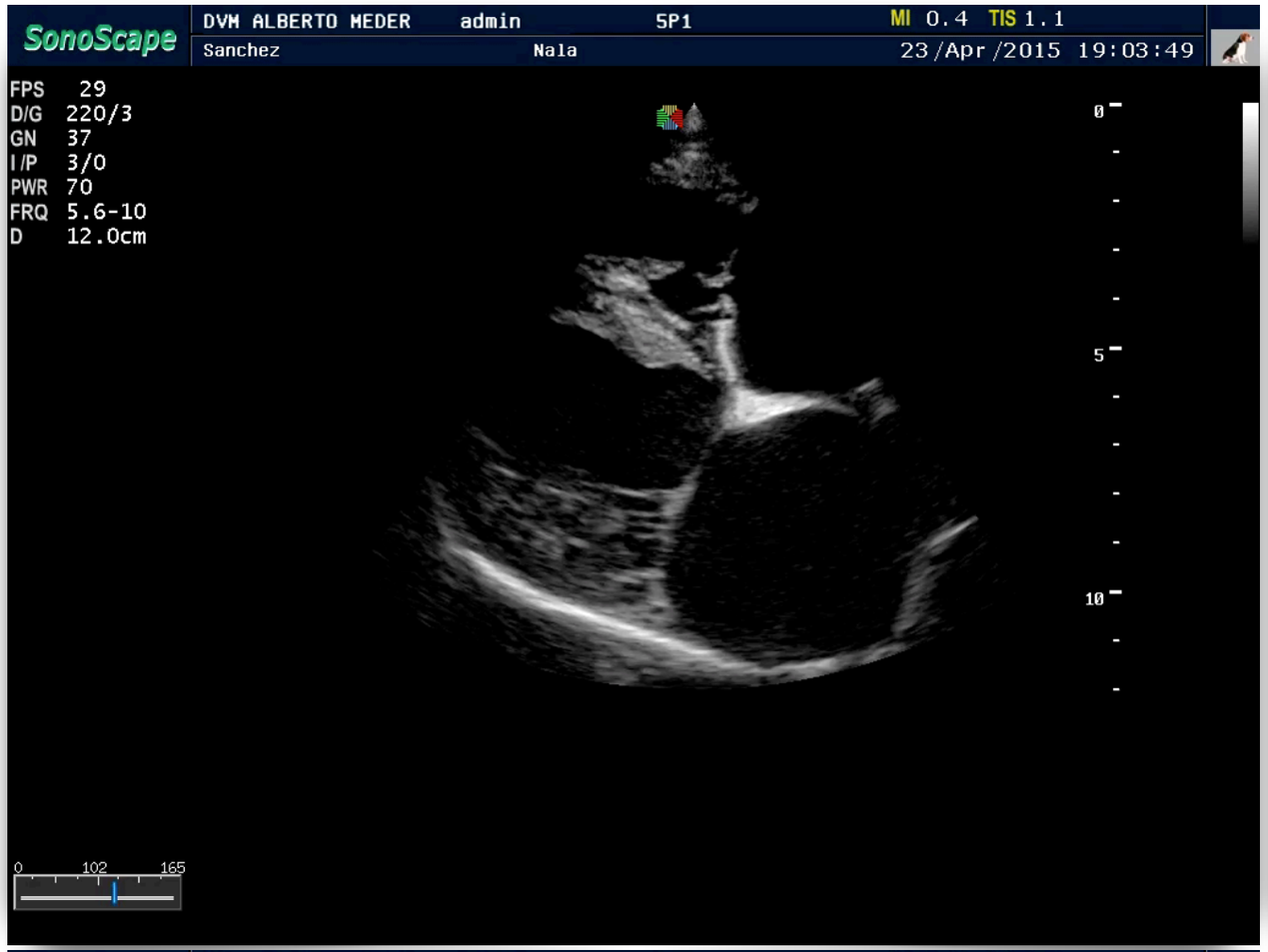
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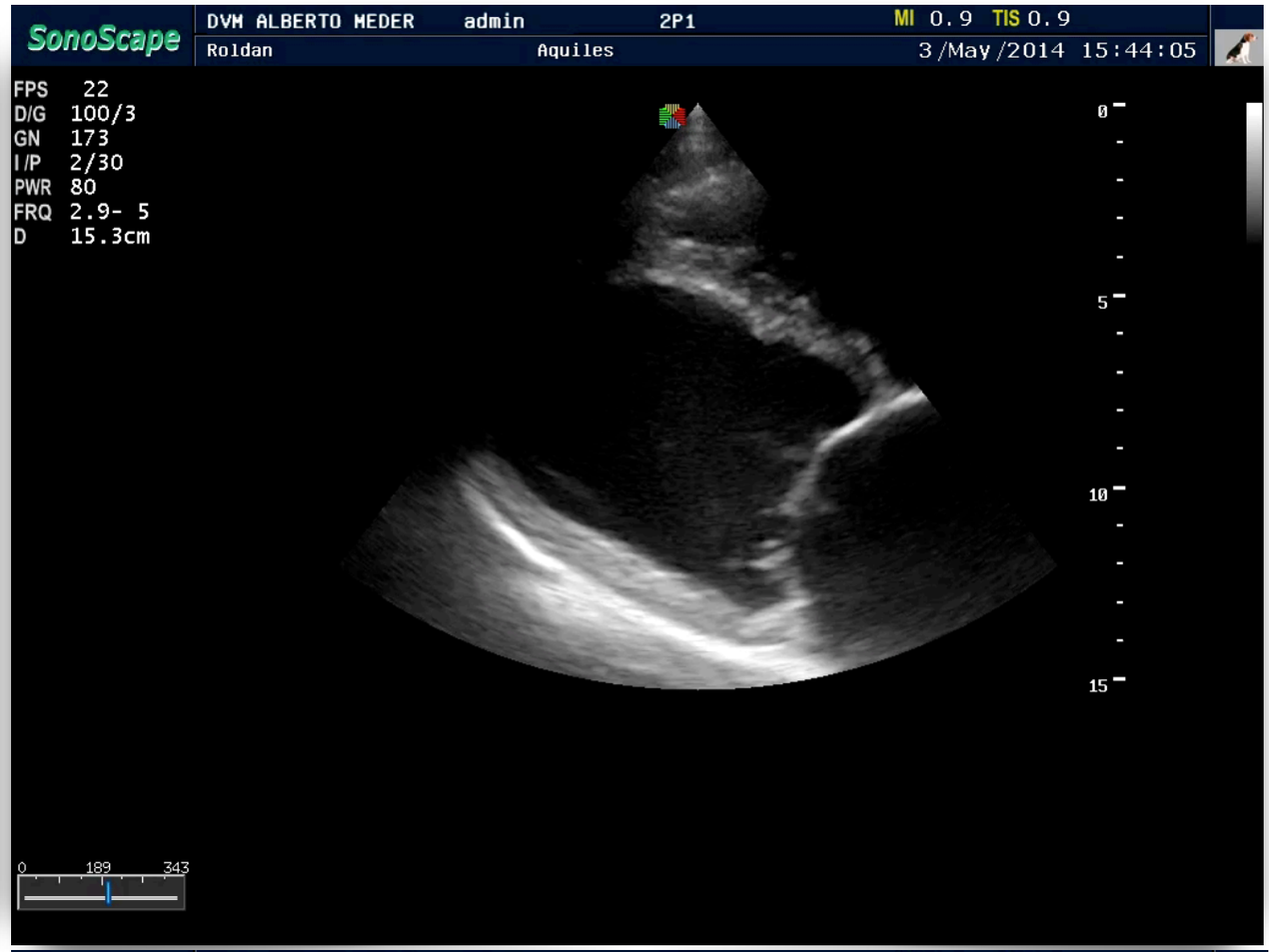
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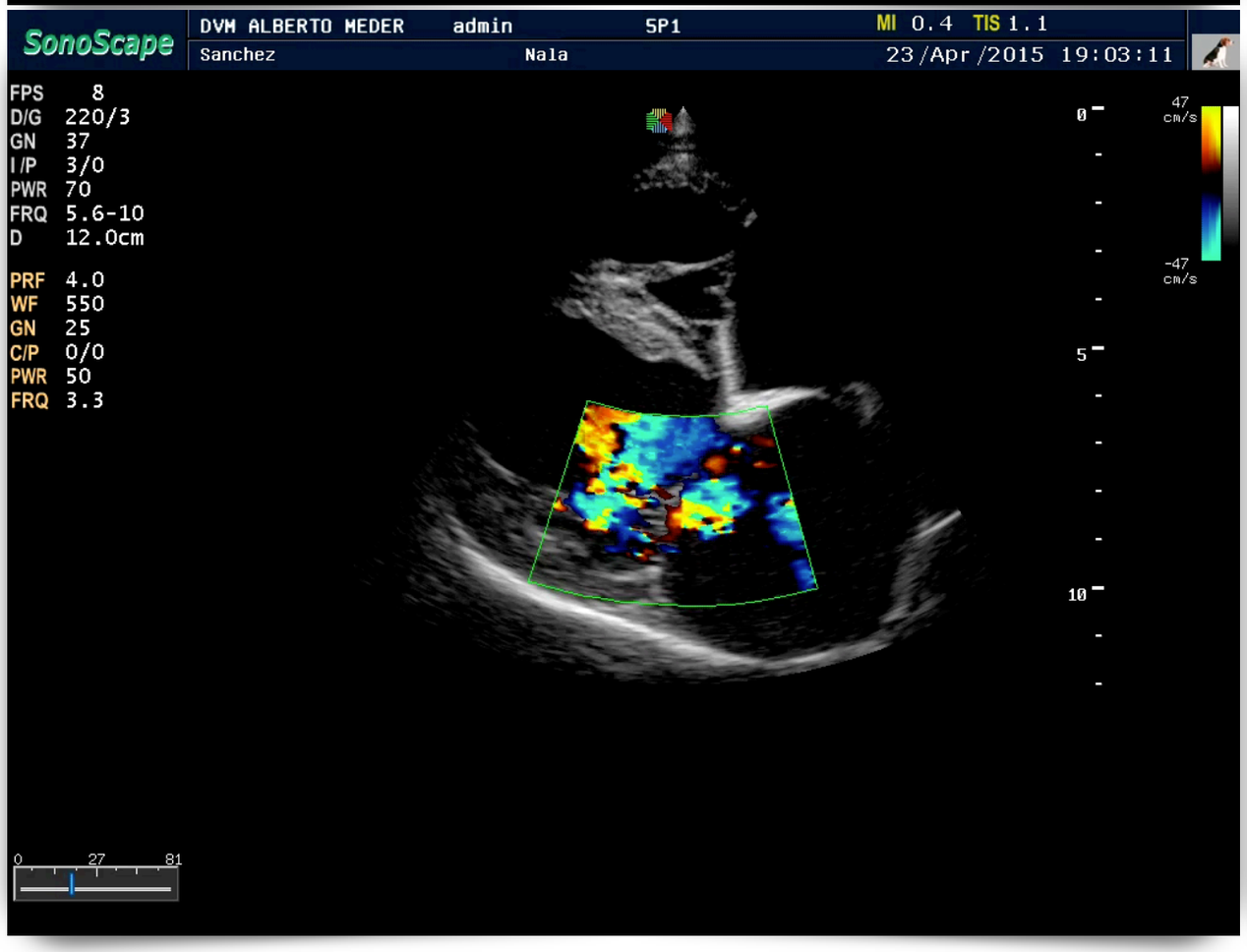
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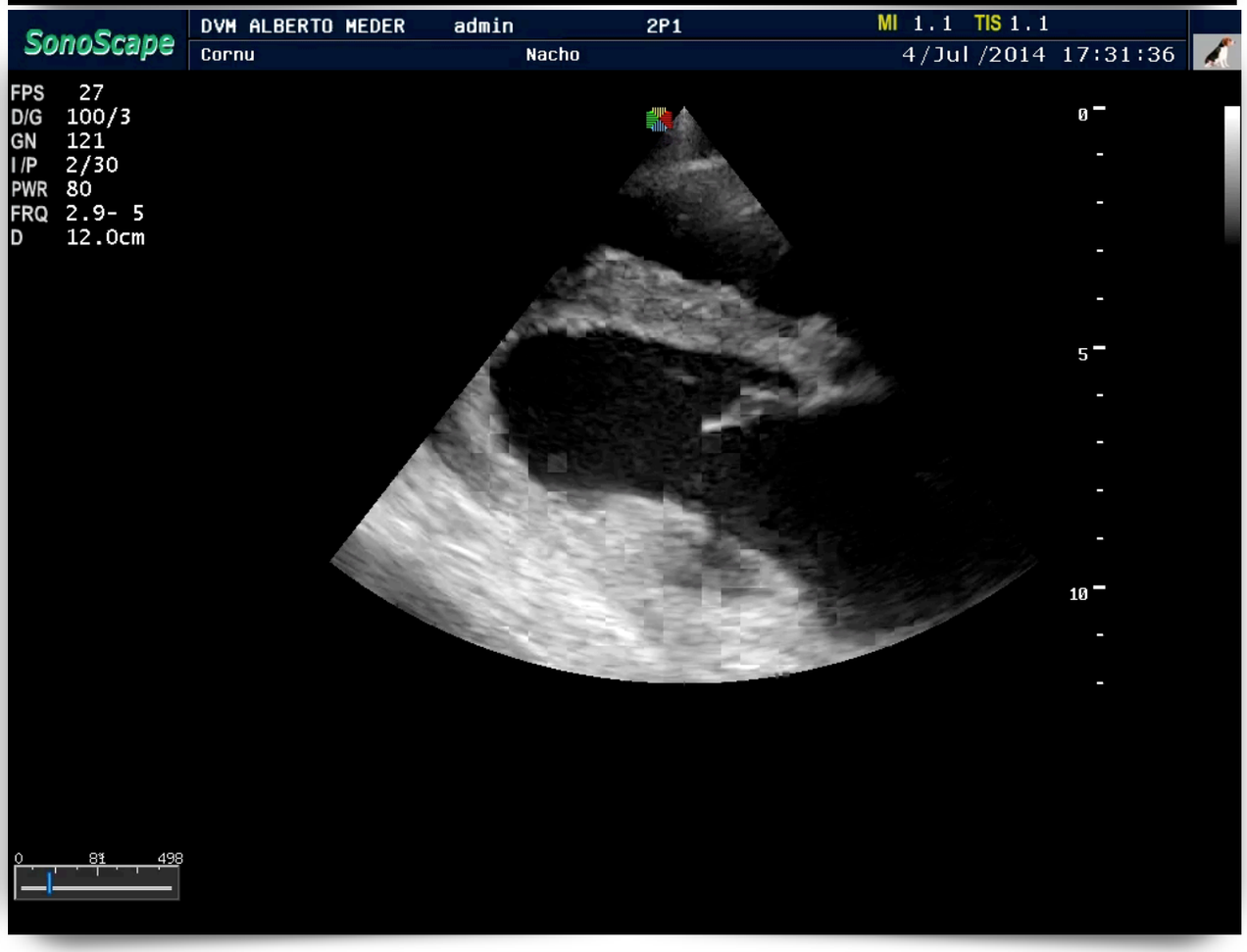
