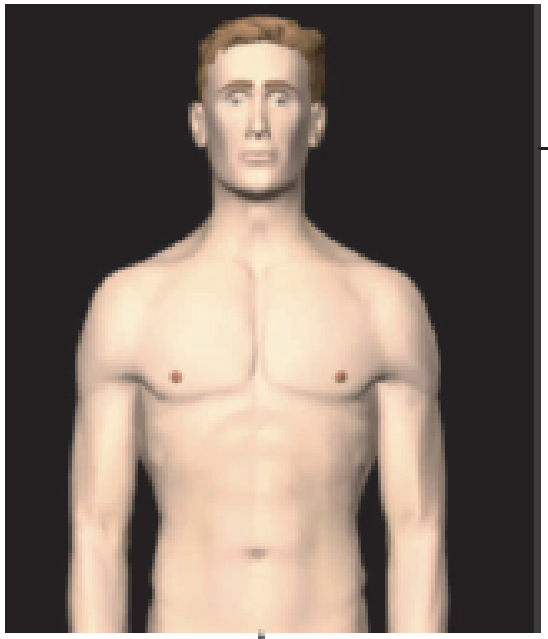
AULA 03 – PTC 3435

09/08/2018

THE BLEEDING EDGE - NETFLIX

https://www.youtube.com/watch?v=uNn2PGPcFzk

SISTEMA DE INSTRUMENTAÇÃO BIOMÉDICA



Sensor

Calibration

Analog Processing

A/D

Control & Feedback

Signal Processing

Data Storage

Output Display

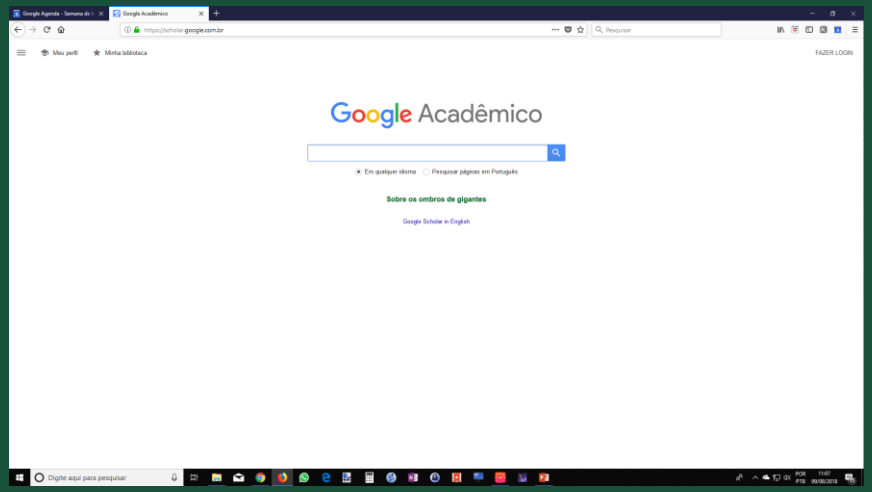
Data Transmission

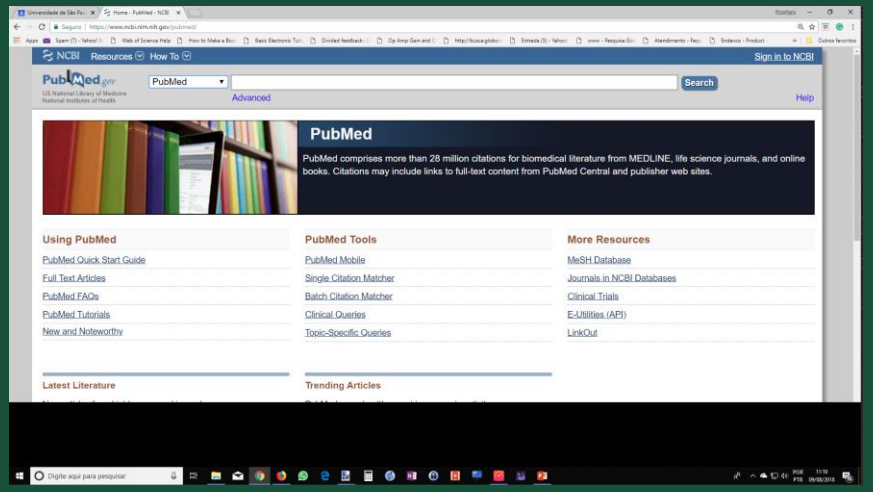
(Bioinstrumentation, Enderle)

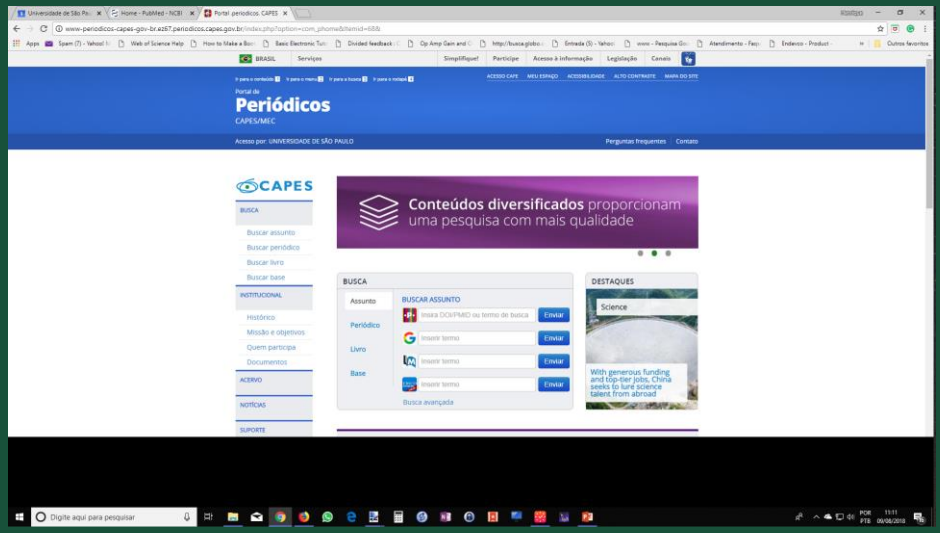
DEFINIÇÃO DAS VARIÁVEIS A SEREM MEDIDAS

Qual efeito fisiológico queremos medir?

