PREMATURE NEONATE WITH OLIGURIC ACUTE RENAL FAILURE AND ASPHYXIATED HYPOXIC-ISCHEMIC ENCEPHALOPATHY

NEONATO PREMATURO, EN FALLO RENAL AGUDO OLIGÚRICO CON ENCEFALOPATIA ASFÍXICA-HIPÓXICA-ISQUÉMICA

How often have I said to you that when you have eliminated the impossible, whatever remains, however improbable, must be the truth

Sherlock Holmes

A fictional detective created by Scottish author and physician Sir Arthur Conan Doyle

¡Cuántas veces te he dicho que cuando se ha eliminado lo imposible, lo que queda, por improbable que parezca, debe ser la verdad

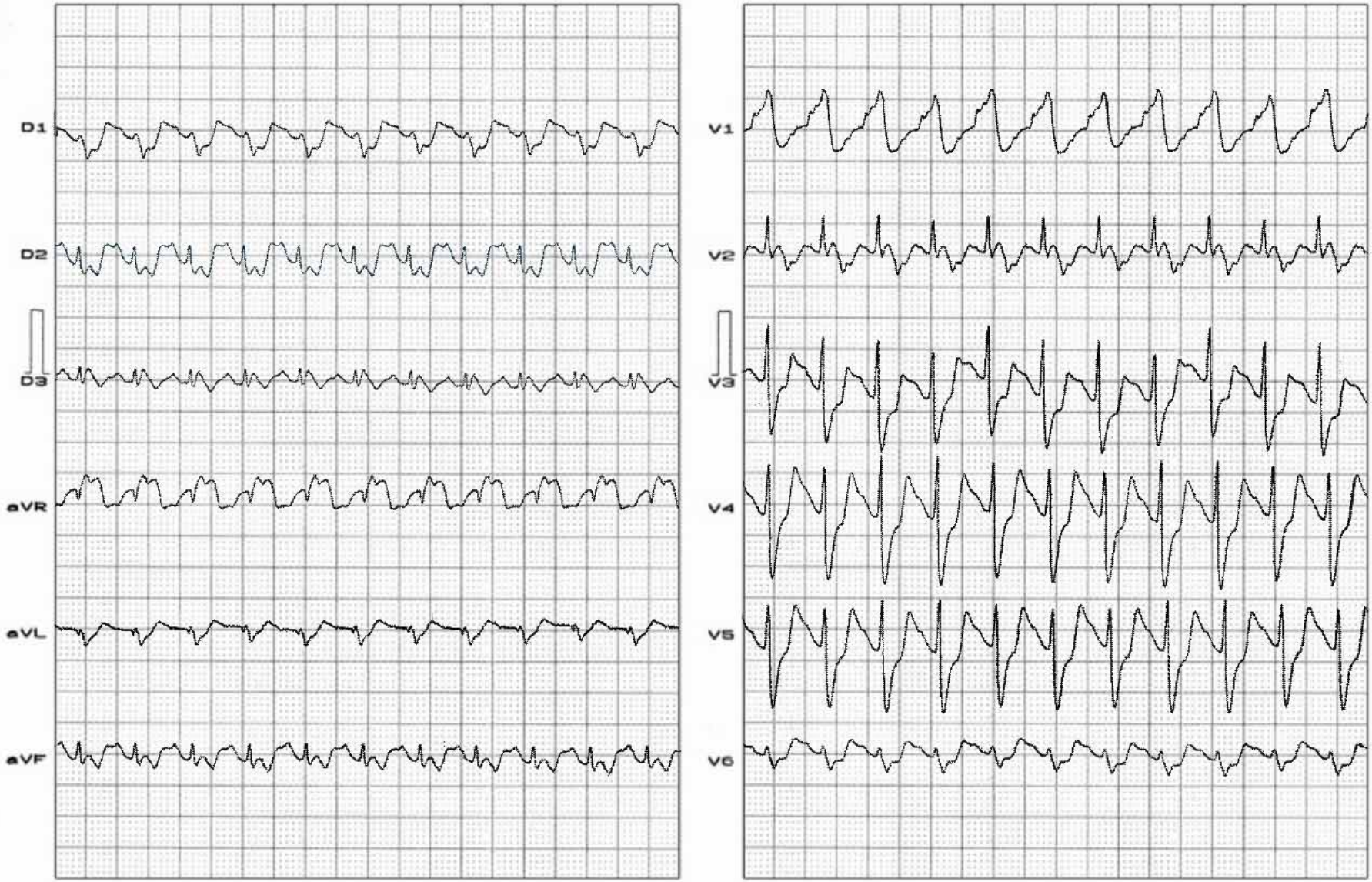
By Andrés Ricardo Pérez-Riera M.D. PhD.

Premature newborn with acute renal failure following perinatal severe birth asphyxiated hypoxic-ischemic encephalopathy.
Fourth day of his life Interned in our intensive care Unit.
Infant lethargic, mild hypotonic, absent suck, weak Moro, miosis and focal seizures.
Congenital Patent Ductus Arteriosus treated with indomethacin (nonsteroidal anti-inflammatory) to close. Less than 48 hours first indomethacin dose: 0.2 mg/kg IV before 48h. Second dose: 0.1 mg/kg IV. Third dose: 0.1 mg/kg IV. Doses was given at 12 to 24 hour intervals. Persisted oliguric renal failure (urine output of 0.34ml/kg/h), blood urea of 80 mg/dl, creatinine more than 2,5 mg/dl. Apgar score 4 done five minutes after birth, and repeated later because the score remained low.
Normal maternal renal function.

Which is the ECG diagnosis? Which is the underlying cause?

Recién nacido prematuro con enefalopatia. asfixica hipóxico-isquémica cuarto día de nacido con insuficiencia renal aguda oligúrica después severa asfixia perinatal al nacer.
Internado en nuestra unidad de cuidados intensivos.
Lactante hipotónico, leve letárgia, mamada ausente, Moro débil, miosis y crisis focales.
Portador de ductus arterioso persistente tratado con indometacina (anti-inflamatório no hormonal). la primera dosis de indometacina administrada con menos de 48 horas 0,2 mg / kg IV . Segunda dosis: 0,1 mg / kg IV. Tercera dosis: 0,1 mg / kg IV. Las dosis se le dio a intervalos de 12 a de 24 horas. Persistía la insuficiencia renal oligúrica (diuresis de 0.34ml/kg/h), urea en sangre de 80 mg / dl, creatinina de más de 2,5 mg / dl. Apgar 4 puntuacion calculado cinco minutos después del nacimiento, y repitido más tarde, con el resultado que sigue siendo bajo. Función renal materna normal.

¿Cuál es el diagnóstico de ECG? ¿Cual es la causa subyacente?