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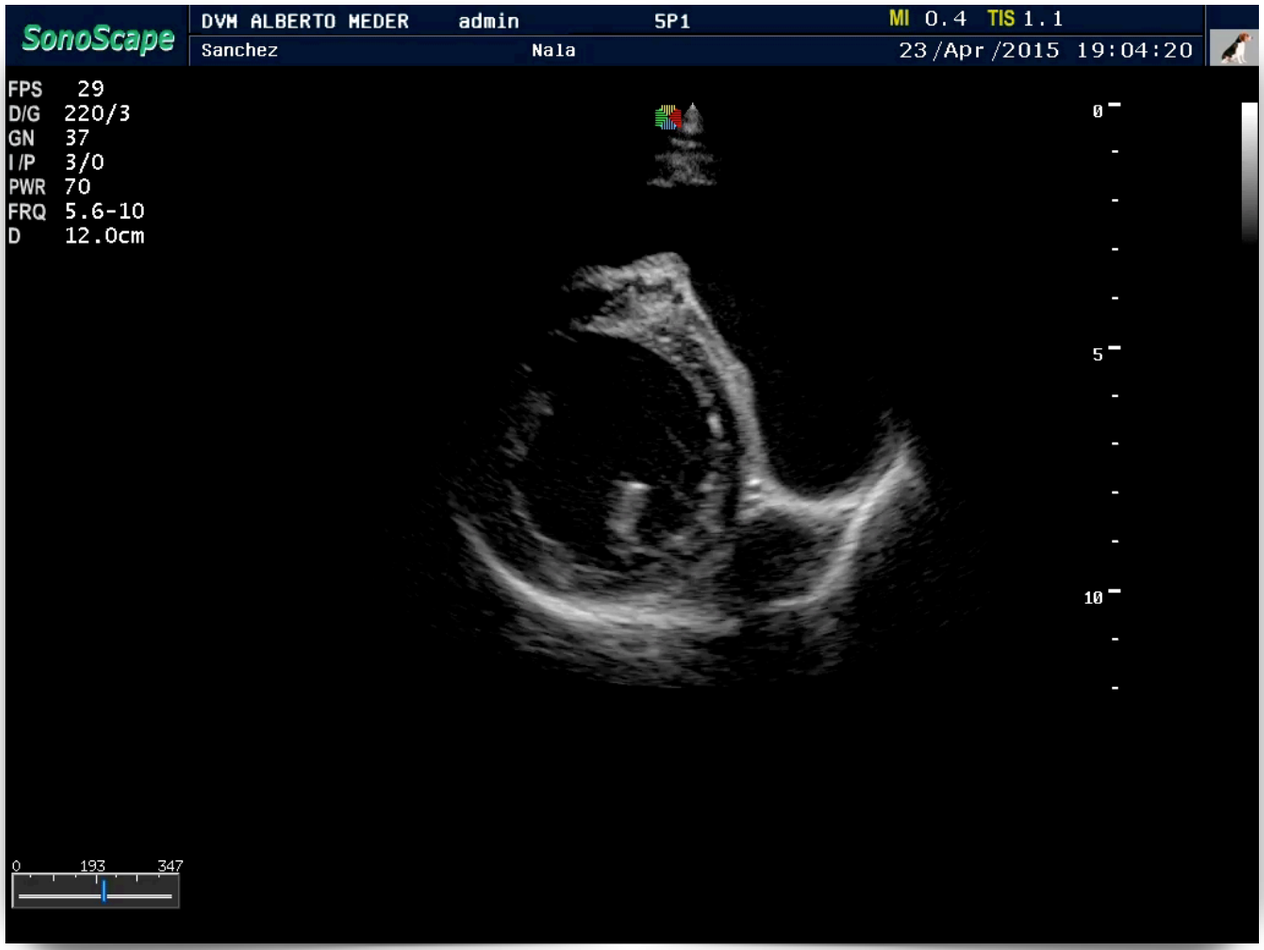
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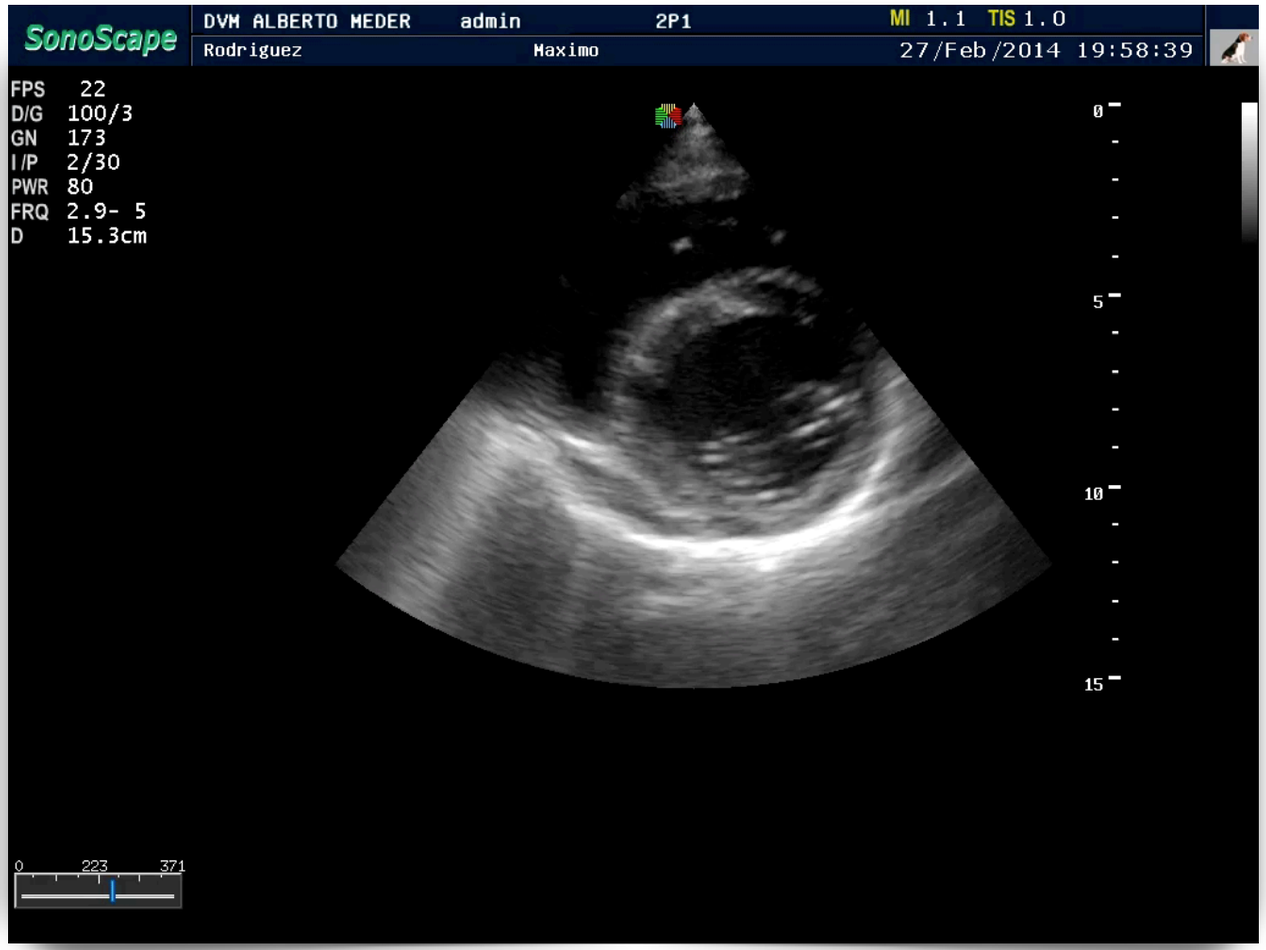
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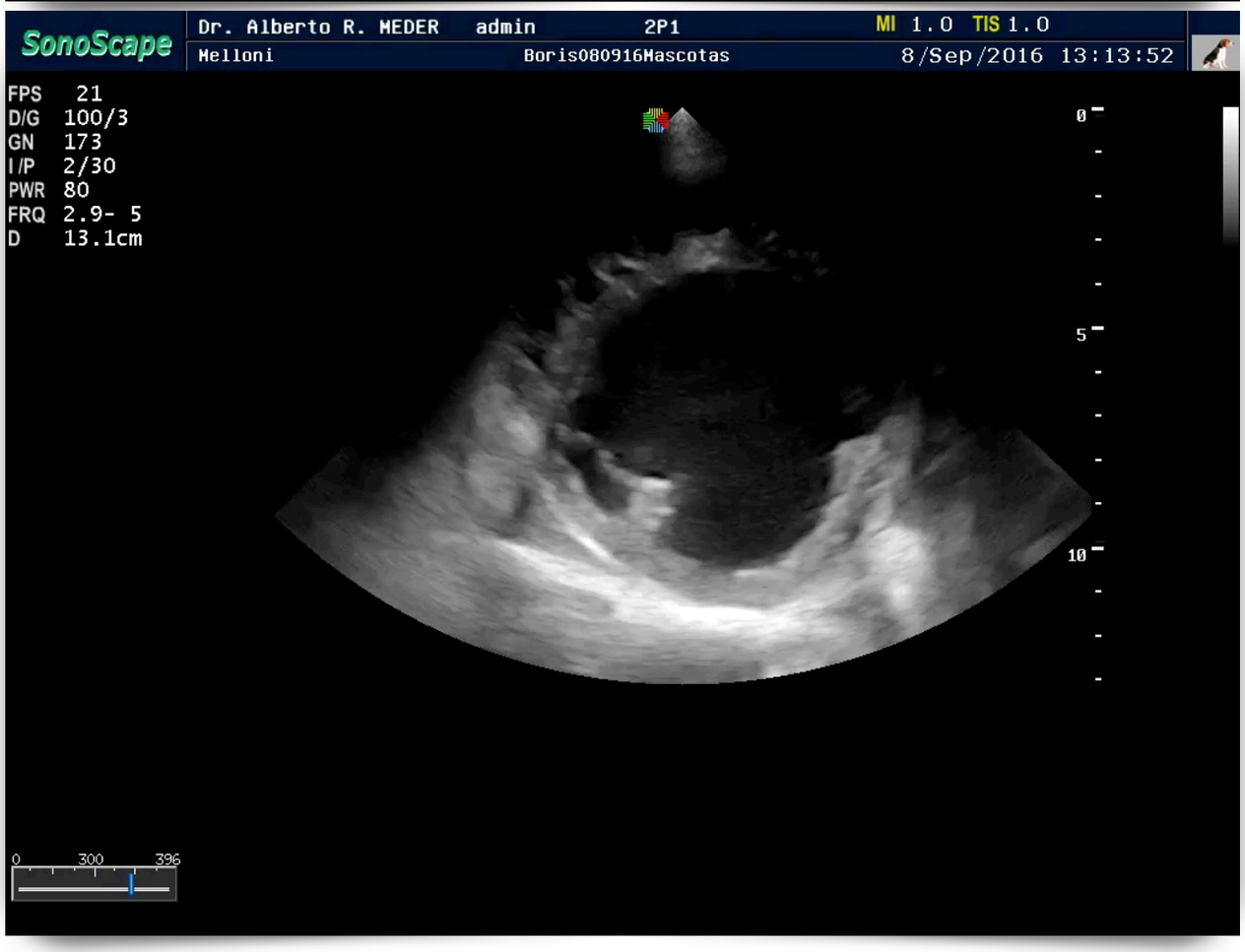
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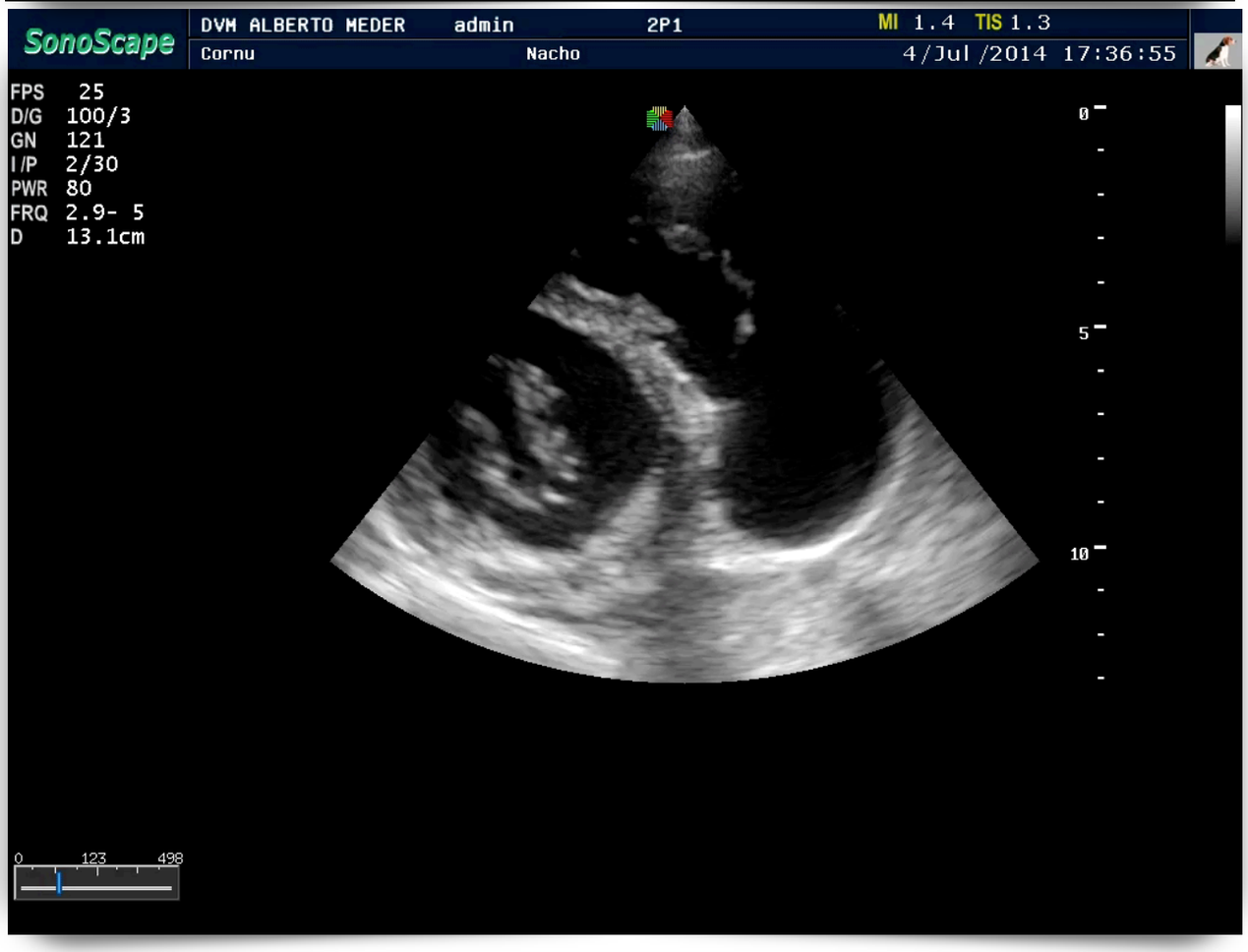
