Dr. Javier Bocos
Dra. Lorena Muñoz

Servicio de Anestesia Reanimación y Tratamiento del Dolor
Consorcio Hospital General Universitario
Valencia

Sartd-CHGUV Sesión de Formación Continuada
Valencia 4 de Marzo 2008

CASO CLÍNICO

- EDAD:27 A
- SEXO: VARÓN
- AP: ENOLISMO MODERADO.
- ACCIDENTE MOTO
- LESIONES PRIMARIAS: ESCORIACIONES TORAX Y ESPALDA, FX ESCAFOIDES DERECHO, FX TERCIO MEDIO FÉMUR DERECHO, SIN PÉRDIDA DE CONCIENCIA.
- **INTERVENCIÓN QUIRÚRGICA:48 H**
- LESIONES SECUNDARIAS: HIPOXEMIA, PETEQUIAS TRONCO Y DESATURACIÓN BRUSCA COINCIDIENDO CON TRANSFUSIÓN DE UN CONCENTRADO DE HEMATIES.
- GSA: HIPOXEMIA, HIPOCAPNIA, ANEMIA, HIPOCALCEMIA
- CLÍNICA POSTERIOR: SDR. CONFUSIONAL Y AGITACIÓN PSICOMOTRIZ TRAS EXTUBACIÓN

Diagnóstico diferencial

- TEP (embolismo graso)
- Síndrome de abstinencia alcohólica
- Distress asociado a la transfusión

EMBOLIA GRASA
HISTORIA

- Zenker FA.
  Beitraege zur anatomie und physiologic der lunge.
  Dresden: J.Braunsdorf: 1861

- Bergman EB.
  Ein fall todlicher fettembolie.
  Klin Wochenschr.1873; 10:385-387

EMBOLISMO GRASO
EPIDEMIOLOGIA

- PREVALENCIA: 90% POLITRAUMÁTICOS
- EDAD: 20- 30 AÑOS
- MENOR INCIDENCIA EN EDAD INFANTIL
- MORTALIDAD: 5-20%

SEG
ETIOLOGIA TRAUMÁTICA

- >90% ÉMBOLOS GRASOS
- 4% SEG
- 50% HIPOXEMIA RELATIVA
- 0.5-2% FX ÚNICAS HUESOS LARGOS
- 5-11% CON FX MULTIPLES HUESOS LARGOS Y PELVIS
- 85% EN PRIMERAS 48 H POST TRAUMA
- POLITRAUMATISMO
- NO CORRELACIÓN SEVERIDAD DEL POLITRAUMATISMO Y SEVERIDAD DEL SEG
- FRACTURAS CERRADAS >>ABIERTAS

SEG
ETIOLOGIA NO TRAUMÁTICA

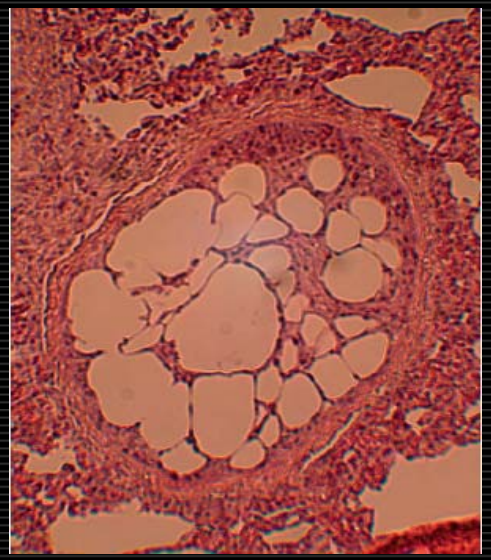
- LIPOSUCCIÓN
- HISTEROSCOPIAS
- INFECCIONES
- CETOACIDOSIS
- ECLAMPSIA
- PANCREATITIS
- NPT
- TX MÉDULA ÓSEA
- ESTEATOSIS HEPÁTICA
- DESCOMPRESIÓN BRUSCA
- LESIONES TEJIDOS BLANDOS
- DIABETES MELLITUS
- QUEMADOS
- ALCOHOLISMO
- OSTEOMIELITIS
- TRANSFUSIÓN SANGUÍNEA AUTÓLOGA
- BYPASS CARDIOPULMONAR
- ANEMIA CÉLULAS FALCIFORMES
- TX E INFARTOS RENALES
- CORTICOIDES
- CICLOSPORINA A
- ANESTÉSICOS INHALADOS
- FRACTURAS VERTEBRALES