D1
D2
D3
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

Queridos amigos: Trataré de analizar el caso del recién nacido enviado por nuestro maestro Profesor Andrés Ricardo Pérez-Riera.
Este es un aleteo auricular con frecuencia auricular de 500 lpm y bloqueo del nódulo auriculo ventricular 2/1. Esta frecuencia tan rapida (similar a un ratón) se debe a que los canales de salida tardia de potasio "rectifier delayed potassium channel no se desarrollaron todavia, permitiendo una frecuencia muy elevada.
El bloqueo de rama derecha muy ancho para esta edad podria ser consecuencia de una cardiomiopatia dilatada pero no estoy muy seguro.
Segun mi experiencia un recién nacido (y hasta 6 meses) (puede conducir hasta 250 latidos sin bloqueo intraventriculares.
El patrón de V1 pude ser que indique una patologia cardiaca.
Un fraternal abrazo
Samuel Sclarovsky

Dear friends I will treat to analyze the case of newborn sent by our teacher Professor Andres Perez Riera This is an atrial flutter with frequency of 500bpm associated to AV block 2:1.
This often so fast like a mouse is consequence of immaturity of potassium delayed rectifier channels not yet developed, allowing a very high frequency.
The right bundle branch block too wide for this age could be due to a dilated cardiomyopathy but I'm not sure.
In my experience a newborn (and up to 6 months) can to conduce until 250 bpm without intraventricular block.
V1 pattern could be indicating of cardiac pathology.
A fraternal hug

Samuel Sclarovsky

el lead, enves de V1 debe ser DI, Le quisiera pedir informacion de la velocidad aricular del flutter En la auricula izquierda existe un canal de potasio que se denomina" hyper fast" , que hay investigadores que dicen que este canal existe ya al nacimiento, y esto puede ser posible desde el punto de vista evolutivo, ya que el recien nacido necesita trecuencias muy altas para mantener el cardiac output,ya que en la vida fetal el ventriculo izquierdo no tiene funcion hemodinamica.,Y verdaderamente a los primeros latidos , hay una cantidad de genes ,receptores y subtancias estimulantes que deben producir hipertrofia fisilogica , disminuir la presion de la circulasion pulmonar , cerrar el ductus ,absorver, el liquido pulmonar La produccion de proteinas contractiles es muy lenta ,y por eso la taquicardia escritica Para esto madre natura ,desarrollo los"rectifier
delay potassium channels" despues de los 6-7 meses de vida , para evitar que los canales que determinan el periodo refractario no molesten depolarizar el miocardio a alta frecuencia(esto se demostro en ratones que desarrollan velocidades sinusales en reposo hast 700 lpm , y en esfuerzo hsta 1300 lpm ,sin transtornos de conduccion , y como es bien sabido que el potencial del raton, es de 50ms, y carece de la fase 2 terminal y fase 3 , Lo unico que posee es fase uno ,que es determinado por la salida y desaparicion rapida
En fin este electros muestran los secretos de la vida , que se puede leer en simple y viejo metodo yo como siempre comodice:" Cacho de BS AS por esta .... costumbre" de filosofar ante un simple ecg. pero me parece que esto es de cardiologo viejo y un poco hicha p...Tambien quisiera saber quien el cardiologo que escribia este email, y donde trabaja Mi personal curiosidad es saber quien son las personas que intervienen en este majestuoso e impresidible forum un fraternal abrazo samuel sclarovsky.

Muy interesante ECG.
Me faltan algunos datos clínicos de importancia como ser la edad gestacional al nacimiento? dato importante en la evaluacion del ECG asi como los datos del ecocardiograma actual: el ductus esta cerrado?,
Como es la función ventricular?,
y el pericardio?.
De todos modos creo se trata de una taquicardia-flutter auricular con QRS disminuídos de amplitud en derivaciones de miembros, bloqueo de la rama derecha del haz de His, descenso del punto J, descenso del intervalo ST, intervalo QTc normal.

Saludos a todos

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Respuesta:
Es un recién nascido pré-término de 35 semanas y de bajo peso 2350Kg Ductus permance habierto. Funcion ventricular izquierda adecuada y sin problemas pericárdicos.
Andrés.

……
Nosotros hemos acompañado hasta la fecha en el servicio 6 casos de Flutter neonatal, todos con corazon sano y sin comorbilidades, ninguno tenia QRS ancho y las ondas P eran de morfologias totalmente diferentes a este caso, pienso que el QRS tan ancho sea por algun trastorno hidroelectrolitico, esta en IRA, respeto la opinión del Dr Samuel por ser un gran maestro, simplemente les estoy trasmitiendo mi pequeña experiencia de haber acompañado 6 neonatos con flutter en otro escenario clinico, este tiene ductus+IRA .
CARLOS RODRÍGUEZ ARTUZA

Maestro Pérez-Riera el ECG muestra datos generalizados de lesión e isquemia secundarias a la hipoxia severa de este pequeño. Se cataloga como cardiopatia anoxo-isquémica. Igualmente otros órganos se afectan (neurologico, renal, etc.)
Se titularon enzimas cardiacas ?

JRMC

Estimado JRMC me inmagino que estas queriendo decir que este prematuro padece del llamado síndrome hipóxico-isquémico (Hypoxic-ischemic syndrome) ou Hypoxic ischemic encephalopathy (HIE) Este es un terminos usado para describir una variedad reacciones externas o internas en neonatos.

Meu caro Andrés:
Embora o nível de potássio sérico não tenha sido comunicado, a meu ver trata-se de um caso de hiperpotassemia e o traçado mostra ausência de ondas P e um QRS bizarro. Minha hipótese: devido à hiperpotassemia, ocorre um bloqueio sino-atrial completo (o que explica a ausência de inscrição de ondas P no traçado) e a condução é sino-ventricular, isto é o estímulo originado nao nível do nó sinusal se conduz através do feixe internodal posterior ou feixe de Thorel e, através das fibras de James, atinge o sistema de condução abaixo do nó A-V. A condução intraventricular se faz de fibra a fibra o que explica o aspecto bizarro dos complexos QRS.
Um abraço do
Hélio Germiniani MD PhD Curitiba Brazil

Congratulation teacher you are the best!!!!!
Your endless pupil
Andres,

Our diagnosis

“*It seems, from what I gather, to be one of those simple cases which are so extremely difficult”*

Sherlock Holmes.

ECG diagnosis

Rhythm: Sino ventricular. Absence of P wave. The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded).

Heart rate: 166bpm. In premature babies some authors admit up to 210 bpm may be normal. Mean HR in premature 125 bpm. Inferior limit for normality: 100 bpm in the day and 80 bpm during sleep. Possible relative bradycardia between 70 bpm and 80 bpm may be found. In preterm infants, the HR rose more slowly than term infants. The median HR is <100 bpm at 1 min after birth. After 2 min it is uncommon to have a HR <100 bpm. In preterm infants and those born by caesarean section the HR rose more slowly than term vaginal births(1).

Severe hyperkalemia:Note ST segment depression and SR elevation observed, known as “dialyzable injury current” and the almost vertical upstroke of T waves in V_3-V_5, one of the patterns of severe hyperkalemia Potassium level = 7.9 mEq/L

Clinical diagnosis

1. Perinatal hypoxic-ischemic encephalopathy in acute period
2. CNS tissue damage
3. Persisted oliguric renal failure
4. Congenital Patent Ductus Arteriosus

1.Dawson JA, Kamlin CO, Wong C, et al. Changes in heart rate in the first minutes after birth. Arch Dis Child Fetal Neonatal Ed. 2010 May;95(3):F177-81.

ECG IN PREMATURE BABIES

RHYTHM: Phasic or respiratory sinus arrhythmia is the rule. High incidence of junctional escapes is observed.

P WAVE: SâP: between 0º & 90º; Pduration: 60ms ± 2 ms. P voltage: maximal limit accepted is 2.5 mm (other authors admit 3 mm). P Polarity: always positive in II, I & aVR, and from V3 to V6, always negative in aVR, variable in II, aVL, V1 & V2. In the two last it may be "plus-minus".

In premature babies polarity inversion by junctional rhythm or of coronary sinus may be frequent.

PR interval: discretely shorter than newborn babies born full-term. In average 90 ms (0.09 s).

QRS axis (SAQRS) in the frontal & horizontal planes:

FP: +85º in average. **HP:** in left anterior quadrant. In full-term newborn babies more to the right.