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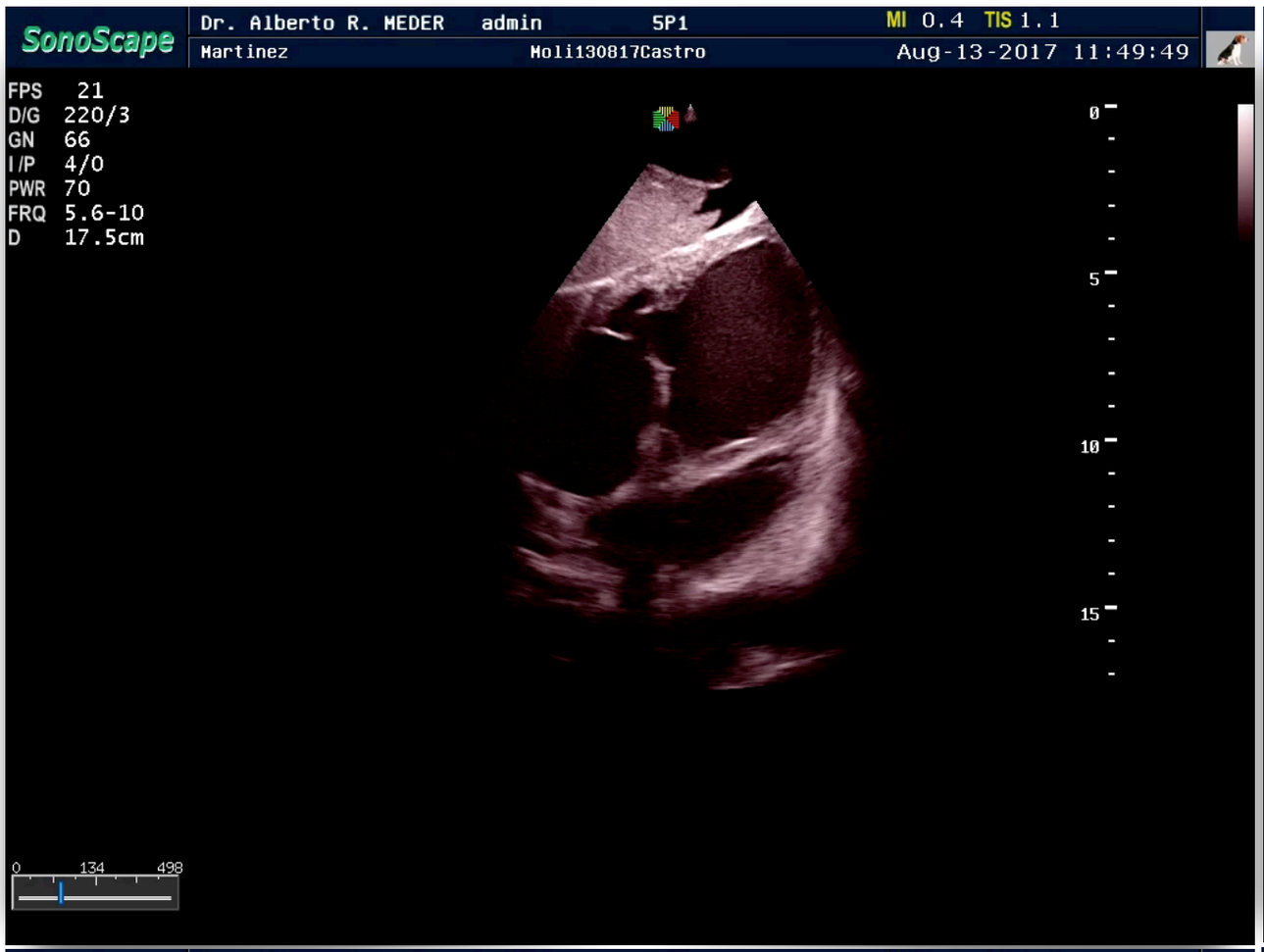
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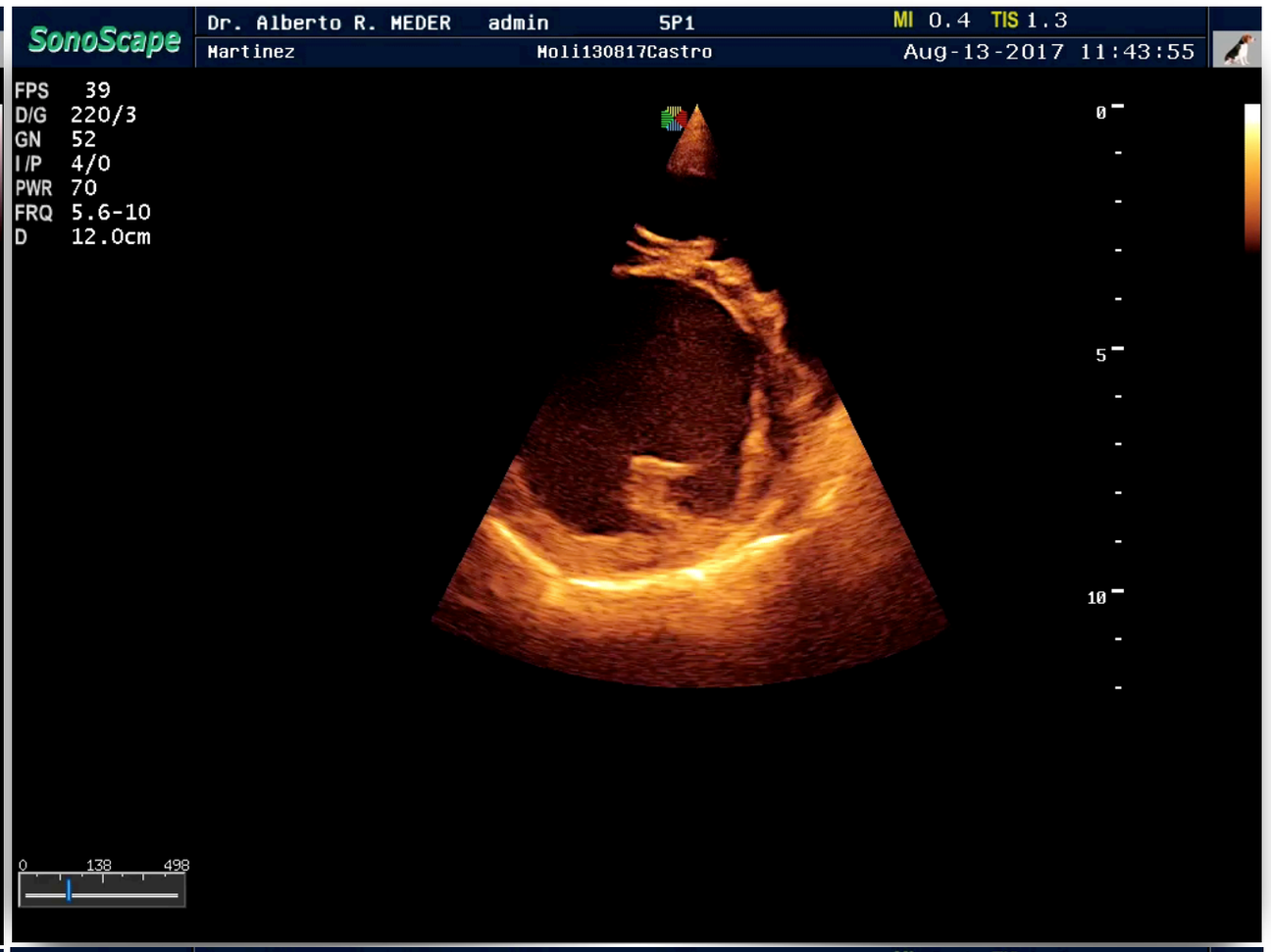
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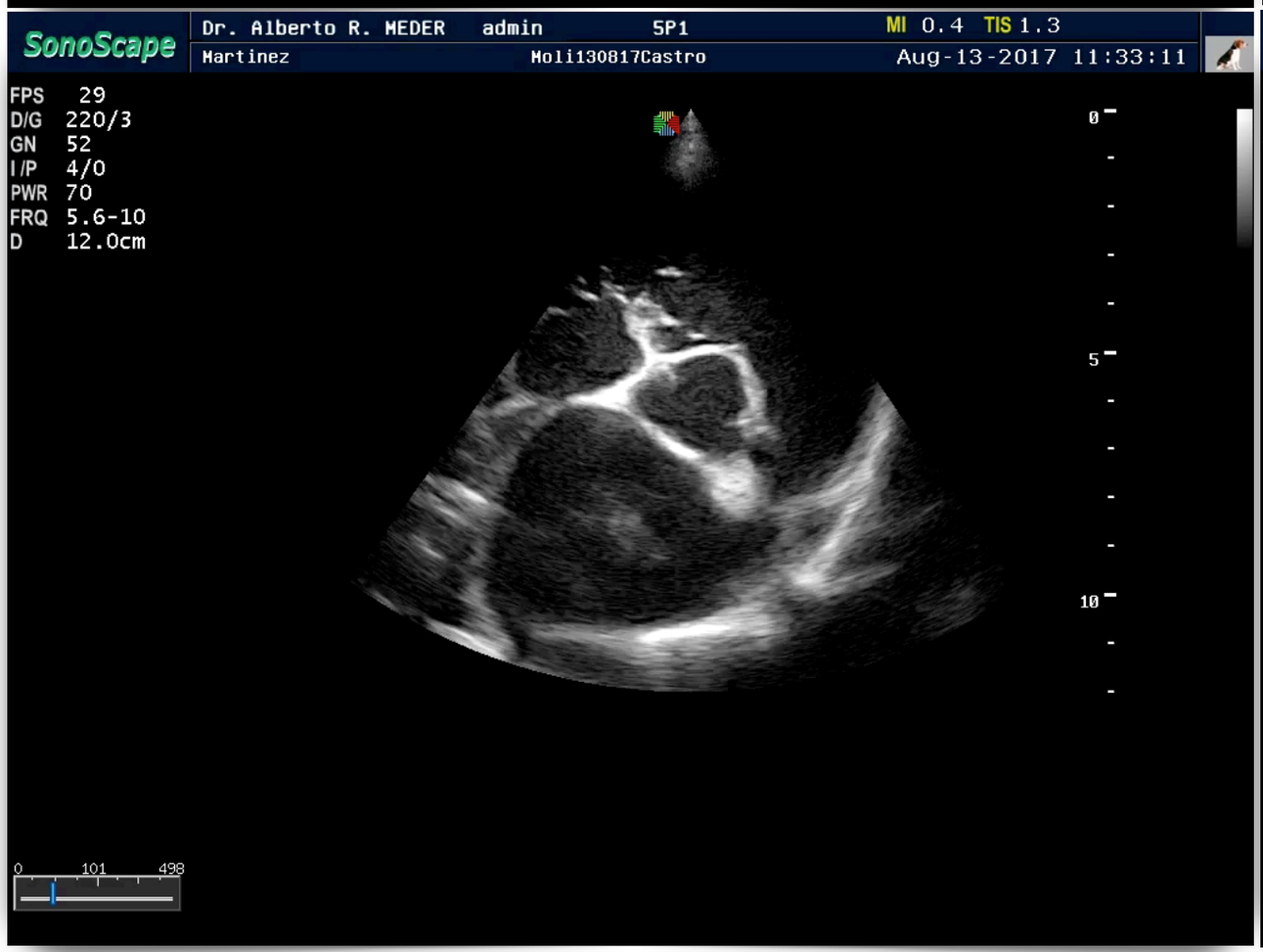