DEFINIÇÃO DAS VARIÁVEIS A SEREM MEDIDAS







DEFINIÇÃO DAS VARIÁVEIS A SEREM MEDIDAS

[Original Research **Sleep Disorders**]

CHEST

Different Craniofacial Characteristics Predict Upper Airway Collapsibility in Japanese-Brazilian and White Men

Fabiola Schorr, MD; Fabiane Kayamori, PT; Raquel P. Hirata, PT; Naury J. Danzi-Soares, RN; Eloisa M. Gebrim, MD; Henrique T. Moriya, PhD; Atul Malhotra, MD; Geraldo Lorenzi-Filho, MD; and Pedro R. Genta, MD

BACKGROUND: OSA pathogenesis is complex and may vary according to ethnicity. The anatomic component predisposing to OSA is the result of the interaction between bony structure and upper airway soft tissues and can be assessed using passive critical closing pressure (Pcrit). We hypothesized that Japanese-Brazilians and whites present different predictors of upper airway collapsibility, suggesting different causal pathways to developing OSA in these two groups.

METHODS: Male Japanese-Brazilians (n = 39) and whites (n = 39) matched for age and OSA severity were evaluated by full polysomnography, Pcrit, and upper airway and abdomen CT scans for determination of upper airway anatomy and abdominal fat, respectively.

RESULTS: Pcrit was similar between the Japanese-Brazilians and the whites (-1.0 ± 3.3 cm H_2O vs -0.4 ± 3.1 cm H_2O, $P = .325$). The Japanese-Brazilians presented smaller upper airway bony dimensions (cranial base, maxillary, and mandibular lengths), whereas the whites presented larger upper airway soft tissue (tongue length and volume) and a greater imbalance between tongue and mandible (tongue/mandibular volume ratio). The cranial base angle was associated with Pcrit only among the Japanese-Brazilians ($r = -0.535$, $P < .01$). The tongue/mandibular volume ratio was associated with Pcrit only among the whites ($r = 0.460$, $P < .01$). Obesity-related variables (visceral fat, BMI, and neck and waist circumferences) showed a similar correlation with Pcrit in the Japanese-Brazilians and the whites.

CONCLUSIONS: Japanese-Brazilians and whites present different predictors of upper airway collapsibility. Although craniofacial bony restriction influenced Pcrit only in the Japanese-Brazilians, an anatomic imbalance between tongue and mandible volume influenced Pcrit among the whites. These findings may have therapeutic implications regarding how to improve the anatomic predisposition to OSA across ethnicities.

CHEST 2016; 149(3):737-746

KEY WORDS: computed tomography; Pcrit; ethnicity; OSA

ABBREVIATIONS: AHI = apnea-hypopnea index; MPH = distance from the hyoid to the mandibular plane; Pcrit = passive critical closing pressure; PSG = polysomnography; TV/MV = tongue/mandibular volume; Vimax = peak inspiratory flow

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DOI: http://dx.doi.org/10.1378/chest.15-0638

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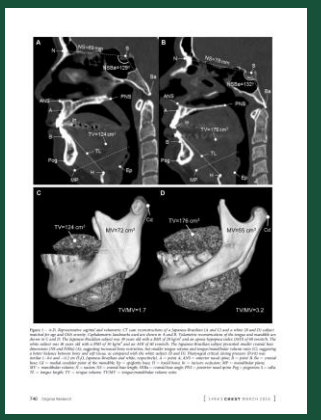

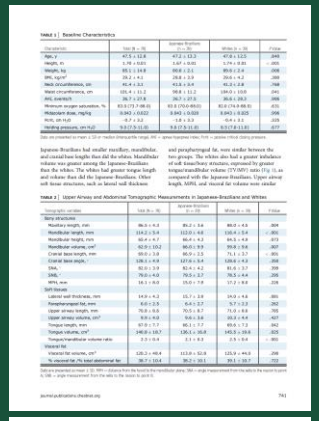

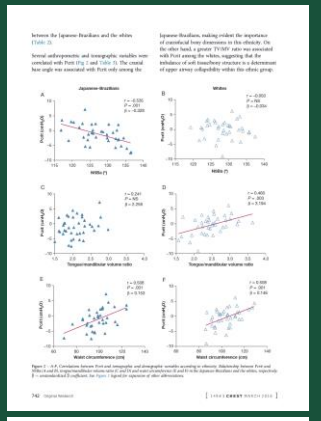

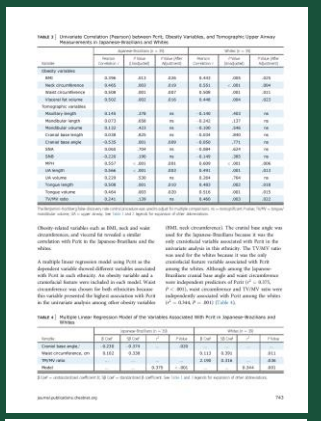

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CHEST

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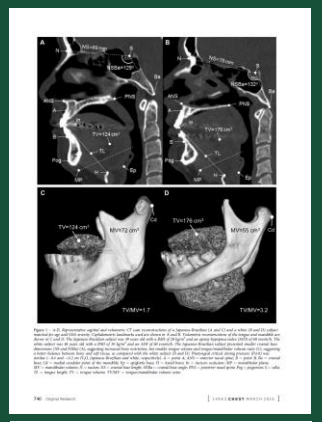