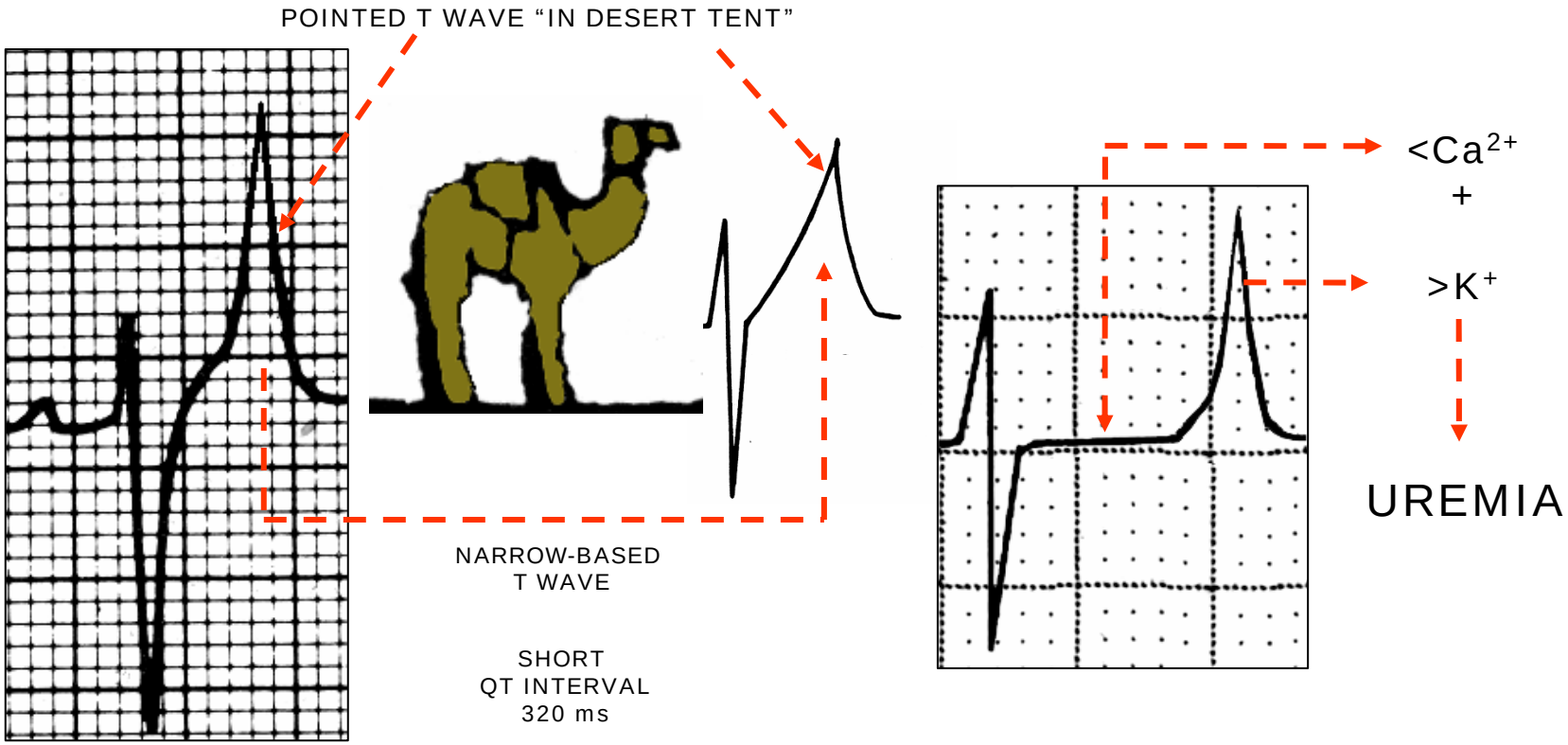
Duration of QRS: discretely smaller. In average 40 ms (0.04 s), but it could reach up to 70 ms as maximal limit.

Voltage of QRS: low voltage in bipolar leads of the limbs, and after 30 days, voltage increases in left precordial leads, which may be confused with LVE.

QRS morphologies in different leads: It reveals less predominance of the RV. Thus, up to 31 weeks the weight of the left ventricle is greater than that of the right one; by the 33rd week of gestation the LV weight/RV weight ratio is = 1/1, and in full-term newborn babies this ratio is 0.8/1<u>1</u>. This lower right predominance (or absence) is translated in ECG in premature babies by:

More incidence of deep Q waves in left precordial leads indicating a good manifestation of left potentials. Frequent complexes of the qR type in V6. The normal maximal limit of Q wave duration is 20 ms (0.02 s or ½ small square) and its normal maximal depth is 8 mm. In newborn babies, there should never be Q waves in I and aVL. Their presence suggests anomalous origin of the left coronary artery from the pulmonary artery or Bland-White-Garland syndrome (it is present in more than 80% of the cases). In this entity the depth of the Q wave in I is usually greater than 50% of voltage of the subsequent R wave.

In the HP, unlike full-term newborn babies, the loop has a counterclockwise rotation and is located predominantly to the left.
In the FP AQRS is found to the left of +90º while in full-term newborn babies to the right of +90º.
Maximal vector around +80º and in the frontal plane.
T loop in the HP is heading backwards and to the left.

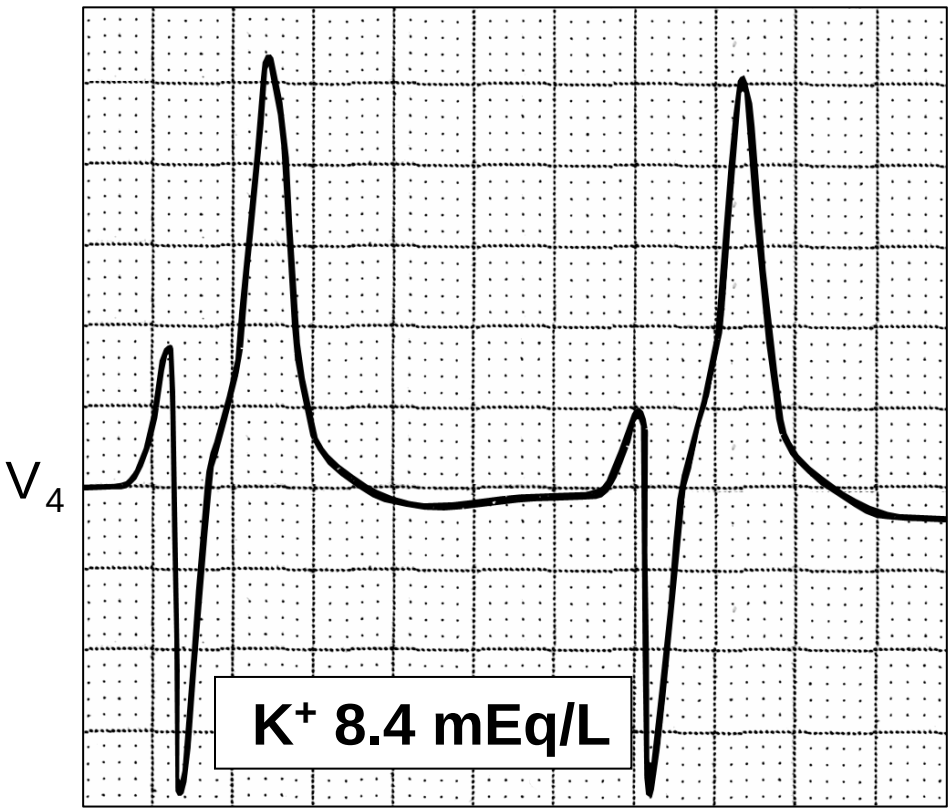


ECG MANIFESTATIONS IN HYPERPOTASEMIA AND RELATIONSHIP TO CATION SERUM LEVEL.
Even with characteristic modifications being described and correlated to potassium serum level, several ECG patterns may occur, and not always with these characteristics.
Normal referential value of serum potassium: 3.5 to 5 mEq/L.