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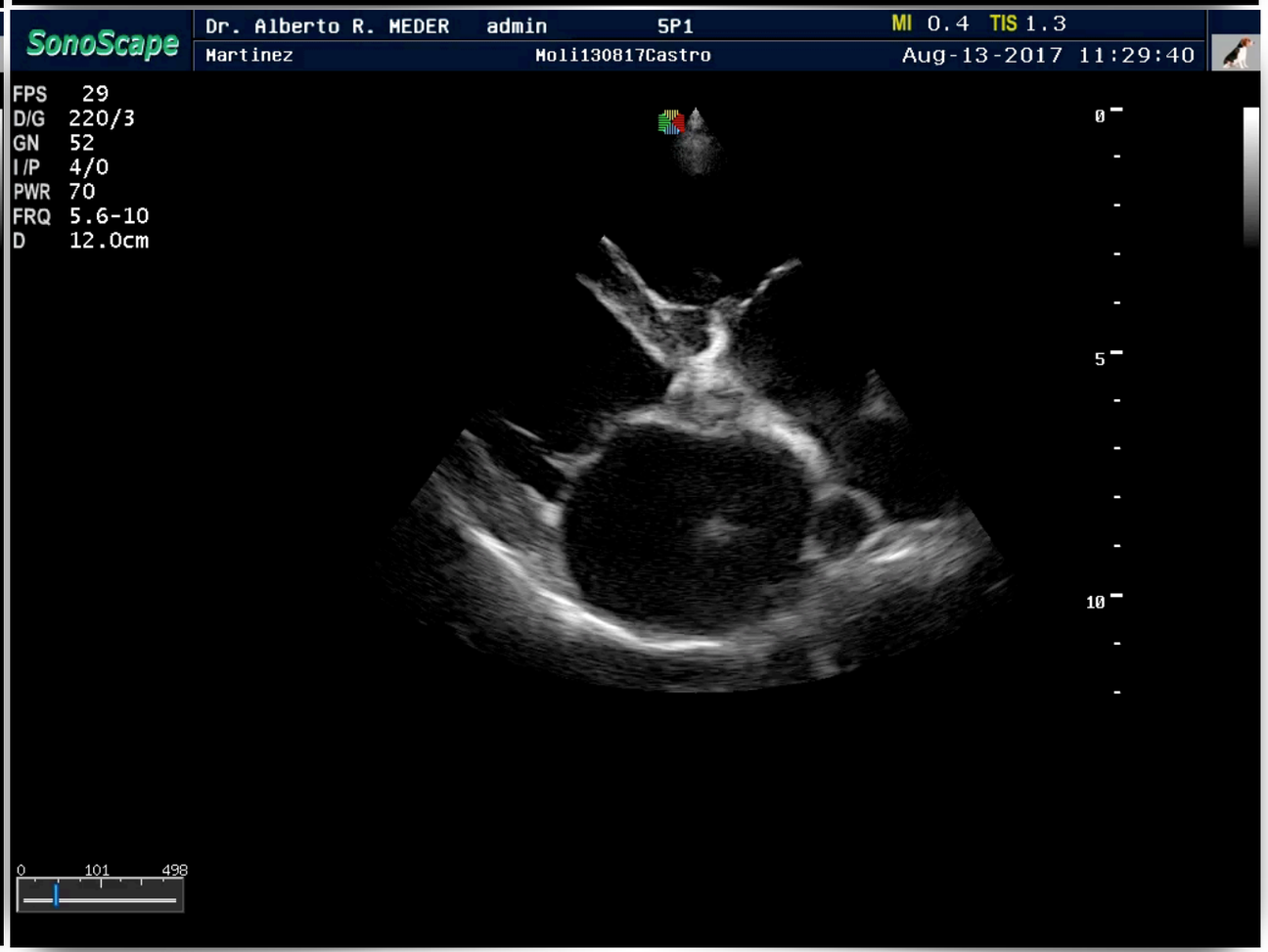
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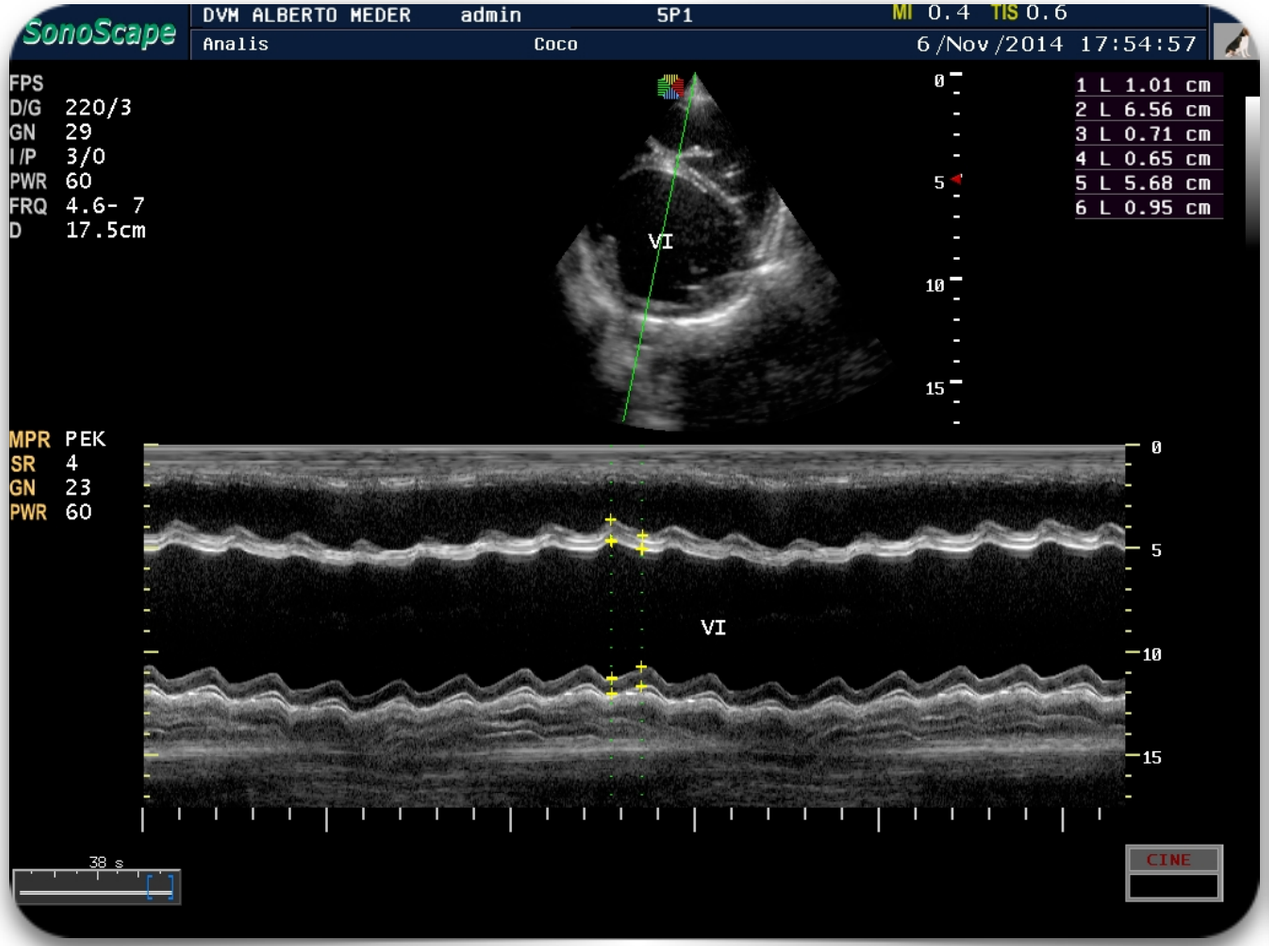
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2 L 6.56 cm
3 L 0.71 cm
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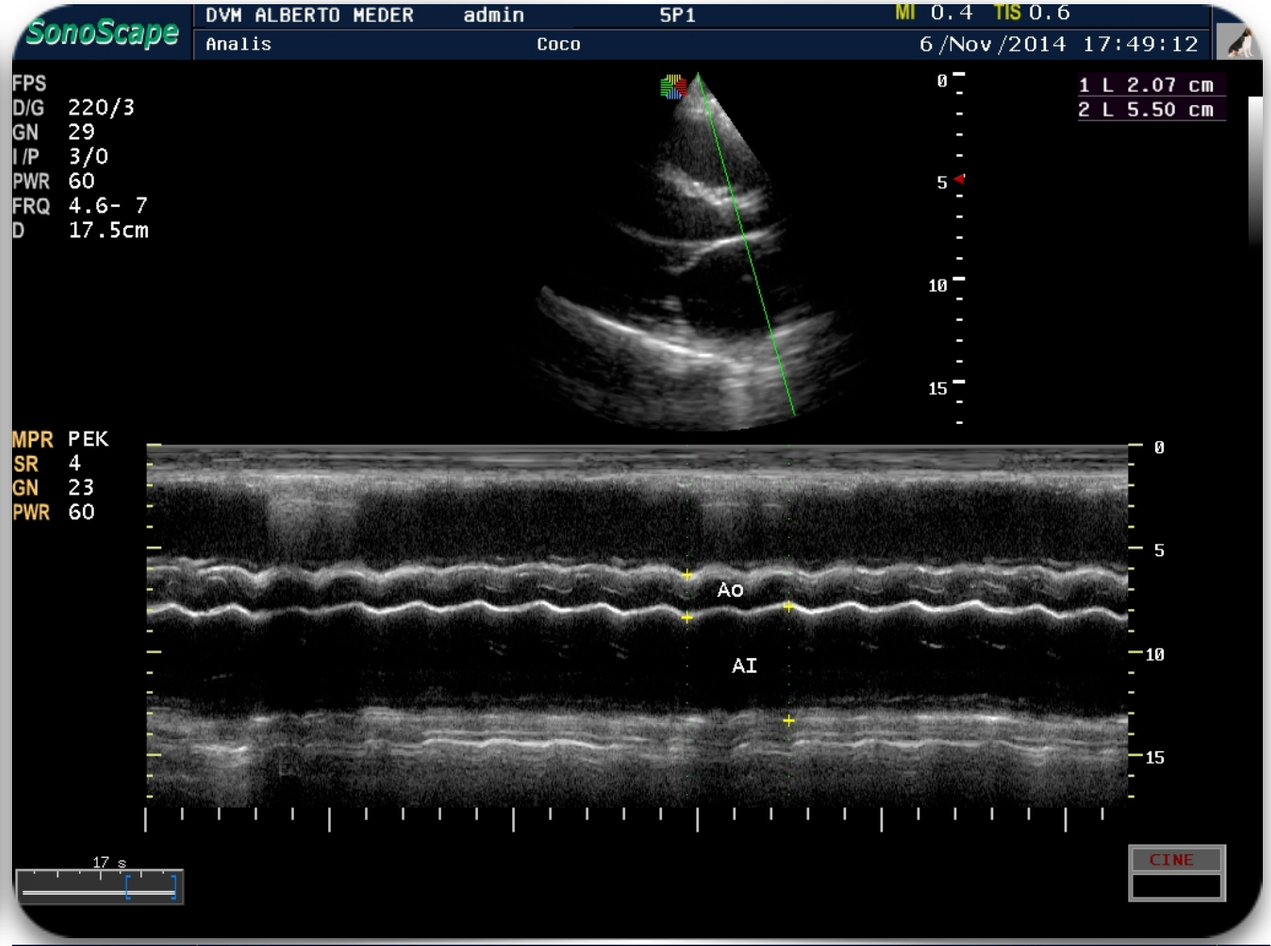