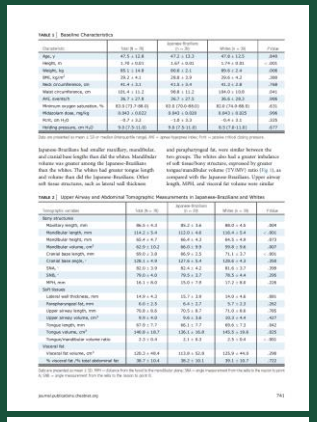

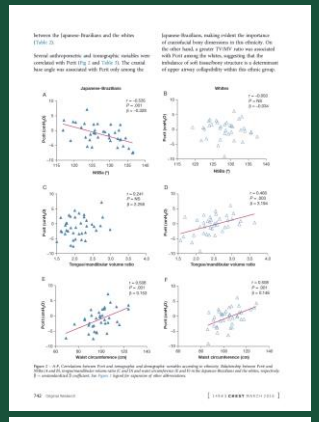

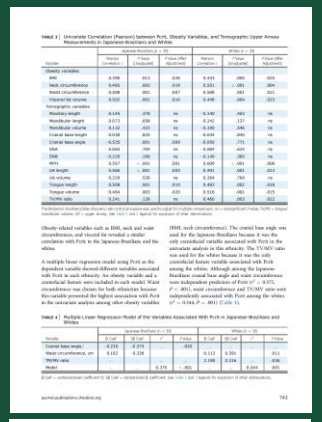

DEFINIÇÃO DAS VARIÁVEIS A SEREM MEDIDAS

OSA is a common disorder among adults that is defined and graded by severity by the apnea-hypopnea index (AHI).[1] The mechanisms that lead to OSA are complex and not completely understood. The balance between the craniofacial bony structure and the upper airway soft tissues is thought to determine an anatomic predisposition to OSA.[2] Obesity may modify the upper airway through the enlargement of the upper airway soft tissues, especially the tongue.[3,4] Several other factors, including neuromuscular modulation, control of breathing, and arousal threshold, may contribute to OSA severity as expressed by AHI.[5,6] All these factors may interact differently according to sex, age, and ethnicity. Because of the differences in craniofacial characteristics and body composition among ethnicities, interethnic studies provide an attractive model for studying the anatomic component of OSA pathogenesis. Asians are thought to be predisposed to OSA because of craniofacial bony restriction. Asians with OSA have been shown to have a shorter cranial base length than do whites.[7,8] In contrast, whites have been shown to have larger upper airway soft tissue, such as enlarged tongue dimensions, when compared with Asians.[7,8] Despite all these differences, OSA prevalence is strikingly similar in Asians and Western countries.[9-11]

Several issues may have limited our understanding of the ethnic differences in OSA. Some interethnic studies included only people with OSA and may have not been able to characterize the full spectrum of the differences among ethnicities. In addition, most studies were controlled by BMI or AHI.[7,8,12] BMI may not be a good metric to compare ethnicities because of ethnic differences in body composition.[13-15] AHI cannot distinguish between anatomic predisposition and the other factors that lead to OSA. The passive critical closing pressure (Pcrit) can assess anatomic predisposition to OSA.[5,16] With a view toward understanding the causal pathways to OSA development, we hypothesized that Japanese-Brazilians and whites would present different predictors of upper airway collapsibility. To test this hypothesis, Japanese-Brazilians and white men well matched for age and OSA severity (full polysomnography [PSG]) underwent Pcrit measurements and had their upper airway anatomy studied by CT scan and body fat composition determined by abdominal CT scan.

Materials and Methods

Study Design

The study consisted of a clinical interview and physical examination, baseline PSG, upper airway and abdominal CT scans, and Pcrit determination. All procedures were performed within 14 days; however, in most subjects, CT scans were performed during the afternoon before baseline PSG, and Pcrit determination was performed the following morning. The study was approved by the Hospital das Clínicas ethics committee (protocol number 0238/08; SDC 3235/08/134). All subjects gave written informed consent before the study started.

Subjects

Male Japanese-Brazilians and whites (18 to 70 years old) referred to the Hospital das Clínicas sleep clinic were recruited. To study the full spectrum of airway collapsibility, healthy subjects from the outpatient primary care clinic were also included. We excluded women; subjects with craniofacial abnormalities; those with comorbidities as defined by COPD, heart failure, chronic kidney disease, or neuromuscular disorders; and those currently using sedative medications. The Japanese-Brazilians and the whites were matched for age (± 7 years) and OSA severity by AHI (± 5 events/h). All subjects underwent a detailed clinical evaluation and a physical examination including height, weight, and waist and neck circumference. Ethnicity was self-reported. All the Japanese-Brazilians confirmed that the previous three generations in their family were either Japanese or Japanese-Brazilian without any miscegenation with other races.

Polysomnography

Subjects were evaluated by full PSG during natural sleep to identify the presence and severity of OSA. Monitoring included EEG, electrooculography, chin and leg electromyography, electrocardiography, oximetry, measurements of airflow (pressure controls and oronasal thermistor), and measurements of thoracic and abdominal movements during breathing (Alice 5; Philips Respironics). Sleep stages were scored manually according to American Academy of Sleep Medicine recommendations.[17] Apnea was defined as complete cessation of airflow (thermistor) for ≥ 10 s. Hypopnea was defined as a > 30% reduction in airflow (nasal pressure) for at least 10 s associated with a 3% oxygen desaturation or cortical arousal.[17]

Upper Airway Collapsibility Determination

Passive Critical Closing Pressure During Pcrit determinations, all polysomnographic channels used in the diagnostic PSG were recorded, except for nasal pressure and thermistor. Pcrit measurements were performed with subjects in the supine position. Each subject was fitted with a nasal mask attached to a heated pneumotachograph (3700A; Hans Rudolf, Inc) and a differential pressure transducer (MP30-14-871; Validyne Engineering) for measurement of airflow. Mask pressure was measured by another pressure transducer (MP45-20-871; Validyne Engineering). Respiratory signals (airflow and mask pressure) were conditioned (CD 280; Validyne Engineering) and recorded on a personal computer using an analog-to-digital converter (PCI-6024; National Instruments) and custom-designed data-acquisition software (LabVIEW; National Instruments). A modified CPAP device (Philips Respironics) that could deliver both positive and negative airway pressure was attached to the mask. Sleep was induced with midazolam as described previously.[11] Briefly, 0.1 mg of midazolam diluted in a saline solution was slowly infused IV (1 min). If the subject awoke and was not able to fall asleep again within 10 min, an additional dose of midazolam was administered until stable sleep was achieved. After sleep onset, CPAP was increased to abolish airflow limitation. This level was used as the building

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DEFINIÇÃO DAS VARIÁVEIS A SEREM MEDIDAS

Acknowledgments

References

746 Original Research

[CHEST MARCH 2016]