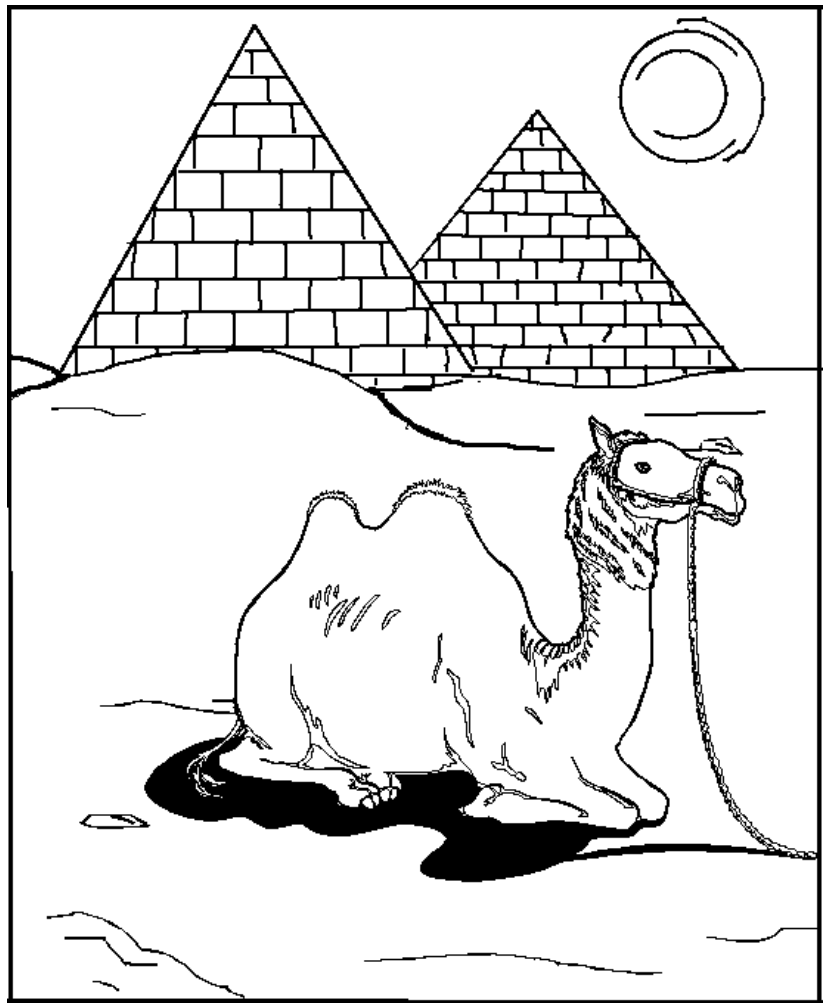
SERUM POTASSIUM: 5.7 mEq/l
Pointed T waves, with increased voltage, symmetrical and narrow based: “desert tent” wave, short QTc interval and ST segment may be depressed.

Modifications in the electrocardiogram with slightly increased serum potassium levels (above 5.7 mEq/L): T wave in "tent" with a narrow base and with short QT interval. The outline additionally shows ECG modifications when hypocalemia is associated to hyperpotasemia.

POINTED T WAVE OF
HYPERPOTASEMIA
IN “DESERT TENT”

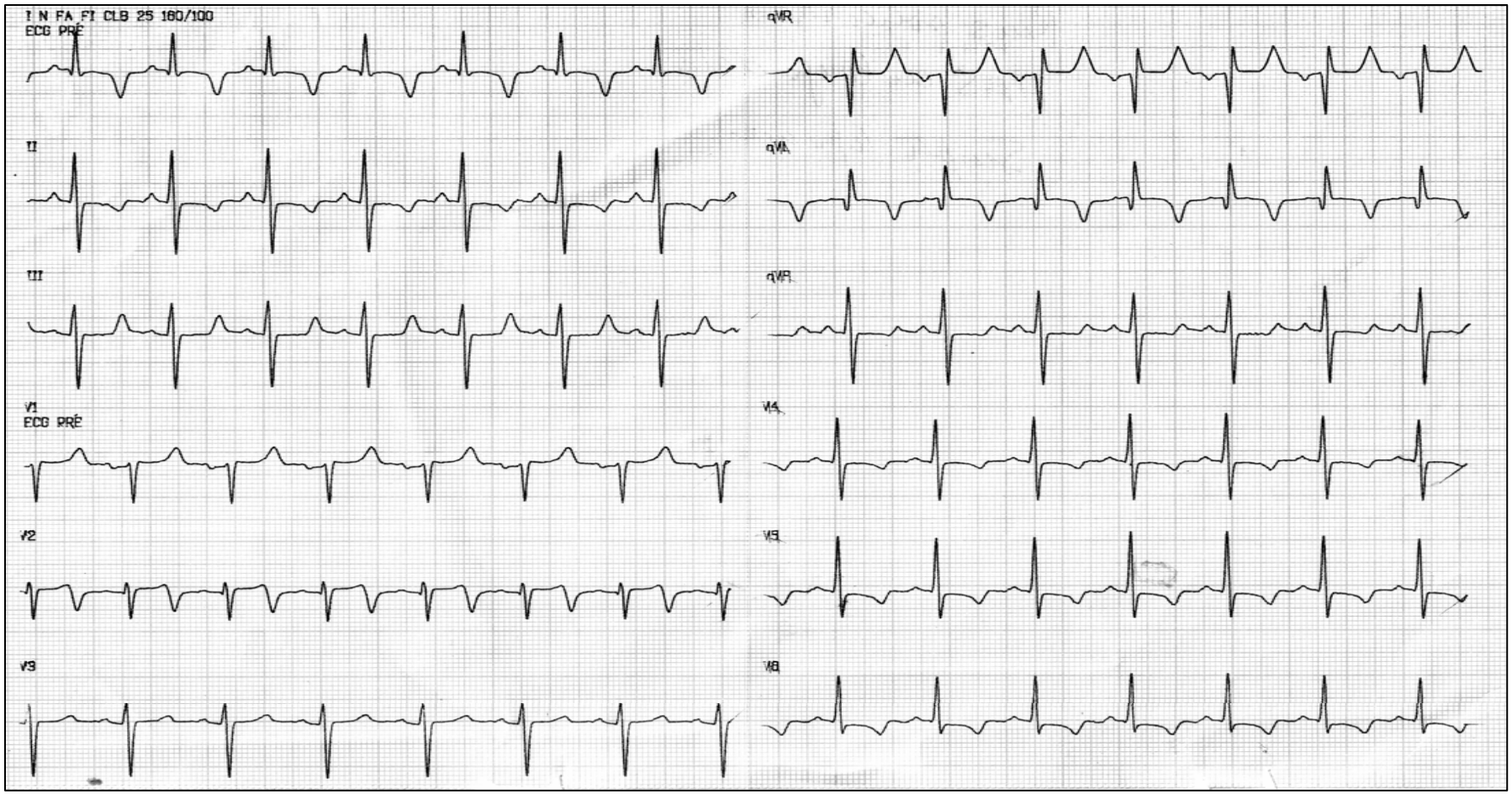


WITH GREAT VOLTAGE,
SYMMETRICAL AND NARROW BASED



Typical example of T wave in "desert tent" characteristic of great-voltage and narrow-based hyperpotasemia.