FISIOPATOLOGIA SEG

- TEORÍA MECÁNICA
- TEORÍA BIOQUÍMICA
- TEORÍA INMUNOLÓGICA

Teoria mecánica SEG

- GRASA MEDULAR
- CIRCULACIÓN MEDULAR
- FOCO FRACTURA
- CAPILARES < 20 MICRAS

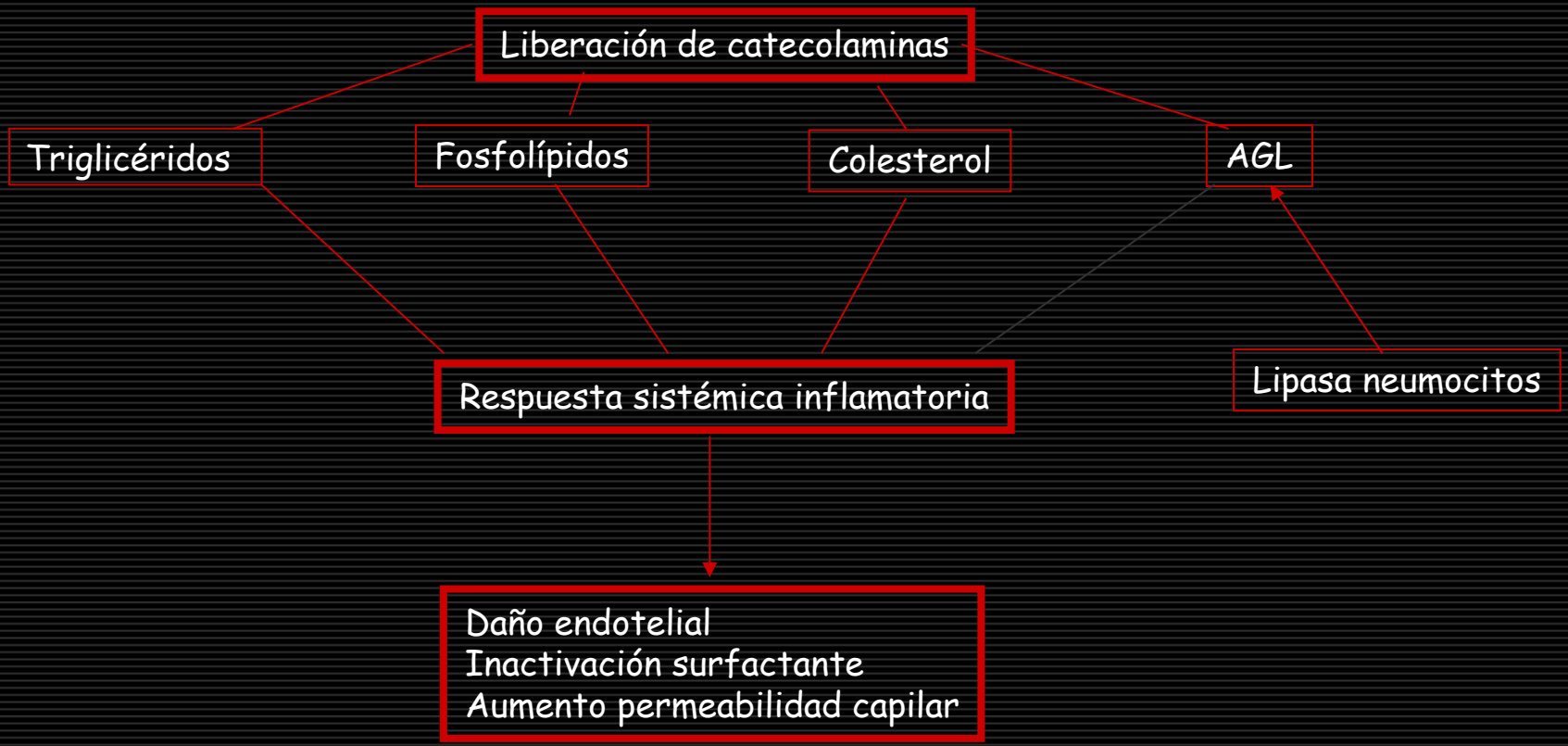


Experimental Cerebral Fat Embolism: Embolic Effects of Triolein and Oleic Acid Depicted by MR Imaging and Electron Microscopy