SINAIS BIOELÉTRICOS

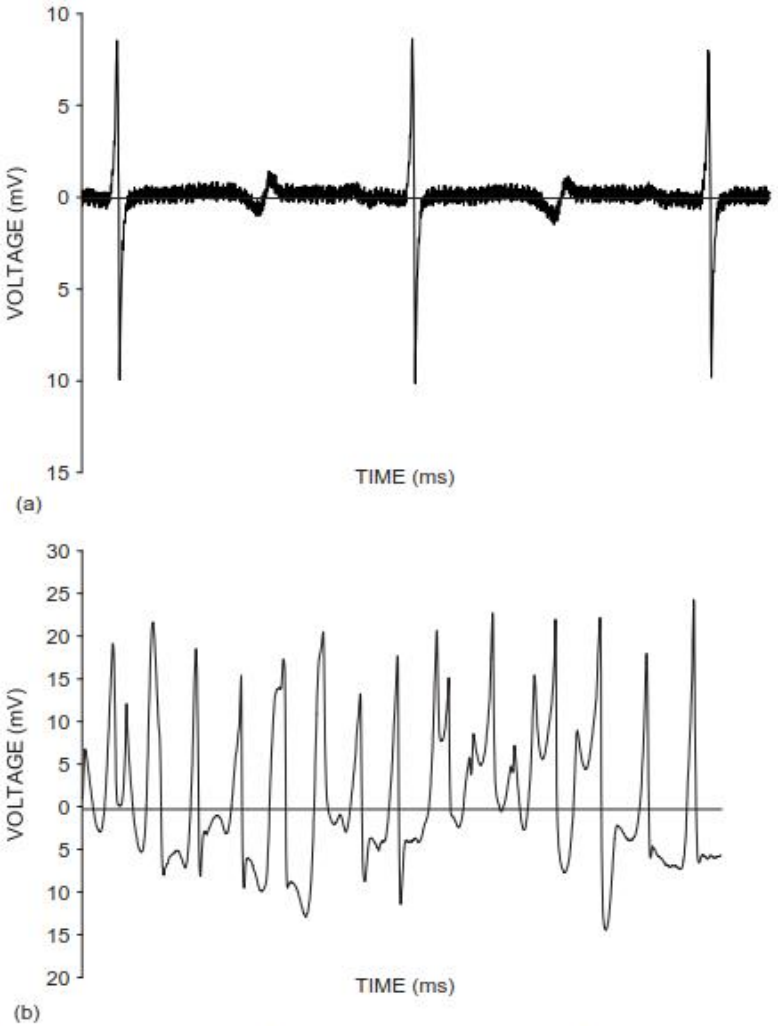


FIGURE 11.1 (a) Electrogram recorded from the surface of a pig's heart during normal sinus rhythm. (b) Electrogram recorded from the surface of the same pig's heart during ventricular fibrillation (VF). (Sampled at 1,000 samples/s.)

(Introduction to Biomedical Engineering 3ª ed, Enderle, Bronzino (eds), Biosignal Processing, M. Escabí)

ELETROCARDIOGRAMA

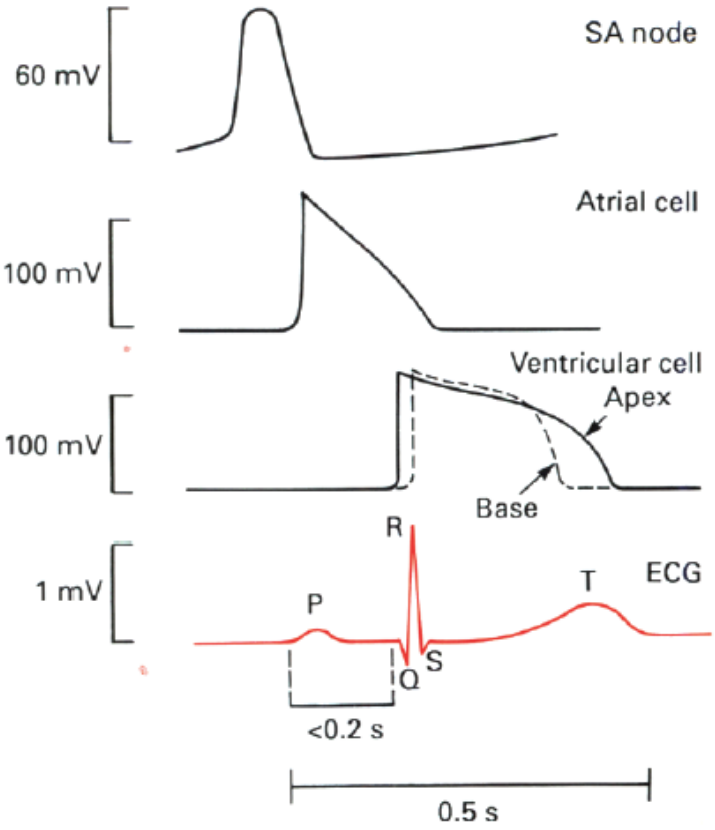


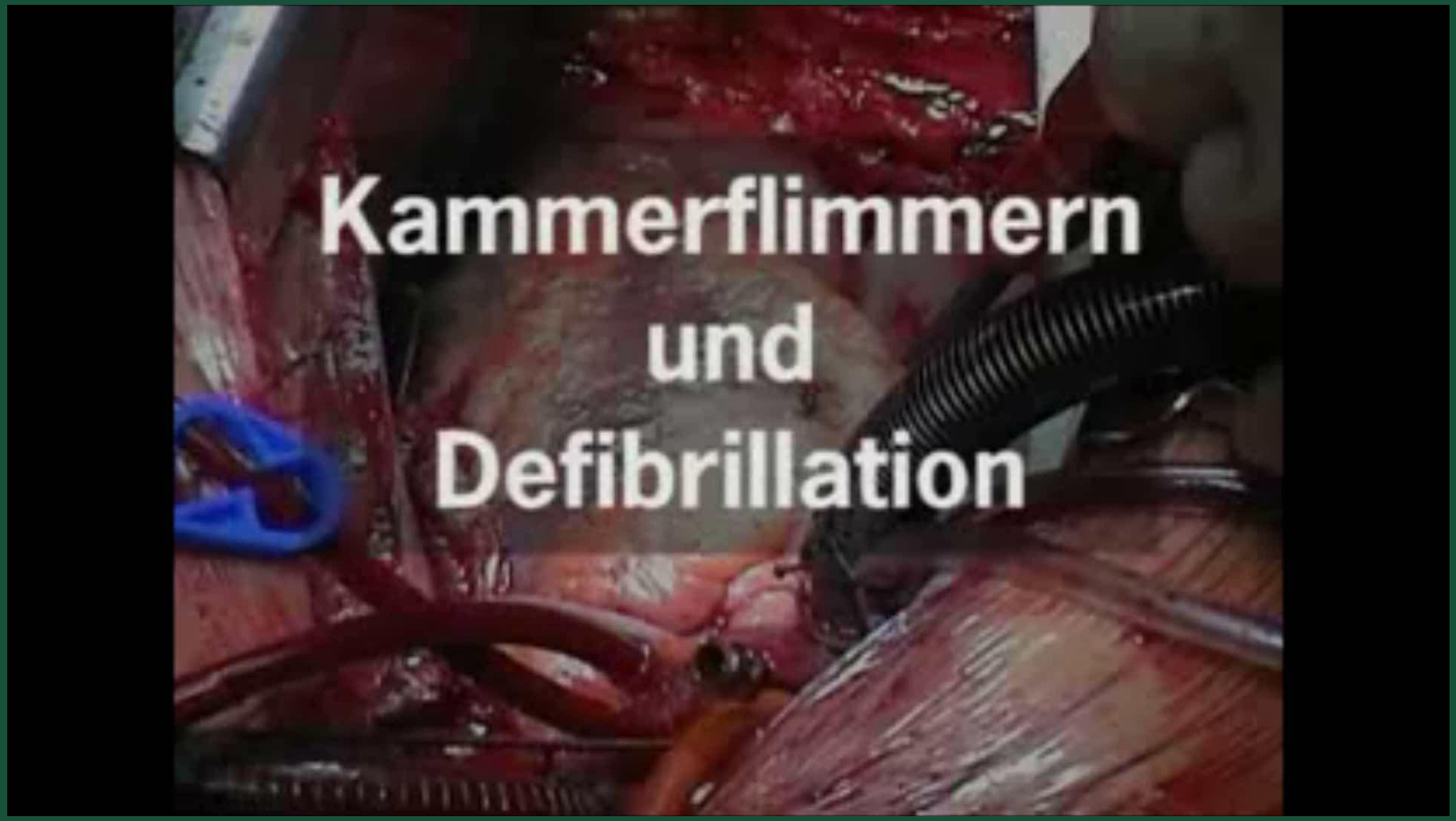
Figure 4.3 Timing of the ECG waves compared with intracellular recordings at different sites, including two sites in the ventricle (apex and base). Note that the ECG voltage scale is much smaller than that for the membrane potentials. Note also that the base (dashed line) repolarizes before the apex, which is why the T wave is upright