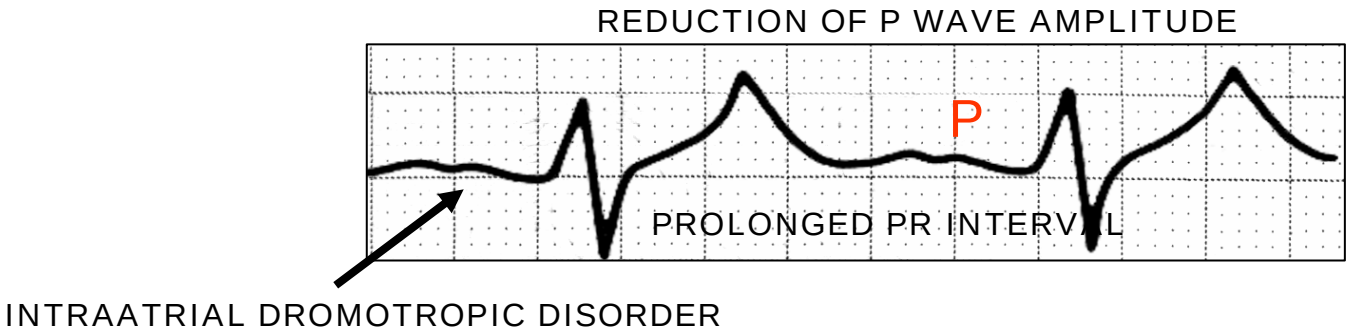
HYPERPOTASEMIA ASSOCIATED TO HYPOCALCEMIA



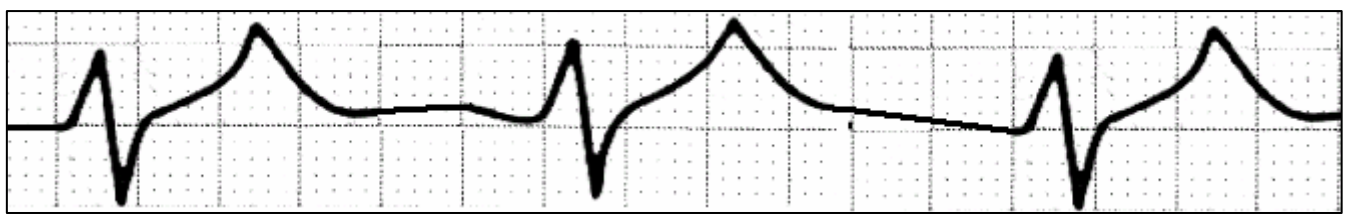
Male patient, 46 years old, hypertensive, carrier of chronic renal insufficiency in dialytic phase by polycystic kidney (creatinine 7.0 mg/%), high serum potassium and low calcium.

ECG of hypertensive, chronic renal patient with high serum potassium and low calcium.

SERUM POTASSIUM: 7 mEq/l



SERUM POTASSIUM: 8.4 mEq/l



SINOVENTRICULAR RHYTHM: absence of P wave. The stimulus originates in the SA node, it is conducted to the AV node through internodal bundles and reaches the junction without depolarizing the atrial muscle (P wave is not recorded).

ECG characteristics with serum potassium values around 7 mEq/L and 8.4 mEq/L.

SERUM POTASSIUM FROM 8 TO 9 mEq/l

QRS IS DESCRIBED:

A) Decrease of R wave voltage;

B) Prominent S waves;

C) Diffuse QRS complexes widening, similar to left or right bundle branch block, associated to anterior or posterior fascicular block by extreme shift of SAQRS in the FP to left or right. This QRS complex widening is differentiated of genuine branch blocks, because in them, the delay is final or middle, while in hyperpotasemia is always global or diffuse.

Characteristics of ECG with serum potassium values between mEq/L and 9 mEq/L.

SERUM POTASSIUM FROM 8 TO 9 mEq/l

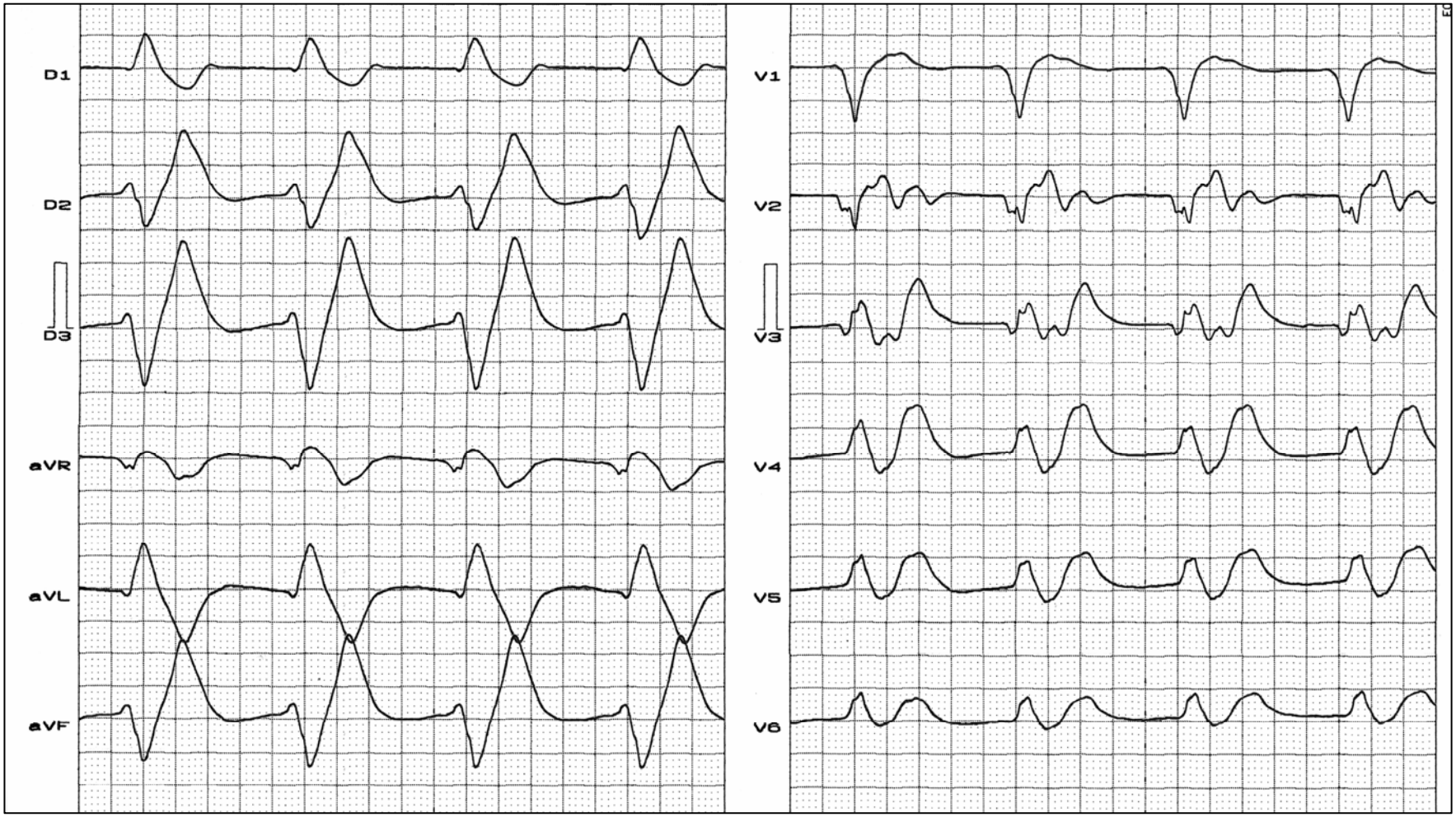
IN BRIEF:

RBBB: final delay.
LBBB: middle-final delay.
Hyperpotasemia: global delay.
In the late phase, a possible convergence of the QRS complex with the T wave is described, outlining a smooth diphasic wave or sine curve associated to concomitant QT interval prolongation.

ST segment depression or elevation may be observed, known as “dialyzable injury current” that may possibly resemble electrocardiographic Brugada-like pattern.

Rare cases are described, which resemble acute antero-septal infarction by absence of R wave from V_1 to V_4 associated to ST segment elevation of the subepicardial injury current.

Characteristics of ECG with serum potassium values between mEq/L and 9 mEq/L.