Hak Jin Kim, Jong Hwa Lee, Chang Hun Lee, Suk Hong Lee, Tae Yong Moon, Byung Mann Cho, Hae Kyu Kim, Byung Rae Park, and Kee Hyun Chang

AJNR Am J Neuroradiol 23:1516–1523, October 2002

Teoría bioquímica SEG

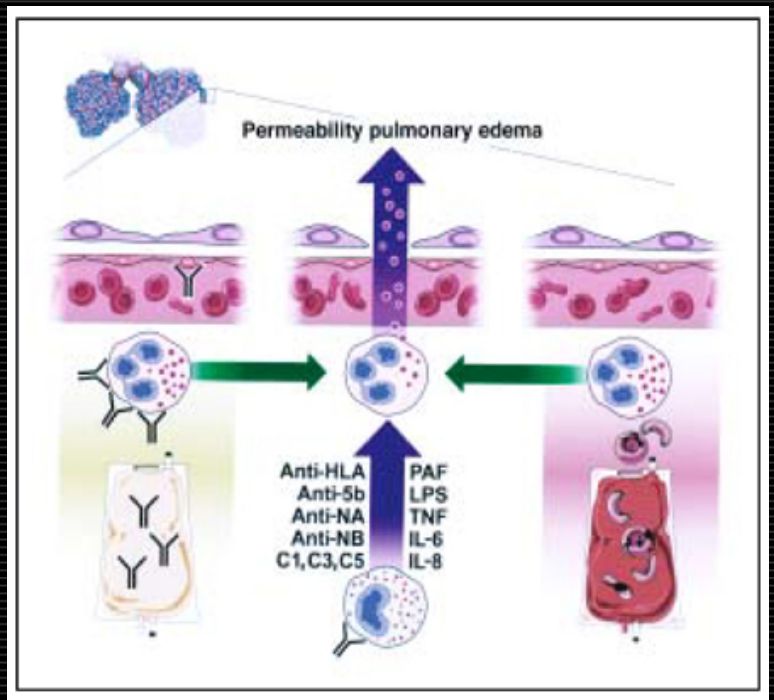


Teoría inmunológica SEG

Serotonina

Histamina

Lisozima



Alteraciones inmunológicas en paciente politraumático

Citoquinas

Proinflamatorias

- IL-6 y TNF-alfa
- IL-8
- IL-10

Antiinflamatorias

- IL-10
- IL-4
- TGF-Beta
- IL-1a

- Activación complemento

Radicales libres

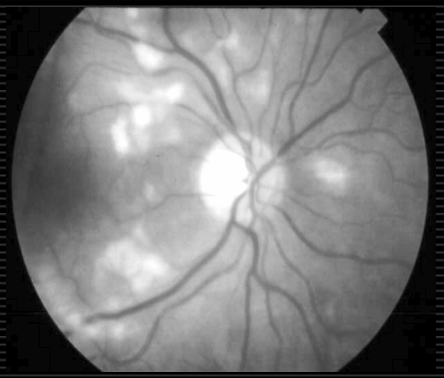
Sepsis-SIRS-MODS

ESTADO INFLAMATORIO PERSISTENTE

Criterios de Gurd-Wilson

1.CRITERIOS MAYORES

- INSUFICIENCIA RESPIRATORIA
- ALTERACIONES NEUROLÓGICAS
- EXANTEMA PETEQUIAL



2.CRITERIOS MENORES

- FIEBRE
- TAQUICARDIA
- FONDO DE OJO ALTERADO (RETINOPATIA DE PURTSCHER)
- ICTERICIA
- ALTERACIONES RENALES (LIPIDURIA/OLIGURIA)