(Levick, J.R.,1991)

ELETROCARDIOGRAMA

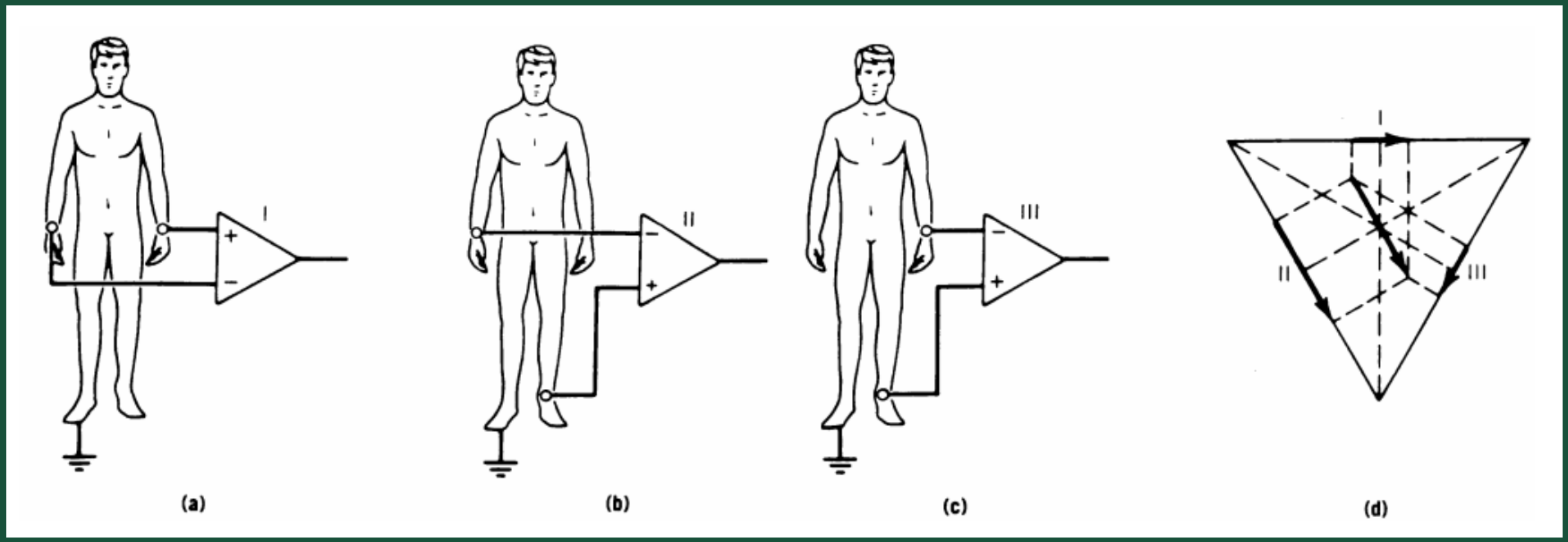
http://www.youtube.com/watch?v=VKxQgjj2yVU