Clinical diagnosis: chronic renal insufficiency in dialysis. The patient delayed 72 hours the dialysis session. Hyperpotasemia of 9 mEq/L.

ECG diagnosis: absence of P wave, sinoventricular rhythm, 57 bpm, morphology of bizarre intraventricular severe disorder (QRSd: 240 ms) that is similar to complete left bundle branch block. T waves with polarity matching with QRS from V3 to V6. Convergence of QRS with T wave that outlines smooth diphasic wave or sine curve.

Typical example of ECG of patient with extremely high level of serum potassium.

HYPERPOTASEMIA ASSOCIATED TO ELECTROCARDIOGRAPHIC BRUGADA-LIKE PATTERN OR BRUGADA PHENOCOPY(1;2)

The so-called Propofol Infusion Syndrome or PRIS is characterized by the association of:
1) Hyperpotasemia;
2) Heart failure[3];
3) Sudden cardiac death;
4) Metabolic acidosis;
5) Rhabdomyolysis;
This entity is observed when the drug is administered in high doses, in rates > 5 mg/kg/hr during a period > 2 days. The average of the used dose for PRIS appearance was 6.5 mg/kg/hr.
Propofol (DiprivanR) is an endovenous sedative-hypnotic anesthetic agent with short duration of action, used to induce anesthesia in children older than 3 years old, and to maintain general anesthesia in adults and > 2 months. The drug is used as general anesthetic agent and to reduce encephalus injury pressure.

1) Riera AR, Uchida AH, Schapachnik E, et al. Propofol infusion syndrome and Brugada syndrome electrocardiographic phenocopy. Cardiol J 2010; 17: 130-135.
2) Nguyen T, Smythe J, Baranchuk A. Rhabdomyoma of the Interventricular Septum Presenting as a Brugada Phenocopy. Cardiol Young 2011 2011 May 4:1-4.
3) Cremer OL, et al. The Lancet 2001;357:117-118.

Characterization of the so-called "Propofol Infusion Syndrome".

HYPERPOTASEMIA ASSOCIATED TO ELECTROCARDIOGRAPHIC BRUGADA-LIKE PATTERN OR BRUGADA PHENOCOPY

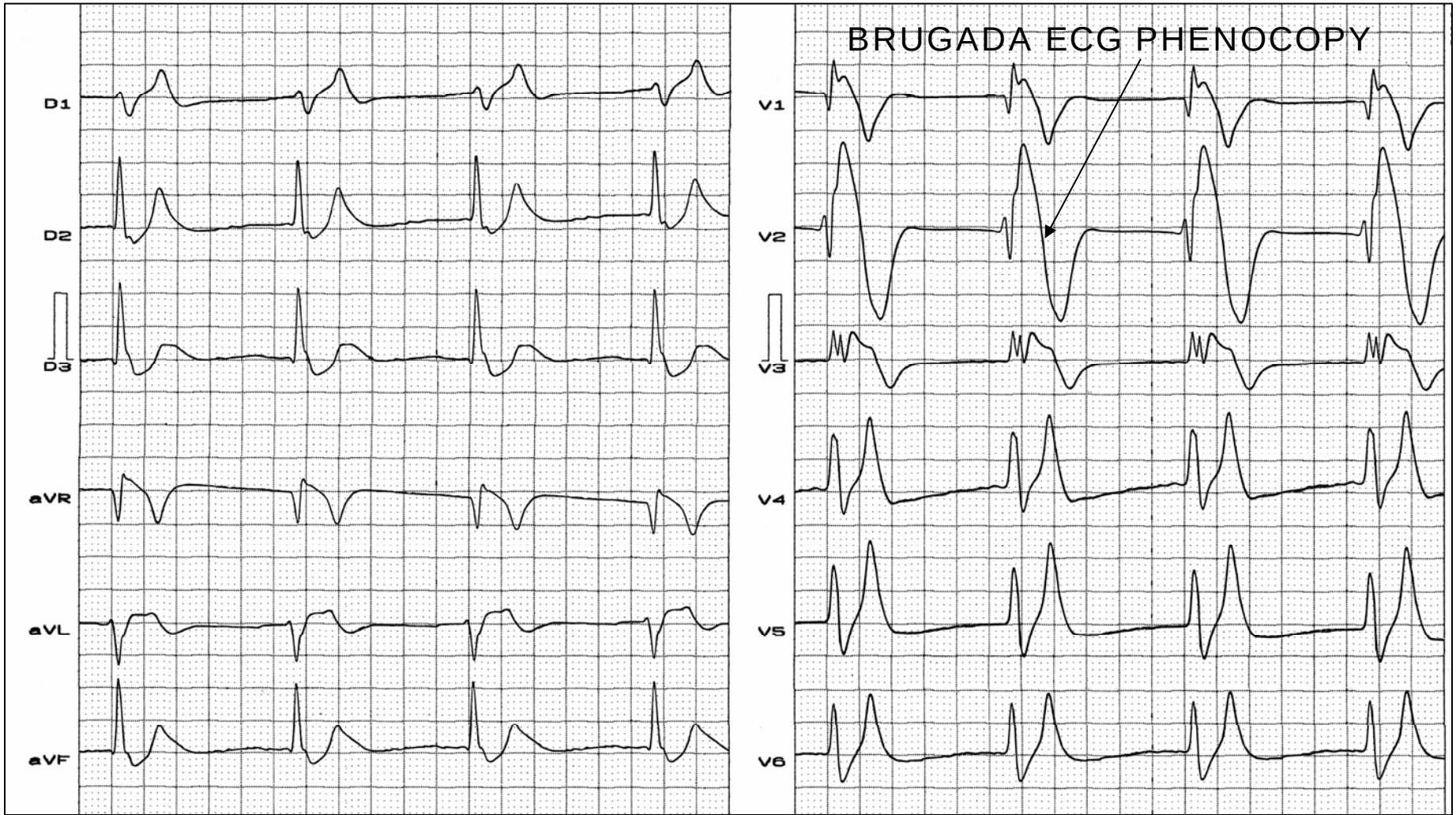
As a consequence of infusion in high doses of propofol from the point of view of ECG, the features are ECG with upwardly convex elevation from V1 to V3 (Brugada-like ECG pattern). The development of this acquired form of Brugada-like syndrome is a sign of electric instability and it is a predictor of imminent sudden death[1].

Severe hypercalcemia may precipitate ECG Brugada-like pattern[2;3;4]. Chronic renal insufficiency has been described transitorily with hypercalcemia and reversed with dialysis[5].

The following ECG shows a case of severe hypercalcemia with electrocardiographic Brugada-like pattern.

1) Vernooy K,et al. Heart Rhythm. 2006; 3:131-137.
2) Grant AO. J Cardiovas Electrophysiol. 2005; 1:3-7.
3) Lopez-Barbeito B, et al. Pacing Clin Electrophysiol 2005;28:730-732.
4) Littmann L. et al.Am Heart J 2003;145:768-778.
5) Ortega-Carnicer J, et al. Resuscitation 2002;55:215-219.