DIAGNÓSTICO: 1 MAYOR + 4 MENORES + MACROGLOBULINEMIA GRASA

Gurd AR, Wilson RI. The fat embolism syndrome. J.Bone Joint Surg Br 1974;56B:408-16

Criterios de Lindeque

1. PAO2 <60

2. PACO2 >55

3. PH <7.3

4. FRECUENCIA RESPIRATORIA> 35

5.↑ TRABAJO RESPIRATORIO

Lindeque et al. Fat embolism and the fat embolism syndrome
J Bones Joint Surg Br 1987;69:128-131

Criterios de SCHONFELD

PETEQUIAS	5
CAMBIOS RX	4
HIPOXEMIA	3
FIEBRE	1
TAQUICARDIA	1
TAQUIPNEA	1
CONFUSION	1

DIAGNÓSTICO: SCORE > 5

Ann Intern Med 1983; 99: 438-443

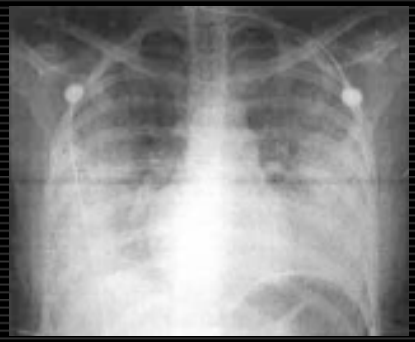
ESCALA DE SHIER

- Cuerpo de fémur — 4
- Cabeza de fémur
- Pelvis
- Tibia
- Húmero — 2
- Radio
- Cúbito
- Peroné — 1

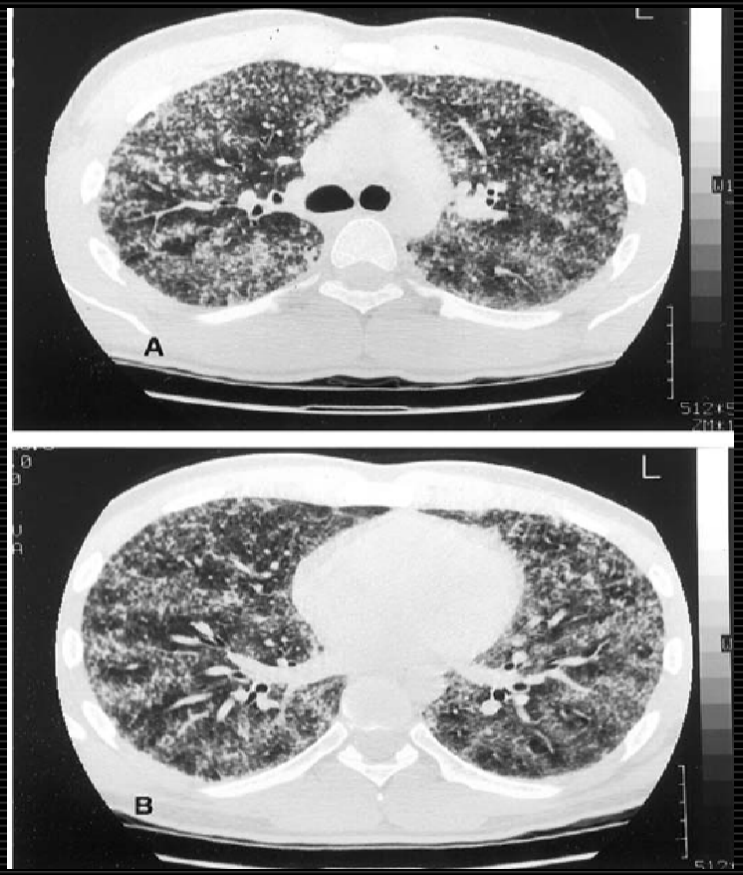
J Trauma 1977; 17 (8): 621-629.