ELETROCARDIOGRAMA

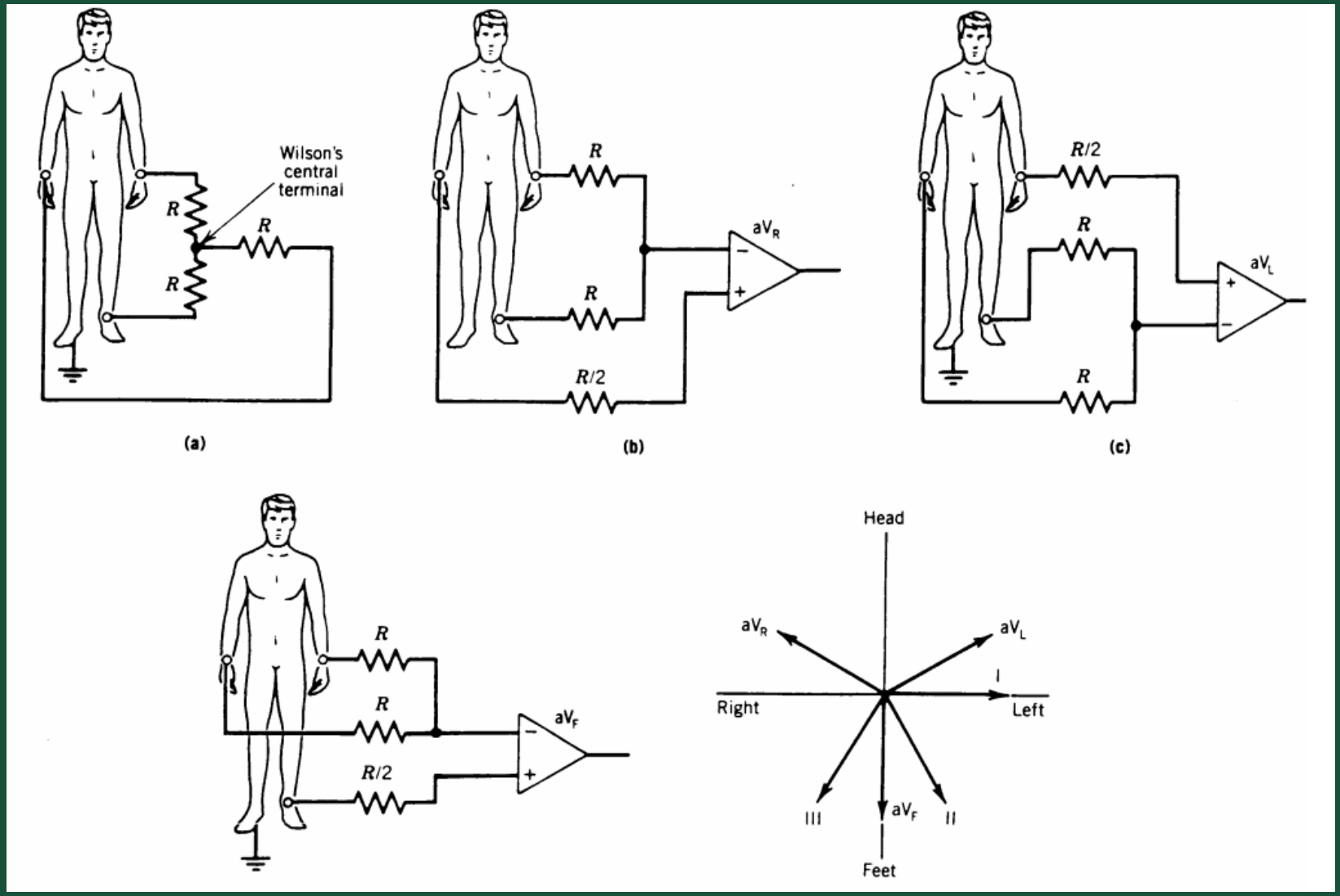


http://www.youtube.com/watch?v=HCbawp9ZSnY

DERIVAÇÕES BIPOLARES



DERIVAÇÕES AUMENTADAS



DERIVAÇÕES PRÉ-CORDIAIS

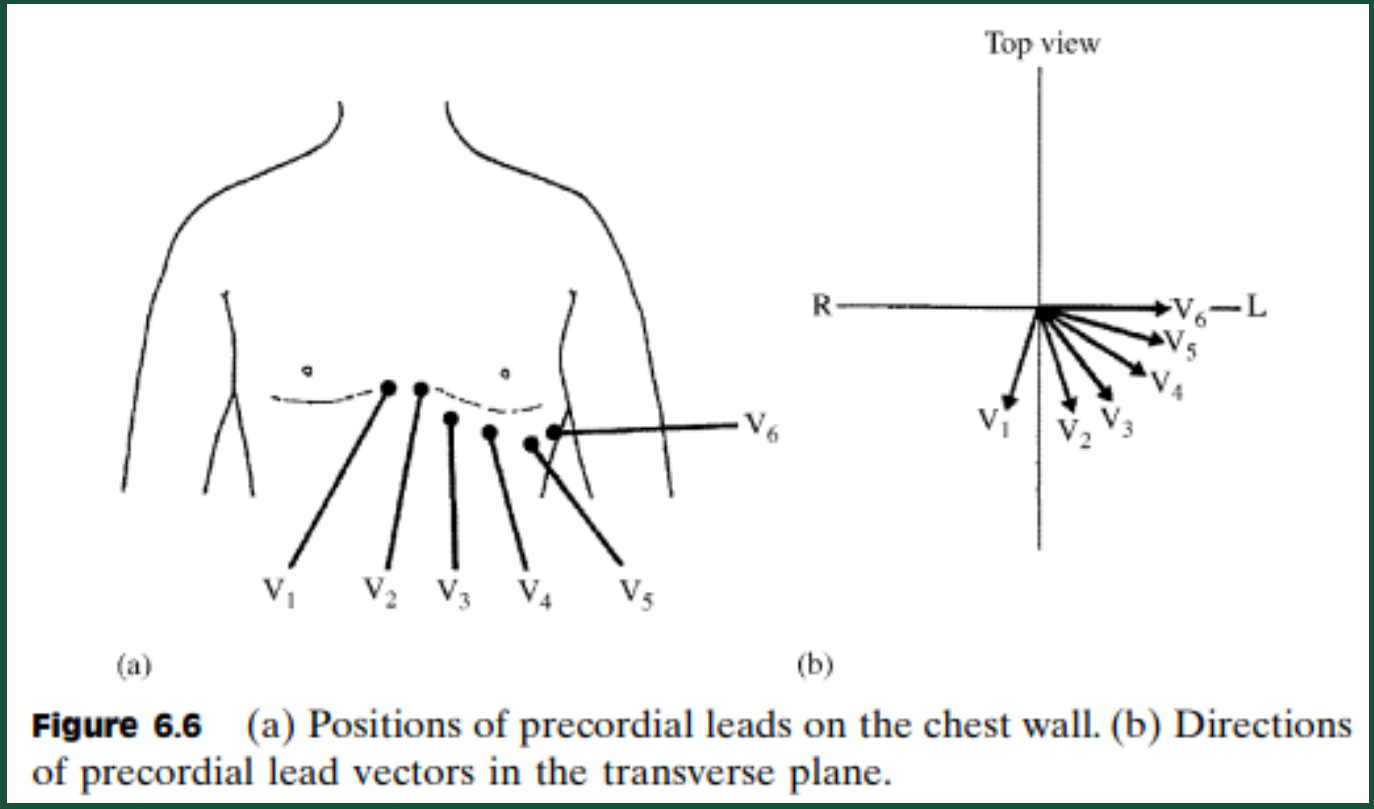


Figure 6.6 (a) Positions of precordial leads on the chest wall. (b) Directions of precordial lead vectors in the transverse plane.