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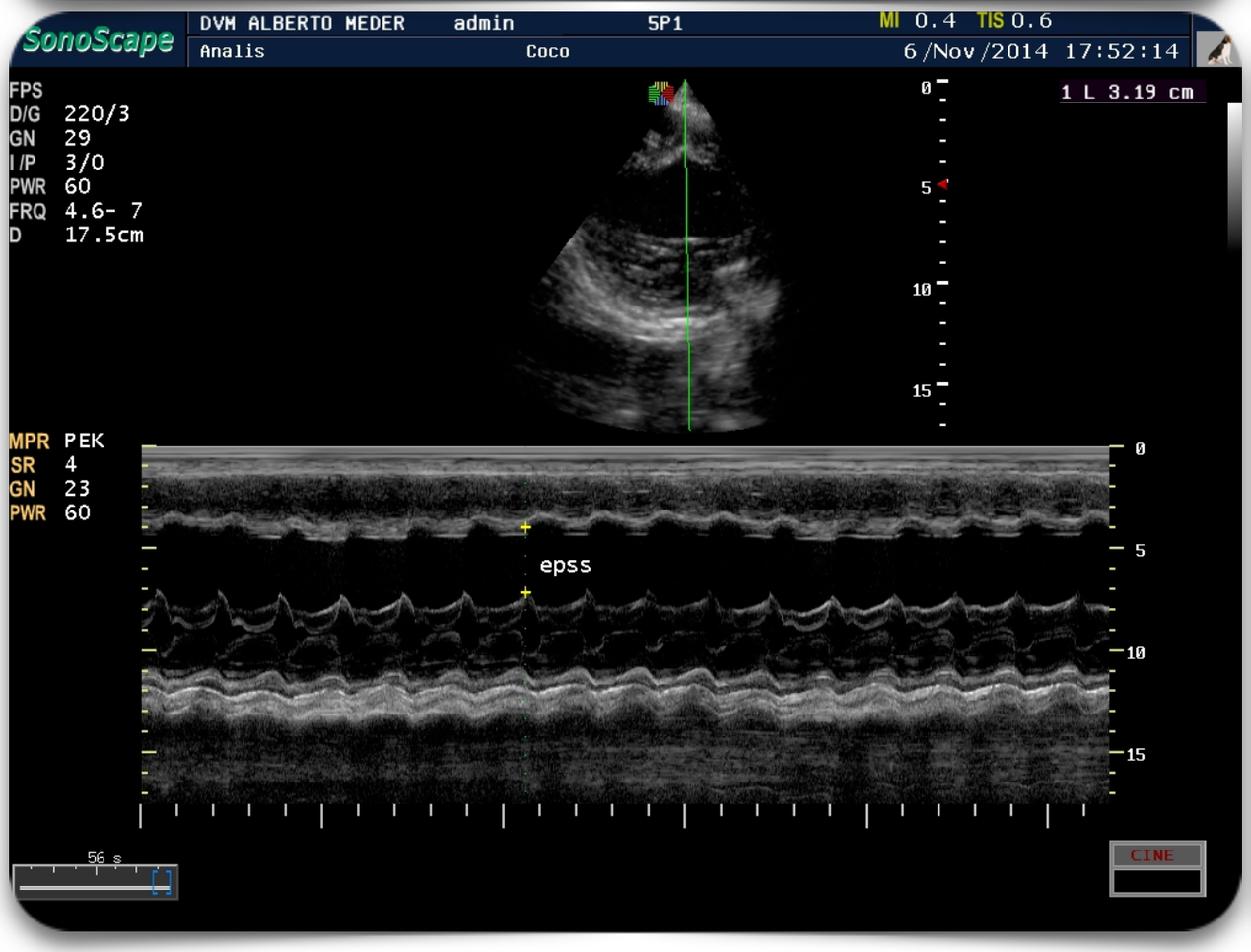
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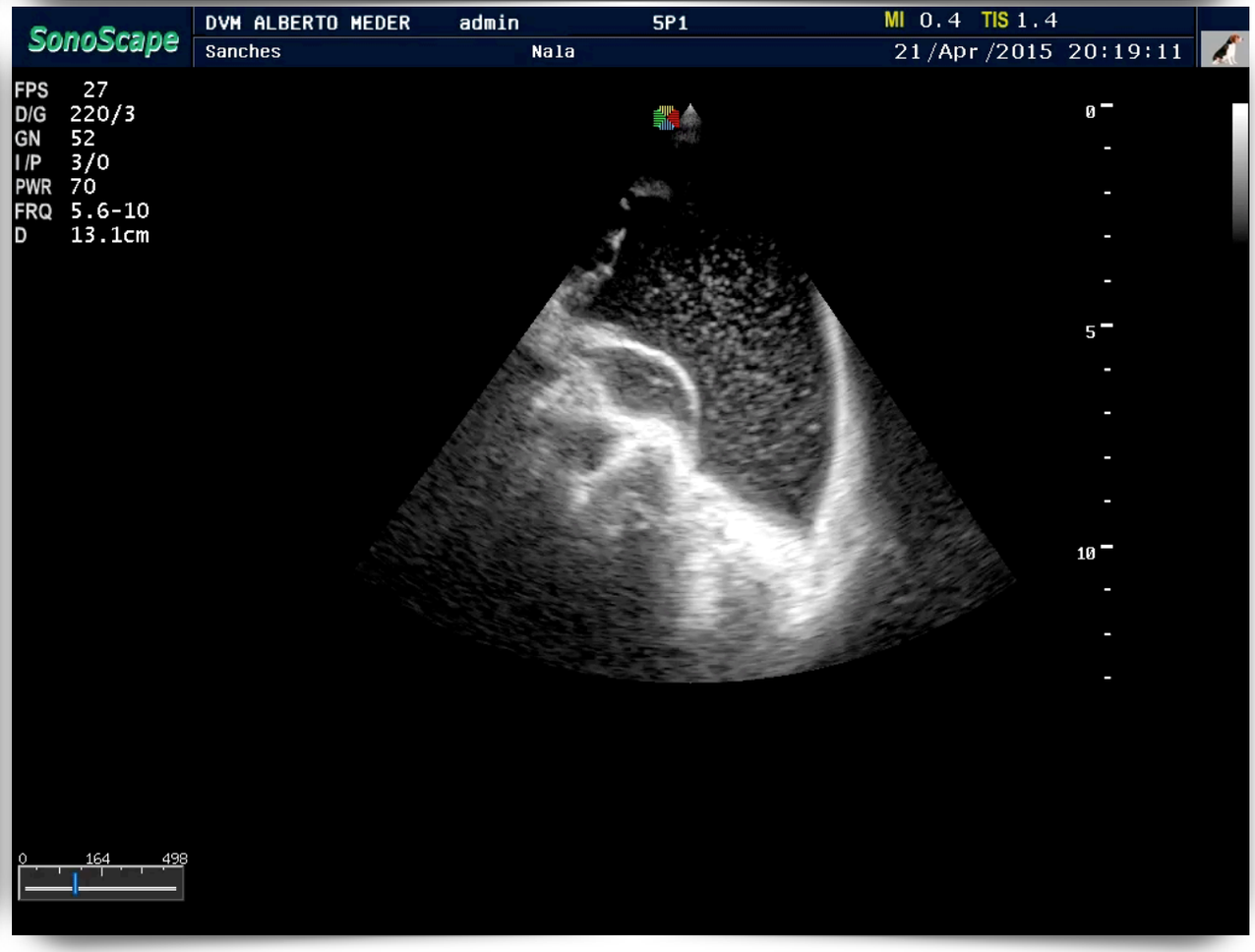
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FPS 27
D/G 220/3
GN 52
I/P 3/0
PWR 70
FRQ 5.6-10
D 13.1cm
0 164 498