HIPOXEMIA
DIAGNÓSTICO RADIOLÓGICO

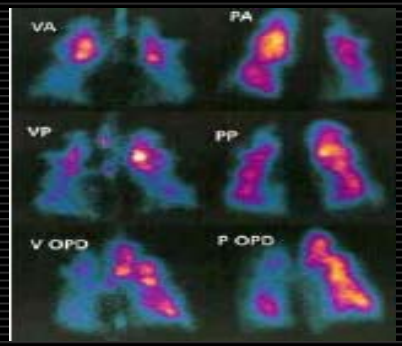
- RX



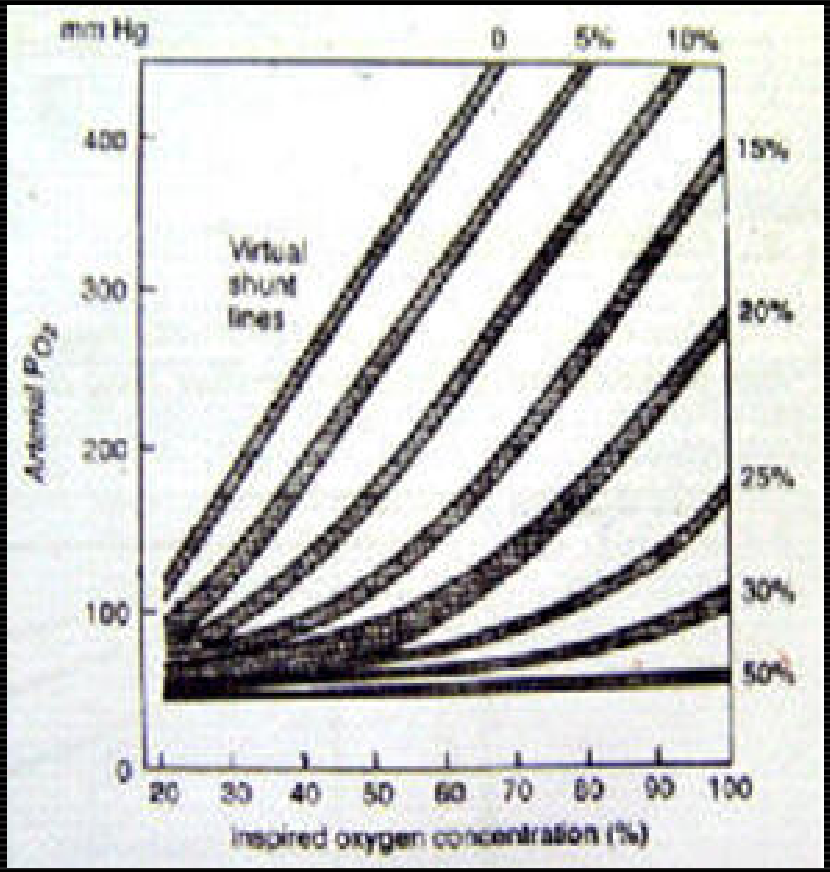
- TC

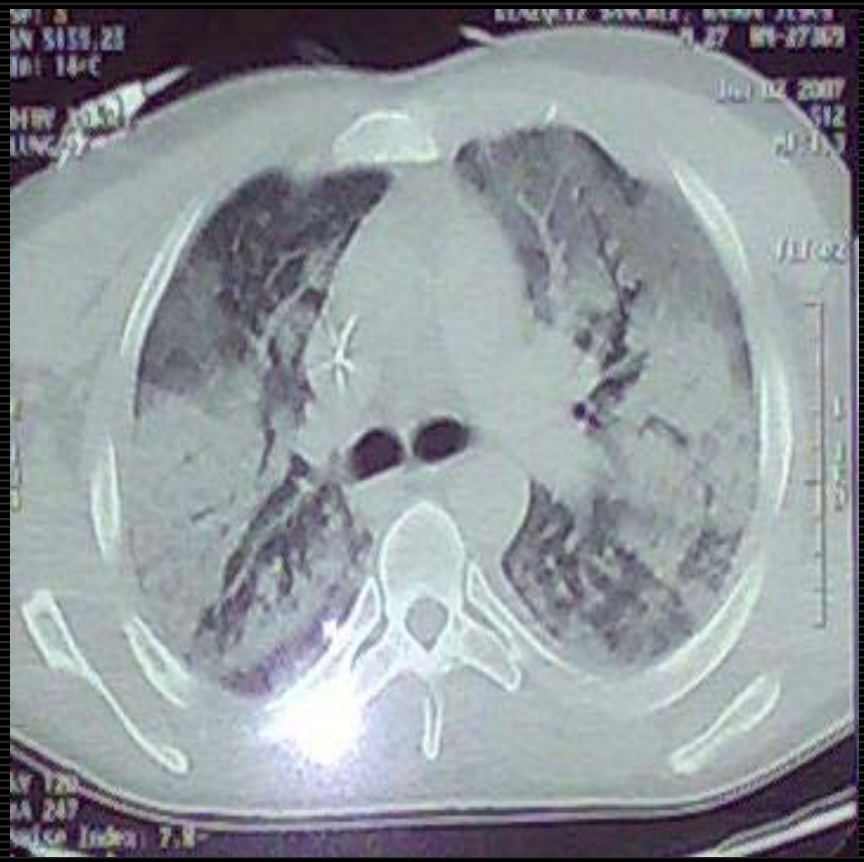


- Gammagrafias de V/P



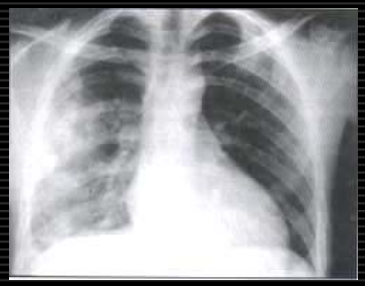
Disfunción pulmonar
SHUNT D-I





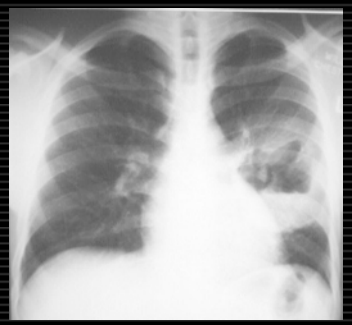
DD
ALTERACIONES RADIOLÓGICAS

- CONTUSION PULMONAR:



33% NEGATIVA INGRESO
0-6-36 H POSITIVIZA
48-72 H RESOLUCIÓN
SENSIBILIDAD 38%
NO RESPETA CISURAS
FX ASOCIADAS
MECANISMO LESIVO