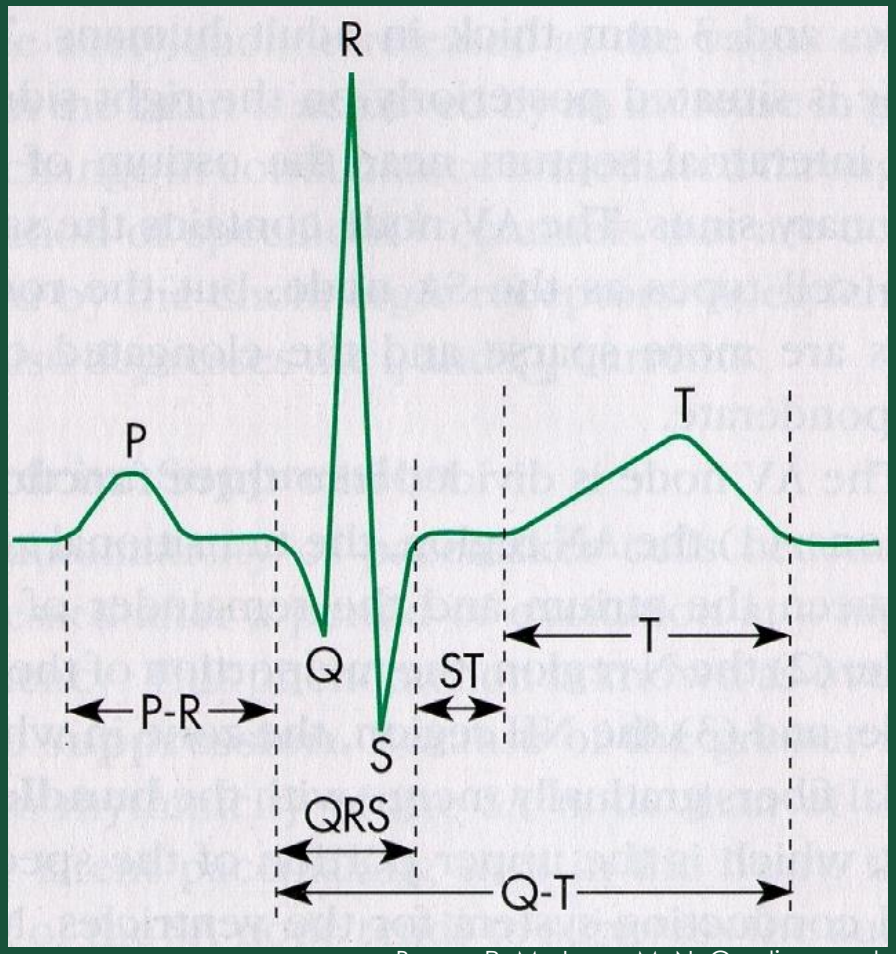
(WEBSTER, J.G.,1998)

DERIVAÇÕES

Tabela I. Sistema padrão de 12 derivações

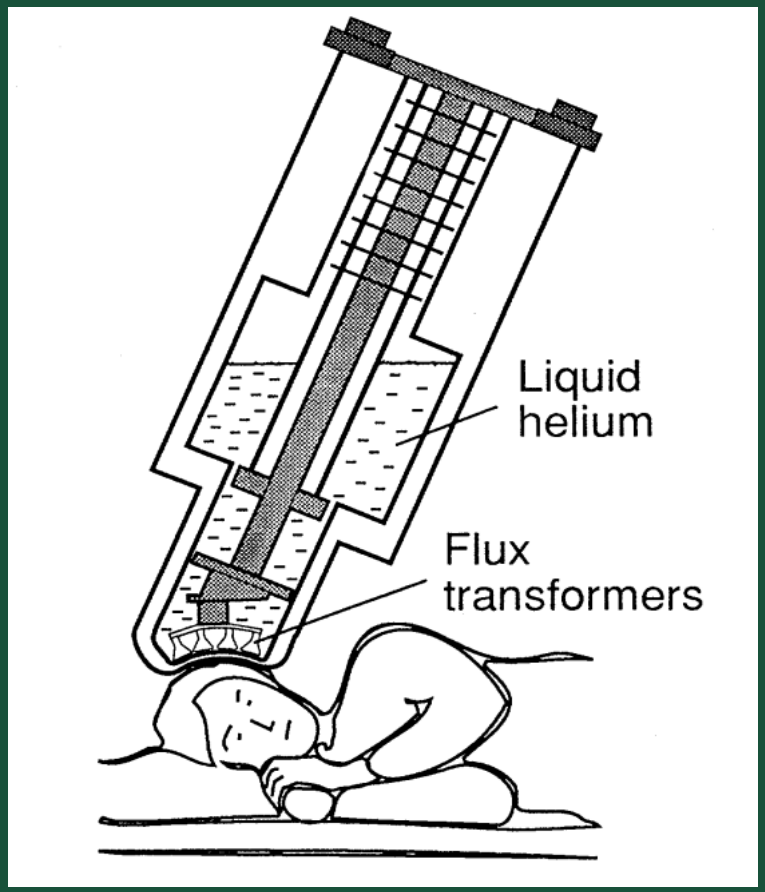
Tipo de derivação	Eletrodos usados	Definição
Bipolar ou derivação de membros	LA, RA, LL, RL	I = LA – RA II = LL – RA III = LL – LA
Aumentada ou derivação unipolar de extremidade (Goldberg	LA, RA, LL, RL	aVR = RA - ½(LA + LL) aVL = LA - ½ (LL + RA) aVF = LL - ½ (LA + RA)
Unipolares precordiais (Wilson)	V1, V2, V3, V4, V5 e V6 (mais 1 em cada braço, 1 em cada perna, sendo a direita aterrada; eletrodo explorador = vi, i entre 1 e 6, uma das posições pré-cordiais)	V1 = v1 - (RA+LA+LL) / 3 V2 = v2 - (RA+LA+LL) / 3 V3 = v3 - (RA+LA+LL) / 3 V4 = v4 - (RA+LA+LL) / 3 V5 = v5 - (RA+LA+LL) / 3 V6 = v6 - (RA+LA+LL) / 3

ELETROCARDIOGRAMA ESCALAR TÍPICO



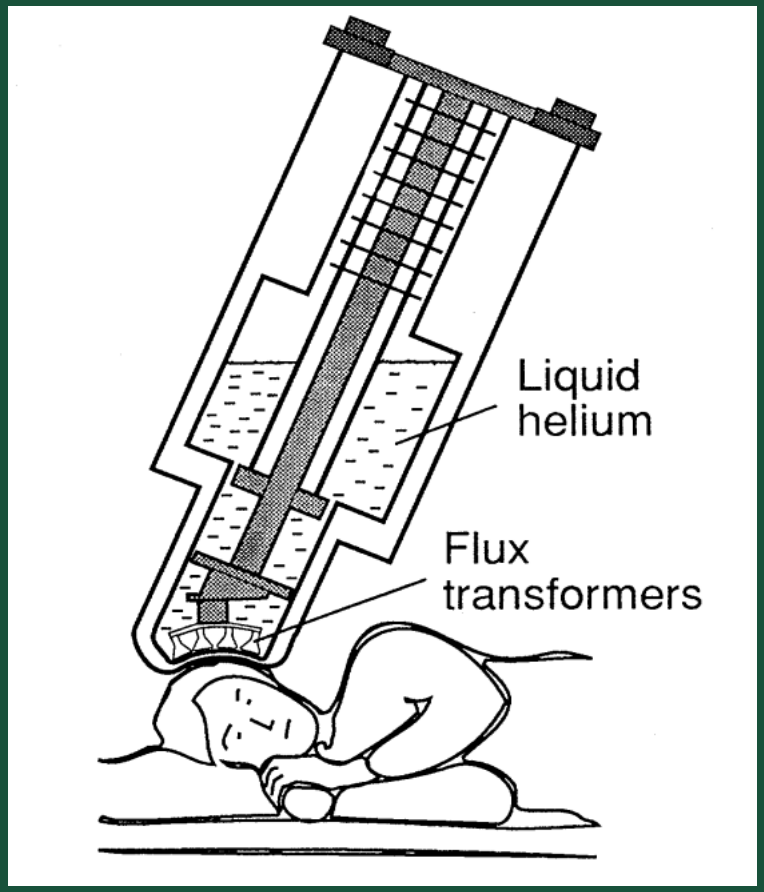
Berne, R. M., Levy, M. N. Cardiovascular physiology. 8th ed. Mosby, 2001.