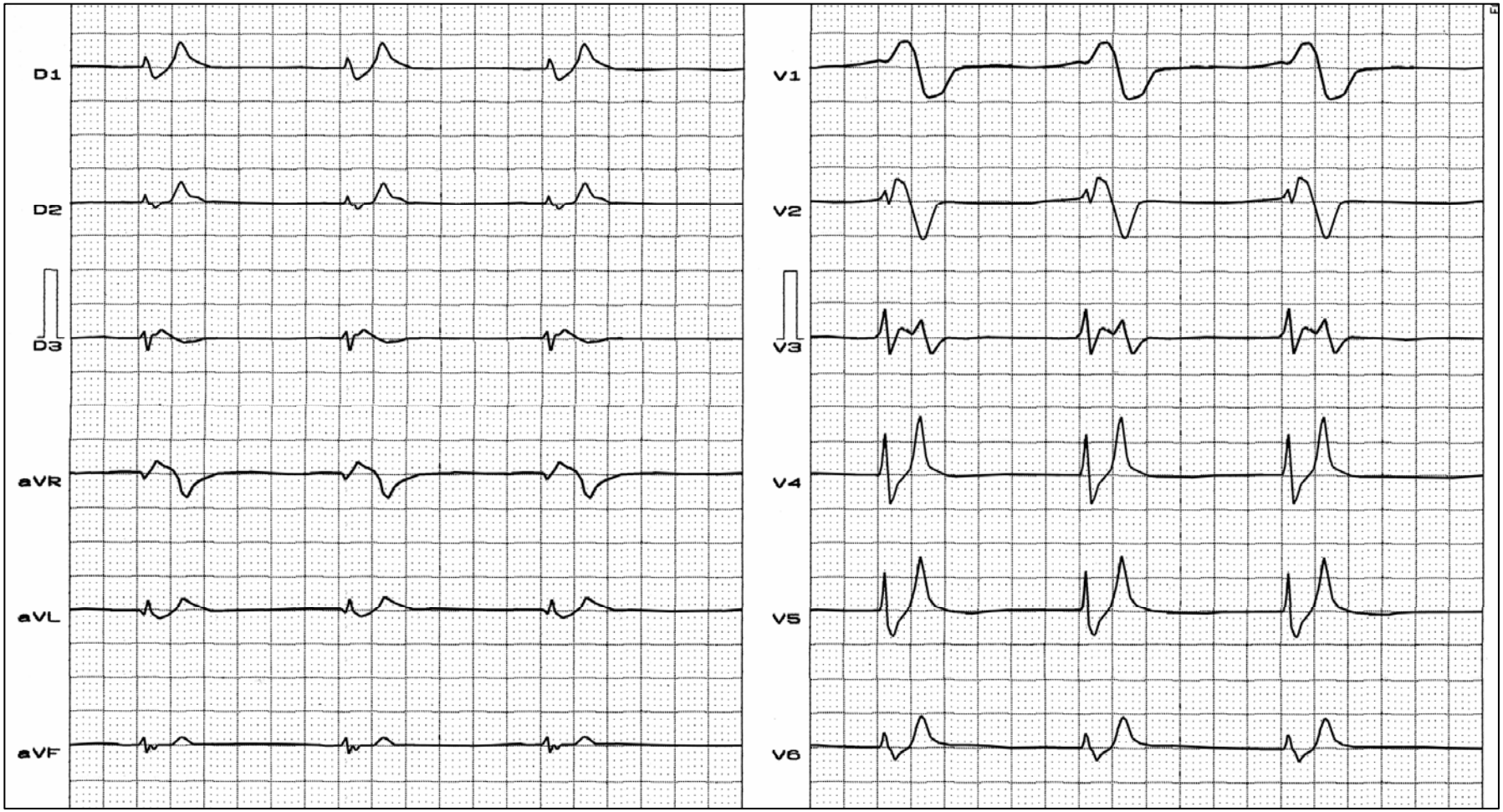
Characterization of hyperpotasemia associated to Brugada-like pattern.



Clinical diagnosis: terminal renal insufficiency. Hypercalemia: K+ 8.7 mEq/L. This sign is known as dialyzable injury current.**ECG diagnosis:** very likely, junctional with P waves near J point, HR: 54 bpm, QRSd: 160 ms, ST segment elevation from V1 to V3 and I, aVL and aVR. V1 to V3 display ST segment upwardly convex pattern, similar to Brugada syndrome, which some authors call “Acquired Brugada Pattern”, typical T waves in “tent”, pointed, and with a narrow base.

Typical electrocardiogram of hyperpotasemia associated to Brugada-like pattern or phenocopy.

Name: FHM; **Sex:** Male; **Age:** 56 y.o.; **Race**: Mulatto;
Weight: 65 Kg; **Height:** 1.62 m; **Date:** 05/11/2003;



P wave is not identified, sinoventricular rhythm. Severe hypercalcemia with electrocardiographic Brugada-like pattern. Chronic renal insufficiency with hypercalcemia has been described transitorily, and reversed with dialysis. Apiculate T waves in "tent" from V4 through V6.

Typical electrocardiogram of hyperpotasemia associated to Brugada-phenocopy.

ARRHYTHMIAS IN HYPERPOTASEMIA

1) SA block, sinus arrest and bradycardia;
2) Active or passive junctional rhythm (escape beats);
3) Mobitz I or II AV block;
4) Advanced AV block;
5) Idioventricular rhythm;
6) Premature ventricular contractions;
7) VT/VF.

Description of the main arrhythmias observed in hyperpotasemia.

Potassium is a major ion of the body. Nearly 98% of potassium is intracellular, with the concentration gradient maintained by the sodium-and potassium-activated adenosine triphosphatase (Na^+/K^+ –ATP_{ase}) pump. Human skeletal muscles contain the largest single pool of K^+ in the body (2600 mmol, 46 times the total K^+ content of the extracellular space).

Intense exercise may double arterial plasma K^+ in one min. This is because of excitation-induced release of K^+ from the working muscle cells via K^+ channels. This **hyperkalemia** is rapidly corrected by reaccumulation of K^+ into the muscle cells via Na^+,K^+ pumps, often leading to hypokalemia. **Hyperkalemia** may also arise from muscle cell damage, excessive oral or intravenous administration of K^+, acidosis, renal failure, depolarization of muscle cells with succinyl choline, activation of K^+ channels by fluoride poisoning, hyperkalemic periodic paralysis, malignant hyperthermia, inhibition of the Na^+,K^+ pumps by digitalis glycosides or treatment with nonselective beta blockers.

Hyperkalemia may cause arrhythmia and can be treated with ß-2 agonists, insulin or hemodialysis. Hypokalemia may be induced by the stimulation of the Na+,K+ pumps in skeletal muscles seen postexercise, or by catecholamines, beta2 agonists, pheochromocytoma, theophylline, caffeine or insulin, by sepsis, myocardial infarction, trauma, burns and heart failure. The ratio of intracellular to extracellular potassium is important in determining the cellular membrane potential. Small changes in the extracellular potassium level can have profound effects on the function of the cardiovascular and neuromuscular systems. The normal potassium level is 3.5-5.0 mEq/L, and total body potassium stores are approximately 50 mEq/kg (3500 mEq in a 70-kg person).Minute-to-minute levels of potassium are controlled by intracellular to extracellular exchange, mostly by the sodium-potassium pump that is controlled by insulin and ß-2 receptors. A balance of GI intake and renal potassium excretion achieves long-term potassium balance.

Hyperkalemia is defined as a potassium level greater than 5.5 mEq/L.(1) Ranges are as follows:
5.5-6.0 mEq/L – Mild; 6.1-7.0 mEq/L – Moderate; 7.0 mEq/L and greater - Severe

1. Tran HA. Extreme hyperkalemia. *South Med J*. Jul 2005;98(7):729-732.

Hyperkalemia results from the following: Decreased or impaired potassium excretion - As observed with acute or chronic renal failure(2) (most common), potassium-sparing diuretics, urinary obstruction, sickle cell disease, Addison disease, and systemic lupus erythematosus. Additions of potassium into extracellular space - As observed with potassium supplements (eg, PO/IV potassium, salt substitutes), rhabdomyolysis, and hemolysis (eg, blood transfusions, burns, tumor lysis). Transmembrane shifts (ie, shifting potassium from the intracellular to extracellular space) - As observed with acidosis and medication effects (eg, acute digitalis toxicity, beta-blockers, succinylcholine)

Factitious or pseudohyperkalemia - As observed with improper blood collection (eg, ischemic blood draw from veni puncture technique), laboratory error, leukocytosis, and thrombocytosis.