VOLUMEN CONTUSIÓN (TC) SDRA

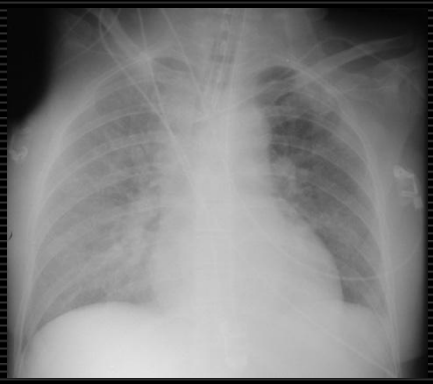
- BRONCOASPIRACION:



4-36 h
CONSOLIDACIÓN ALVEOLAR
RESPETA LÓBULOS
A PARTIR 72 H INICIO RESOLUCIÓN
SEGMENTOS POSTERIORES LS
SEGMENTOS APICALES LI
VOLUMEN ASPIRACIÓN SDRA

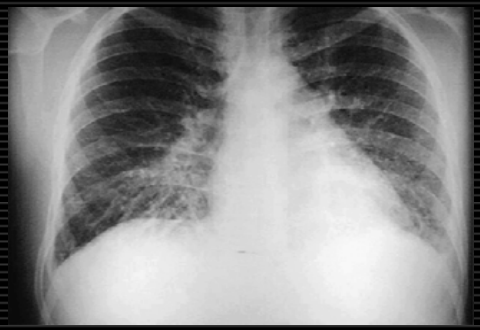
DD
ALTERACIONES RADIOLÓGICAS

- EAP NEURÓGENO:



  - SILUETA CARDIACA N
  - HILIOS NO PROMINENTES
  - PATRÓN MOTEADO PEQUEÑO
  - BRONCOGRAMA AÉREO
  - NO SIGNOS DE HT PULMONAR
  - INICIO Y RESOLUCIÓN < 48-72H
  - BUEN PRONÓSTICO