SonoScape

Dr. Alberto R. MEDER admin 2P1

MI 0.09 TIS 0.9 TIB 1.6

Jan-11-2002 00:23:10

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GN 110
I /P 11/30
PWR 80
FRQ 2.9- 5
D 18.0cm

0
5
10
15

θ= 22 °

PRF 24.0
WF 300
GN 0
FRQ 2.0
PWR 70
DYN 2

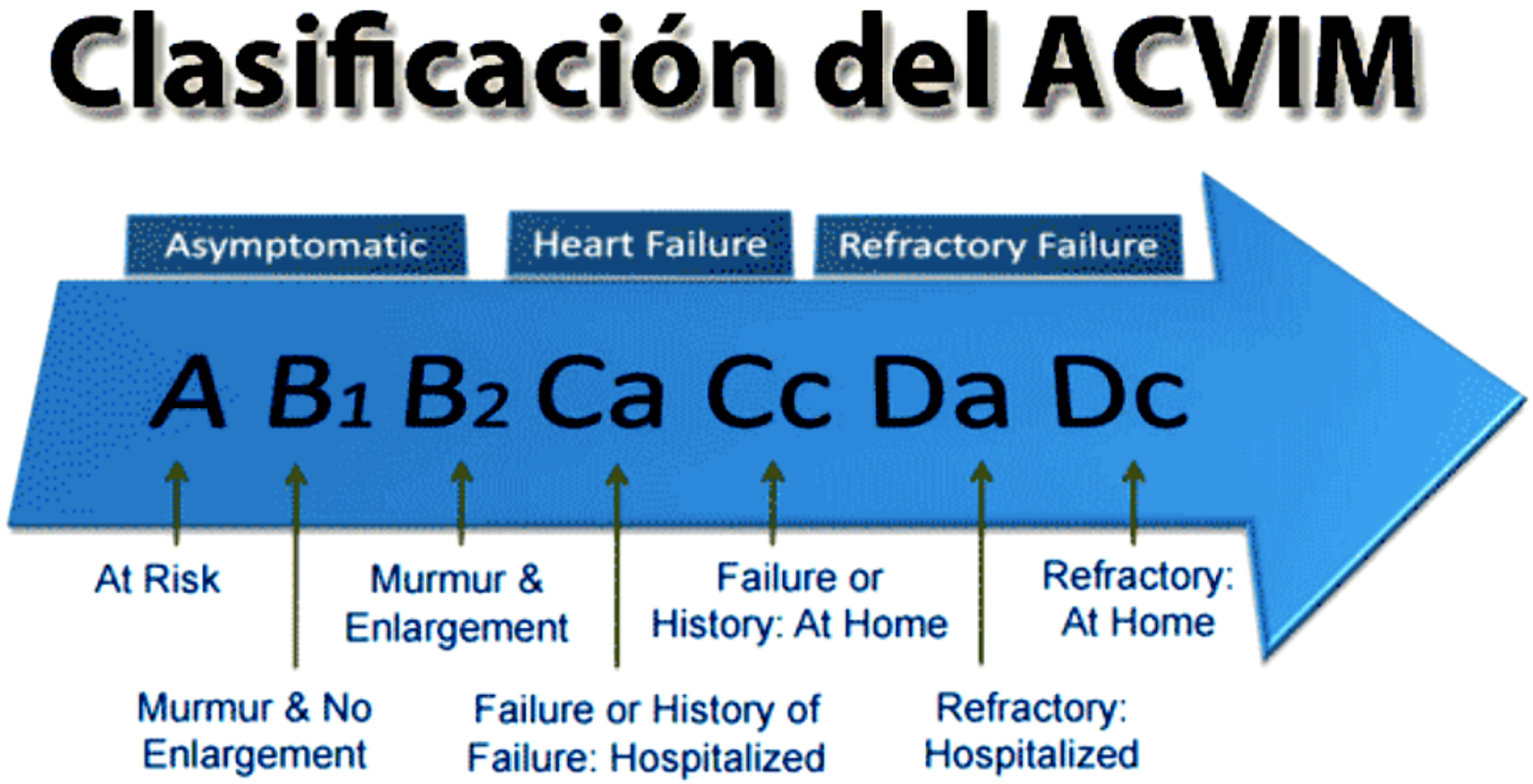
300
200
100
cm/s
-100
-200
-300
-400
-500
-600

22 s

CINE

THI CW

TRATAMIENTO MÉDICO



DIGOXINA

PIMOBENDAN

AMIODARONA

SOTALOL

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Echocardiographic phenotype of canine dilated cardiomyopathy differs based on diet type☆

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Table 2 Median and 95% confidence intervals for echocardiographic variables at diagnosis.