SINAIS BIOMAGNÉTICOS

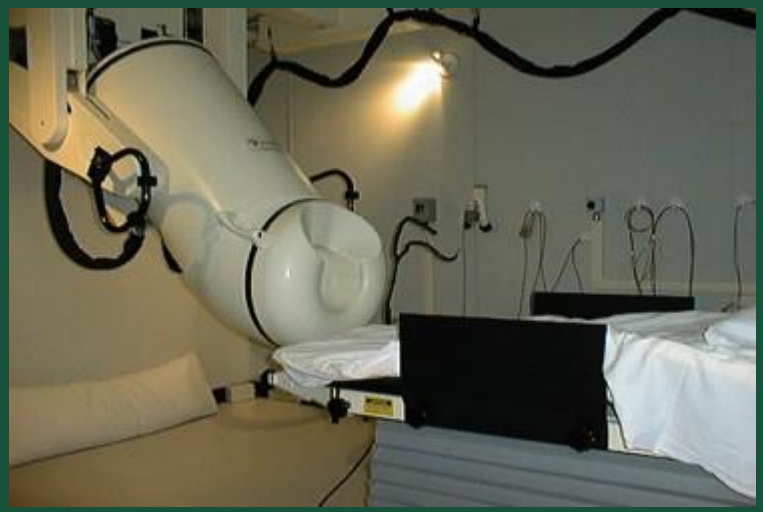


Hämäläinen et al, Rev. Mod. Phys., Vol. 65, 2, 1993

SINAIS BIOMAGNÉTICOS

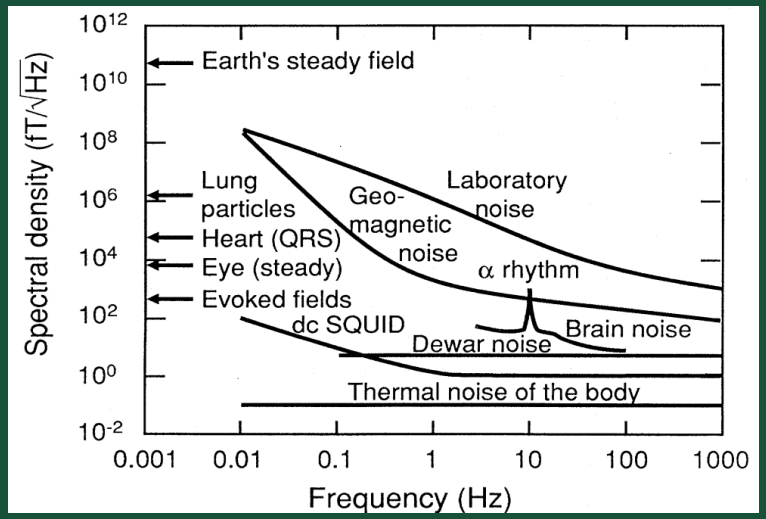


Hämäläinen et al, Rev. Mod. Phys., Vol. 65, 2, 1993



http://www.ucdenver.edu/academics/colleges/medicalschool/departments/psychiatry/Research/BrainImagingCenter/Technologies/Pages/MEG.aspx

SINAIS BIOMAGNÉTICOS



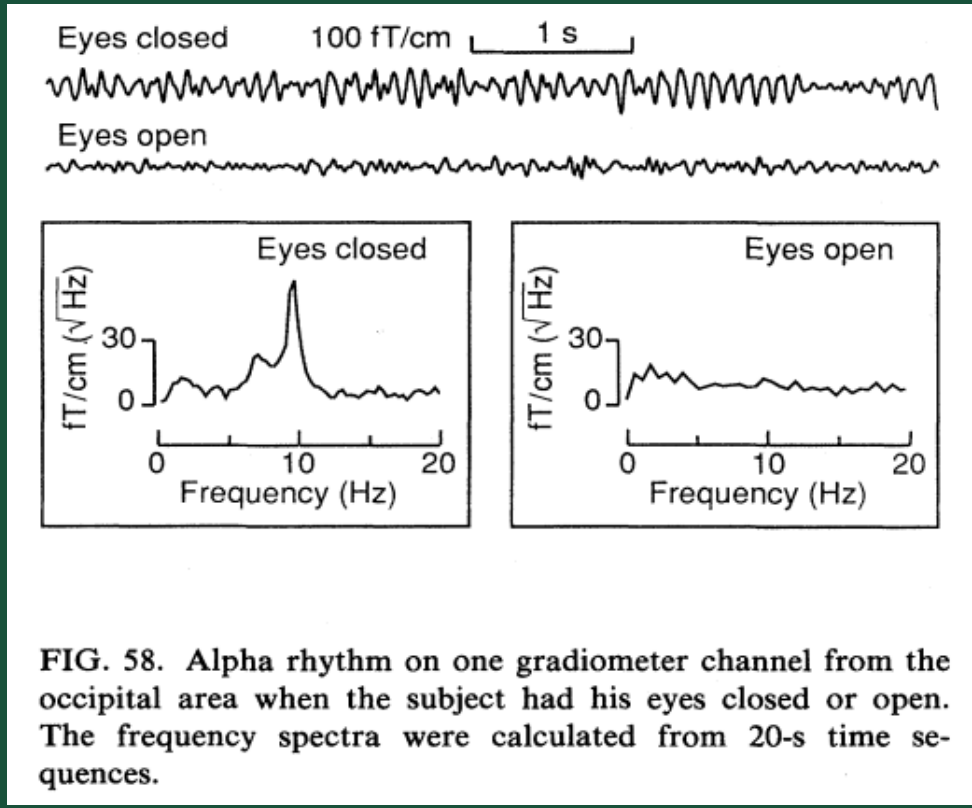


FIG. 58. Alpha rhythm on one gradiometer channel from the occipital area when the subject had his eyes closed or open. The frequency spectra were calculated from 20-s time sequences.

Hämäläinen et al, Rev. Mod. Phys., Vol. 65, 2, 1993