Frequency United States Hyperkalemia is diagnosed in up to 8% of hospitalized patients.

Mortality/Morbidity: The primary cause of morbidity and death is potassium's effect on cardiac function.(3) The mortality rate can be as high as 67% if severe hyperkalemia is not treated rapidly.(4)

Sex: The male-to-female ratio is 1:1.

Hyperkalemia can be difficult to diagnose clinically because complaints may be vague. The history is most valuable in identifying conditions that may predispose to hyperkalemia.

Hyperkalemia frequently is discovered as an incidental laboratory finding.

Cardiac and neurologic symptoms predominate.

Patients may be asymptomatic or report the following: Generalized fatigue, weakness, paresthesias, paralysis, palpitations

2) Einhorn LM, Zhan M, Hsu VD, et al. The frequency of hyperkalemia and its significance in chronic kidney disease. *Arch Intern Med*. Jun 22 2009;169:1156-1162.

3) Segura J, Ruilope LM. Hyperkalemia risk and treatment of heart failure. *Heart Fail Clin*. Oct 2008;4:455-464.

4) Weisberg LS. Management of severe hyperkalemia. *Crit Care Med*. Dec 2008;36:3246-3251.

Hyperkalemia is suggested in any patient with a predisposition toward elevated potassium level. Potential potassium level elevation is observed in the following: Acute or chronic renal failure, especially in patients who are on dialysis, trauma, including crush injuries (rhabdomyolysis), or burns, ingestion of foods high in potassium (eg, bananas, oranges, high-protein diets, tomatoes, salt substitutes). This alone is not likely to cause clinically significant hyperkalemia in most people; it is often a contributing factor to an acute potassium elevation, medications - potassium supplements, potassium-sparing diuretics, nonsteroidal anti-inflammatory drugs (NSAIDs),
ß-blockers, digoxin, succinylcholine, and digitalis glycoside, medication combinations (ie, spironolactone, ACE inhibitors)(5), redistribution - metabolic acidosis (diabetic ketoacidosis [DKA]), catabolic states. Evaluation of vital signs is essential to determine hemodynamic stability and presence of cardiac arrhythmias related to the hyperkalemia.(2)
Cardiac examination may reveal extrasystoles, pauses, or bradycardia.
Neurologic examination may reveal diminished deep tendon reflexes or decreased motor strength. In rare cases, muscular paralysis and hypoventilation may be observed.
Search for the stigmata of renal failure, such as edema, skin changes, and dialysis sites.
Look for signs of trauma that could put the patient at risk for rhabdomyolysis.

1. **Schepkens H, Vanholder R, Billiouw JM, Lameire N. Life-threatening hyperkalemia during combined therapy with angiotensin-converting enzyme inhibitors and spironolactone: an analysis of 25 cases. *Am J Med*. Apr 15 2001;110:438-441.**
2. **Tran HA. Extreme hyperkalemia. *South Med J*. Jul 2005;98:729-732.**

Pseudohyperkalemia: hemolysis (in laboratory tube) most common, thrombocytosis, leukocytosis, venipuncture technique (ie, ischemic blood draw from prolonged tourniquet application)

Redistribution: Acidosis, insulin deficiency, ß-blocker drugs, acute digoxin intoxication or overdose, succinylcholine(1), arginine hydrochloride, hyperkalemic familial periodic paralysis
Excessive endogenous potassium load: hemolysis, rhabdomyolysis, internal hemorrhage.

Excessive exogenous potassium load: parenteral administration, excess in diet, potassium supplements, salt substitutes.

Diminished potassium excretion: decreased glomerular filtration rate (eg, acute or end-stage chronic renal failure), decreased mineral corticoid activity, defect in tubular secretion (eg, renal tubular acidosis II and IV), drugs (eg, NSAIDs, cyclosporine, potassium-sparing diuretics).

Laboratory error (2)

1. Gronert GA, Theye RA. Pathophysiology of hyperkalemia induced by succinylcholine. *Anesthesiology*. Jul 1975;43:89-99.
2. Hawkins RC. Poor knowledge and faulty thinking regarding hemolysis and potassium elevation. *Clin Chem Lab Med*. 2005;43(2):216-20.

PERINATAL HYPOXIC-ISCHEMIC ENCEPHALOPATHY (HIE)

Pathogenesis: is noninfectious and thought to result from some degree of asphyxia (decreased oxygen delivery to tissues) during birth. The resulting hypoxia causes varying degrees of CNS tissue damage, depending on the age of the fetus and the severity and duration of the hypoxia. Partial prolonged asphyxia may be associated with the development of cerebral edema, focal hemorrhage, and necrosis. It has been proposed that fetal asphyxia may result in accumulation of toxic concentrations of excitatory neurotransmitters (eg, glutamate, aspartate), which allows intracellular influx of sodium and chloride and passive flow of water, leading to neuronal swelling. The production of oxygen free radicals as well as reperfusion injury may also play a role in the pathogenesis of HIE. Perinatal asphyxia may be strongly suspected in cases of dystocia or generalized placental pathology, but could remain unidentified if in utero placental separation occurs.

Clinical Findings: Many different abnormal behaviors may be grouped under the diagnosis of HIE. Signs range from a slow suckle response at birth to hyperexcitability, aimless wandering, depression, recumbence, generalized hypotonia, and seizures. This may reflect different degrees of asphyxia and CNS pathology.

In the classic syndrome, the foal appears normal at birth and progressively loses interest in its dam, loses its suckle reflex, becomes recumbent, develops clonic seizures, and may start vocalizing. The vocalization has been described as that of a barking dog, hence the term “barker” foal.

Diagnosis: Because the signs of HIE are seen soon after birth, it is often associated with failure of passive transfer of antibodies, which can lead to septicemia. There are no definitive blood chemistry or WBC abnormalities that aid in diagnosis, but these tests are helpful in eliminating other causes of the clinical signs. Other clinical syndromes that can present with similar signs and must be differentiated from HIE include hypoglycemia, electrolyte and acid-base derangements, septic meningitis, head trauma, cerebral bleeding, and congenital CNS defects.

Treatment: The management of HIE is supportive. Providing warmth and nutrition is essential. If the foal does not have a suckle response, an indwelling nasogastric tube should be placed and the foal fed mare's milk or a mare milk substitute at 15-25% of its body weight over each 24-hr period. Lactated Ringer's solution plus 5% dextrose IV will ensure hydration and adequate glucose levels. If the foal did not receive adequate colostrum, a plasma transfusion is indicated. Although HIE is not infectious, foals should receive antibiotics to prevent secondary infections. Seizure control is imperative. Diazepam (0.11-0.44 mg/kg) is usually effective. This dosage can be repeated as needed, but if longterm control is required, phenobarbital (2-10 mg/kg, IV, bid-tid) can be given. Dimethyl sulfoxide (1 g/kg in a 10% solution, IV) may be used as an adjunct to decrease cerebral edema. Mannitol (1 g/kg as a 20% solution, IV) has been proposed for similar reasons, but it should be used with caution due to the common presence of

subdural hemorrhage in these foals. Self-trauma can be a problem during seizures and can be prevented by providing a protected or padded environment and a human holder to cradle the animal. These foals are especially susceptible to eye trauma and corneal ulceration during their seizures; fluorescein staining and subsequent ocular treatment are often important.Prognosis for HIE is fair to good if uncomplicated by sepsis; ~75% of HIE foals recover and grow to be normal adults. Generally, improvement is seen each day. The more severely affected foals may take 5-7 days before they are able to recognize their dam and suckle. Less severely affected foals may recover in 48 hr.