	GB (n = 12)	All GF (n = 36)	GF-1 (n = 14)	GF-o (n = 22)	*P* value
FS (%)	13.0 (11.1–17.8)	15.5 (13.8–16.9)	14.5 (11.9–18.2)	16.5 (13.6–17.3)	0.9
LVIDdN	2.13 (2.01–2.20)	2.36 (2.28–2.48)[a]	2.49 (2.36–2.64)[a]	2.22 (2.15–2.42)	0.003
LVIDsN	1.71 (1.57–1.79)	1.85 (1.80–1.99)	1.91 (1.86–2.16)[a]	1.77 (1.68–1.93)	0.01
SI	1.34 (1.30–1.58)	1.27 (1.26–1.37)	1.23 (1.19–1.30)[a]	1.32 (1.30–1.44)	0.02
LA:Ao	1.98 (1.74–2.16)	2.02 (1.88–2.15)	2.11 (1.89–2.39)	1.94 (1.78–2.10)	0.5

FS (%), percent fractional shortening; LVIDdN, normalized left ventricular internal diameter in diastole; LVIDsN, normalized left ventricular diameter in systole; SI, diastolic left ventricular sphericity index; LA:Ao, left atrial to aortic ratio; GB, grain-based diets; All GF, all grain-free diets, GF-1, most common grain-free diet, GF-o, other grain-free diets.

[a] Significantly different from GB.

- *El peso corporal de los caninos que comían dietas a base de carne pero sin granos presentaban un peso menor*
- *Los diámetros ecocardiográficos del ventrículo izquierdo (telediastólico/telesistólico) eran mayores en los perros que comían dietas a base de carne sin granos*
- *Las dietas de calidad media a mala con carne y sin granos presentaban diámetros ecocardiográficos mayores que las de calidad media a alta*
- *Los perros que comían dietas a base de carne sin granos mejoraron en sus variables ventriculares izquierdas cuando se mejoró la dieta o se suplemento con taurina(lenta)*
- *La calidad del alimento puede estar relacionada con la presencia del nutriente, su biodisponibilidad y su procesamiento*

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J Vet Intern Med 2012;26:1337–1349

Efficacy of Pimobendan in the Prevention of Congestive Heart Failure or Sudden Death in Doberman Pinschers with Preclinical Dilated Cardiomyopathy (The PROTECT Study)

N.J. Summerfield, A. Boswood, M.R. O'Grady, S.G. Gordon, J. Dukes-McEwan, M.A. Oyama, S. Smith, M. Patteson, A.T. French, G.J. Culshaw, L. Braz-Ruivo, A. Estrada, M.L. O'Sullivan, J. Loureiro, R. Willis, and P. Watson

Background: The benefit of pimobendan in delaying the progression of preclinical dilated cardiomyopathy (DCM) in Dobermans is not reported.

Hypothesis: That chronic oral administration of pimobendan to Dobermans with preclinical DCM will delay the onset of CHF or sudden death and improve survival.

Animals: Seventy-six client-owned Dobermans recruited at 10 centers in the UK and North America.

Methods: The trial was a randomized, blinded, placebo-controlled, parallel group multicenter study. Dogs were allocated in a 1:1 ratio to receive pimobendan (Vetmedin capsules) or visually identical placebo.The composite primary endpoint was prospectively defined as either onset of CHF or sudden death. Time to death from all causes was a secondary endpoint.

Results: The proportion of dogs reaching the primary endpoint was not significantly different between groups ($P = .1$). The median time to the primary endpoint (onset of CHF or sudden death) was significantly longer in the pimobendan (718 days, IQR 441–1152 days) versus the placebo group (441 days, IQR 151–641 days) (log-rank $P = 0.0088$). The median survival time was significantly longer in the pimobendan (623 days, IQR 491–1531 days) versus the placebo group (466 days, IQR 236–710 days) (log-rank $P = .034$).

Conclusion and Clinical Importance: The administration of pimobendan to Dobermans with preclinical DCM prolongs the time to the onset of clinical signs and extends survival. Treatment of dogs in the preclinical phase of this common cardiovascular disorder with pimobendan can lead to improved outcome.

Key words: Cardiology; Cardiovascular; Clinical trials; Evidence based medicine; Survival; Therapy.

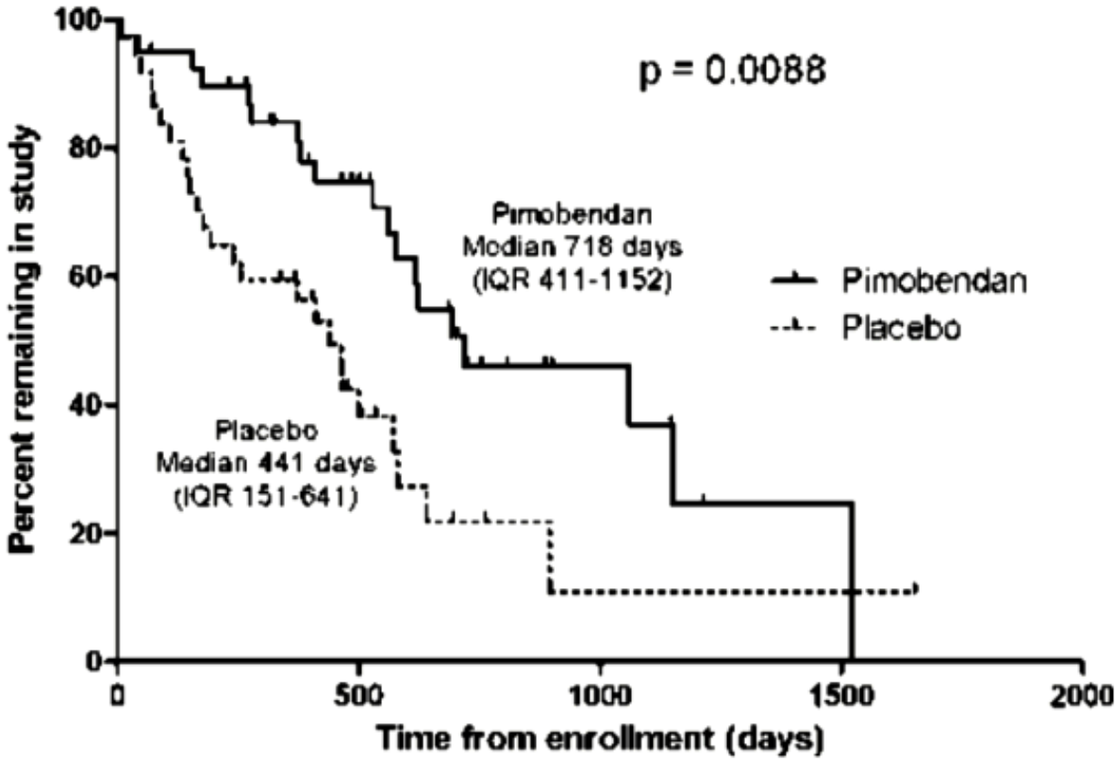

Fig 2. Kaplan Meier survival curves plotting the estimated percentage of dogs in each group that have not yet met the primary endpoint, against time. IQR, interquartile range

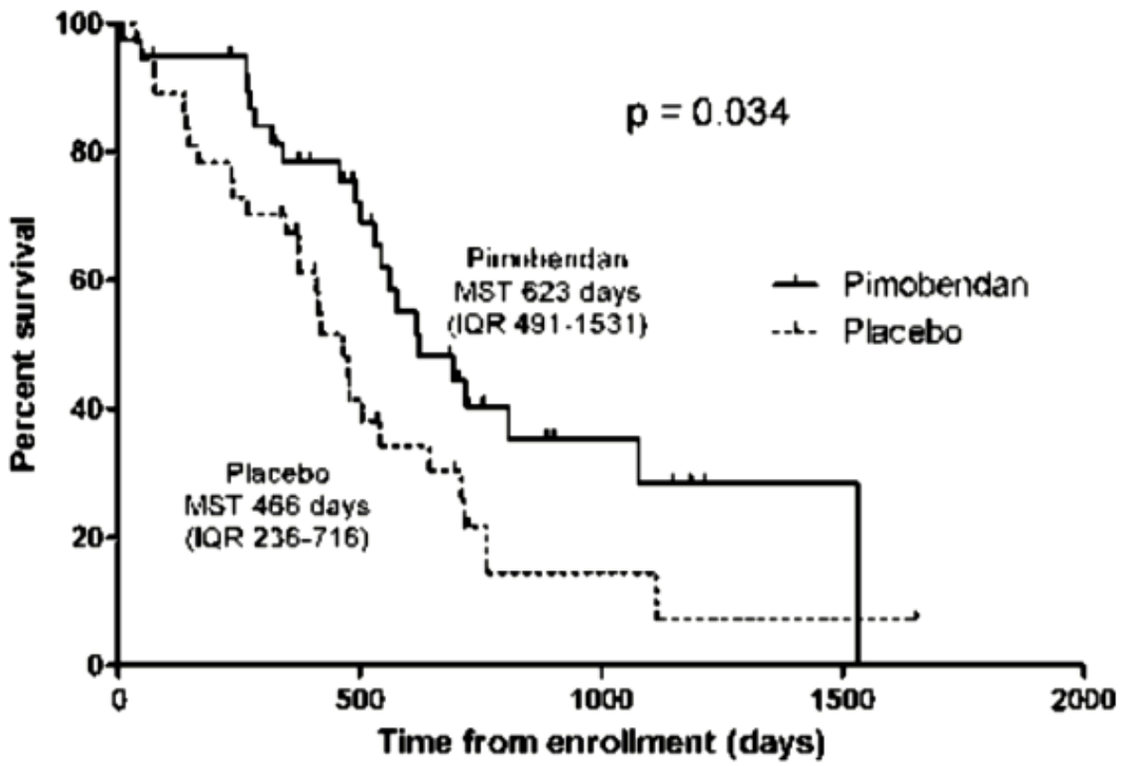

Fig 3. Kaplan Meier survival curves for the all-cause mortality analysis, plotting the estimated percentage of surviving dogs in each group, against time. MST, median survival time; IQR, interquartile range.

- *Los caninos asintomáticos (detectados en la fase oculta de la enfermedad) presentan, en promedio, un año más de vida sin desarrollar signos clínicos de falla cardiaca congestiva que aquellos que no lo toman*

Sin 441 días **Con 718 días**

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Mark A. Oyama, DVM, Diplomate ACVIM (Cardiology), University of Pennsylvania

Cardiac Drugs

for Treatment of Canine Heart Failure

Degenerative mitral valve disease (DMVD) and dilated cardiomyopathy (DCM) are common cardiac diseases in adult dogs. Both diseases can lead to heart failure and loss of quality and quantity of life.

Various cardiac drugs prolong survival and many others help alleviate clinical signs.

DIURETICS

INDICATIONS

[illegible] of therapy in dogs with congestive heart failure (CHF) due to DMVD or DCM.

ADMINISTRATION

Bolus injections of furosemide (2–4 mg/kg Q 2 H) are used in dogs with respiratory distress secondary to acute heart failure until respiratory rate and effort begin to improve.

Alternatively, furosemide can be given as a constant rate infusion. Purported advantages of furosemide infusion over intermittent boluses include enhanced natriuresis and diuresis and less urinary potassium loss. I recommend using 0.66 to 1 mg/kg/H, monitoring respiratory rate every hour.

Once acute heart failure is resolved, chronic oral therapy with furosemide (1–2 mg/kg Q 12 H) is initiated. As disease progresses, many dogs require escalating doses of furosemide to suppress signs of congestion and total doses of 6 to 8 mg/kg Q 24 H are not uncommon.

CONSIDERATIONS

In experimental studies, dogs have rapidly become resistant to chronic furosemide. In clinical patients, high doses of furosemide are often associated with diminishing polyuria and polydipsia and recurrent episodes of heart failure. In these cases, additional diuretics (hydrochlorothiazide or a hydrochlorothiazide–spironolactone combination, 0.5–1 mg/kg Q 24–48 H) help restore diuretic response. In most cases, the additional diuretics are added to the existing furosemide regimen.

In dogs with advanced disease, and particularly in cases of right heart failure, bioavailability of oral medications is likely reduced. Daily subcutaneous injections of furosemide can be used in place of oral administration and are often associated with restoration of copious diuresis.

MONITORING

In all instances of diuretic use, renal [illegible] should be closely monitored.

ACE INHIBITORS

INDICATIONS

[illegible] associated with activation of the renin–angiotensin–aldosterone system (RAAS), which promotes fluid retention, vasoconstriction, and myocardial and vascular remodeling. Use of ACE inhibitors in dogs with CHF is associated with improved quality of life and survival; however, data in support of this statement are less robust for dogs with DCM than for dogs with DMVD. When diuretics such as furosemide are administered, the reduction of plasma volume further stimulates RAAS activity and coadministration with an ACE inhibitor is generally recommended.

ADMINISTRATION

There are a variety of ACE inhibitors available, including enalapril, benazepril, ramapril, and lisinopril (**Table**). Differences are relatively minor, mainly involving route of metabolism/excretion and lipophilicity. From a clinical standpoint, many cardiologists consider them to be interchangeable. In the United States, the two most commonly used ACE inhibitors are enalapril and benazepril; both are associated with clinical benefit in dogs with signs of heart failure.

CONSIDERATIONS

In dogs with DMVD, the use of ACE inhibitors in those without clinical signs remains controversial. Two well-designed studies offer slightly different perspectives. One study in Cavalier King Charles spaniels with mild–moderate DMVD clearly indicated that enalapril did not delay onset of CHF. Another study involving dogs of many different breeds and more advanced DMVD also failed to show benefit with respect to the study's primary endpoint; however, analysis of several secondary endpoints suggested that dogs that received enalapril avoided heart failure longer than dogs that did not.

In my opinion, if ACE inhibitors delay heart failure in dogs with DMVD that show no clinical signs, the effect is inconsistent from individual to individual, relatively small, and unlikely to dramatically change progression of disease. In dogs with severe heart enlargement and at high risk for CHF, I prefer to use an ACE inhibitor in tandem with low-dose diuretic therapy (furosemide, 1 mg/kg Q 24 H), as this more likely reduces plasma volume, intracardiac pressure, and risk of CHF than using an ACE inhibitor alone.

In human patients with asymptomatic DCM, early use of ACE inhibitors is widely recommended. In veterinary medicine, large-scale trials are lacking; however, a small study indicated that ACE inhibitors delayed onset of heart failure in Doberman pinschers with DCM. Thus, in dogs with DCM, I recommend use of ACE inhibitors prior to onset of clinical signs.

MONITORING

Adverse effects of ACE inhibitor treatment are relatively rare, but clinically significant renal dysfunction can occur. Less commonly, systemic hypotension or electrolyte imbalances are encountered. Renal function should be evaluated both before and after initiation of ACE inhibitors and at 3-[illegible]

PIMOBENDAN

INDICATIONS

[illegible] associated with progressive loss of myocardial contractility. Poor contractility is much easier to detect in dogs with DCM as opposed to DMVD, where the presence of a large degree of mitral regurgitation often confounds routine echocardiographic evaluation of contractility.

Pimobendan is a positive inotrope and increases contractility through a mechanism different from that of traditional inotropes such as digoxin—the advantage of which is increased contractility without significant increases in myocardial oxygen demand. Pimobendan also relaxes vascular smooth muscle and elicits modest arterial vasodilation; this dual "inodilating" action is unique. Pimobendan improves survival and quality of life in dogs with DMVD, and very likely does the same in dogs with DCM.

ADMINISTRATION

The recommended dose is 0.5 mg/kg per day, divided into 2 doses that do not necessarily need to be equal.

CONSIDERATIONS

The benefits of pimobendan have been substantiated in dogs showing clinical signs associated with heart disease due to DMVD and DCM; treatment with this agent is recommended only if clinical signs are evident. Thus, in the majority of instances, pimobendan is prescribed only if and when dogs experience congestive heart failure and its attendant clinical manifestations (eg, cough, dyspnea, tachypnea).

Less commonly, dogs with exercise intolerance or syncope are also candidates for treatment. Currently, no evidence exists for the use of pimobendan in dogs with DMVD or DCM prior to the onset of clinical signs.

MONITORING

Pimobendan is generally well tolerated in dogs and no specific monitoring recommendations accompany its use.

continues

Table.
Commonly Used ACE Inhibitors in Dogs with DMVD & DCM

Drug	Dose	Tablet Sizes
Benazepril	0.25–0.5 mg/kg Q 24 H	5, 10, 20, 40 (mg)
Enalapril	0.5 mg/kg Q 12–24 H	2.5, 5, 10, 20 (mg)
Lisinopril	0.5 mg/kg Q 24 H	2, 5, 10, 20, 40 (mg)
Ramipril	0.125 mg/kg Q 24 H	1.25, 2.5, 5 (mg)

ACE = angiotensin-converting enzyme; CHF = congestive heart failure; DMVD = degenerative mitral valve disease; DCM = dilated cardiomyopathy; RAAS = renin–angiotensin–aldosterone system

SPIRONOLACTONE

SPIRONOLACTONE INDICATIONS

Spironolactone is a relatively weak potassium-sparing diuretic, and its potency prevents its use as the sole diuretic agent in dogs with CHF. The value of spironolactone most likely lies in its action as a specific aldosterone antagonist. Aldosterone is a part of the aforementioned RAAS and promotes fluid retention and vascular and myocardial remodeling. Interestingly, in both humans and dogs with heart disease, aldosterone can be elevated despite the use of ACE inhibitors. Suppression of this "aldosterone escape" by spironolactone is associated with reduced morbidity and improved survival in dogs with DMVD.

ADMINISTRATION

In dogs with signs of heart failure, spironolactone (1–2 mg/kg Q 12 H) is recommended in addition to furosemide, ACE inhibitors, and pimobendan. In Europe, where spironolactone is specifically approved for use in dogs with DMVD, the recommended dose is 2 mg/kg Q 24 H. In the United States, many cardiologists use a lower dose of spironolactone (0.5–1 mg/kg day) or spironolactone coupled with a thiazide diuretic such as hydrochlorothiazide.

MONITORING

Adverse effects can include hyperkalemia (especially in the face of concurrent ACE inhibitor) and azotemia, and routine renal and electrolyte monitoring is recommended.

Treatment Schemes: Degenerative Mitral Valve Disease & Dilated Cardiomyopathy

Treatment scheme for dogs with DMVD as they progress from mild to severe disease (without clinical signs) to CHF and finally to advanced refractory disease. Recommendations in green are supported by veterinary trials; recommendations in orange are advocated by the author.

Treatment scheme for dogs with DCM as they progress from mild to severe disease (without clinical signs) to CHF and finally to advanced refractory disease. Recommendations in green are supported by veterinary trials; recommendations in orange are advocated by the author.

Canine Cardiac Drugs Approved by the FDA

In the United States extralabel use of animal and human drugs is permitted in non–food-producing animal practice except when public health is threatened. However, it should be noted that products approved by the FDA for a particular species and use have gone through a rigorous review process.

Benazepril	Not in U.S.; approved in Europe & Canada
Enalapril	Enacard (merial.com)
Furosemide	Lasix (intervet.com); Disal (boehringer-ingelheim.com)
Hydrochlorothiazide	Only approved for use in cattle (Hydrozide; merial.com)
Lisinopril	Not in U.S.
Pimobendan	Vetmedin (boehringer-ingelheim.com)
Ramapril	Not in U.S.; approved in Europe
Spironolactone	Not in U.S.; approved in Europe

BETA-BLOCKERS

BETA-BLOCKERS INDICATIONS

Sympathetic tone is chronically elevated in dogs with DMVD and DCM and is thought to contribute to disease progression. In humans, plasma norepinephrine is a powerful predictor of morbidity and mortality. However, routine use of beta-blockers in veterinary medicine is hindered by lack of well-controlled clinical trials and risk for adverse events when initiating therapy, especially in dogs with advanced disease. In humans, beta-blockade is recommended in virtually all instances of reduced contractility, such as occurs in DCM. Thus, administration of beta-blockers is advocated by many cardiologists in dogs with DCM.

ADMINSTRATION

Because of the risk for acute slowing of heart rate and decreases in contractility, treatment with beta-blockers must be performed with caution. Typically, the dose is up-titrated over 4 to 6 weeks with close monitoring of heart rate, respiratory effort, and blood pressure. Titration is best tolerated in dogs with relatively early DCM.

CONSIDERATIONS

In dogs with DMVD, the use of beta-blockers is controversial and no consensus recommendations can be made.

Take Note

Drug doses listed in this article are guidelines for the "typical" dog with heart failure. They may need to be adjusted based on the patient's severity of signs, renal function, concurrent disease, and response to initial treatment. When prescribing medications to dogs with congestive heart failure due to DMVD or DCM, there is broad consensus that combination therapy with furosemide, an ACE inhibitor, and pimobendan is beneficial.

MONITORING

Practitioners who use beta-blockers must be prepared to monitor dogs closely and to deal with acute decompensation should it occur. Consultation with a cardiologist is recommended.

See Aids & Resources, back page, for references, contacts, and appendices.

ACE = angiotensin-converting enzyme; CHF = congestive heart failure; DMVD = degenerative mitral valve disease; DCM = dilated cardiomyopathy; RAAS = renin–angiotensin–aldosterone system

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