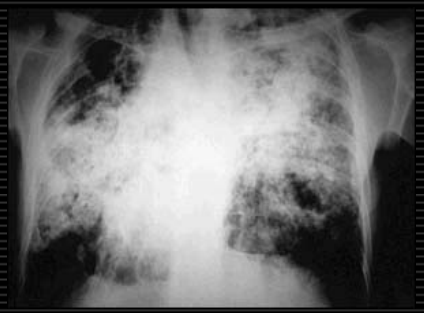
- EAP CARDIOGÉNICO:



  - HILIOS PROMINENTES
  - PATRÓN EN "ALAS DE MARIPOSA"
  - SOBRECARGA HÍDRICA
  - RX PRECOZ

DD
ALTERACIONES RADIOLÓGICAS

- SDRA



FASE INICIAL AUMENTO PERMEABILIDAD

AUMENTO AGUA EXTRAVASCULAR (ELWI)

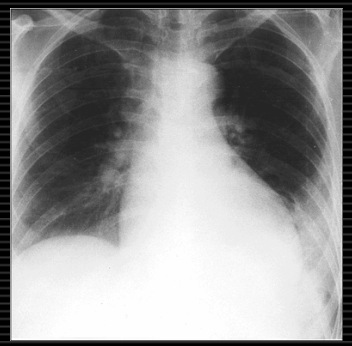
ORIGEN EXTRAPULMONAR:
AUMENTO DENSIDADES PARAVERTEBRALES

ORIGEN PULMONAR:
AUMENTO DENSIDADES PARAESTERNALES

LATENCIA > 24 H
DURACIÓN: SEMANAS

- TEP

N
ATELECTASIA
SIGNO DE WESTERMARK
SIGNO DE FLEISCHNER
GIBA DE HAMPTON
DERRAME PLEURAL



TEMPORALIDAD DE LESIONES E IMÁGENES RADIOLÓGICAS

HORA / LESIÓN	0-6	12-72	96	10 D
CONTUSION LEVE/GRAVE	P	P	A/P	A/P
SDRA	A	P	P	P
SEG	A	P	P	A
BRONCO ASPIRACIÓN	A	P	P	A
EAP CARDIOGÉNICO	P	P	P	P
EAP NEURÓGENO	P	P	A	A

P- PRESENTE
A- AUSENTE

High-Resolution CT Findings in Mild Pulmonary Fat Embolism*

Katerina Malagari, MD; Nikos Economopoulos, MD; Christophoros Stoupis, MD; Zoe Daniil, MD; Spyros Papiris, MD, FCCP; Nestor L. Müller, MD; and Dimitrios Kelekis, MD

(CHEST 2003; 123:1196–1201)

- Opacidades tipo "Ground-glass " 80%
- Septos interlobulares delgados 55%
- Distribución parcheada-geográfica 44%
- Afectación nodular 22%
- Diámetro bronquios y arterias NORMAL
- Derrame pleural 0%

Inicio: 1- 3 dias

Resolución: 7-25 dias

F.Realización: 02/06/07 → **6 dias tras accidente**
Médico y centro solicitante: [redacted], H.GRAL.UNIV.VCIA - MUR
Exploración solicitada:
HM-27369

TC. CEREBRAL, ANGIO A.PULMONARES

INFORMACION CLINICA: paciente comatoso postquirúrgico.

ESTUDIO EFECTUADO: vascular

INFORME: . Cerebral: No se identifican lesiones ocupantes de espacio ni zonas de isquemia o hemorragia intraparenquimatosa aguda. Línea media centrada. Sistema ventricular dentro de la normalidad.
Tórax: No evidenciamos signos de tromboembolismo pulmonar. Adenopatías mediastínicas prevasculares. Patrón intersticial en vidrio delustrado parcheado , bialteral, con zonas de condensación intraparenquimatoso y broncograma aereo en su interior compatible con neumonía a valorar pneumocistis carinii. Discreto derrame pleural bilateral.

Manifestaciones neurológicas del embolismo graso

- 80% SEG
- Agitación
- Confusión
- Estupor
- Focalidades motoras 33%
- Hiperreflexia 33%
- Temblores generalizados
- Rigidez descerebración 15%
- Signos piramidales 30-70%
- TAC normal

Fat Embolism and Neurological Dysfunction

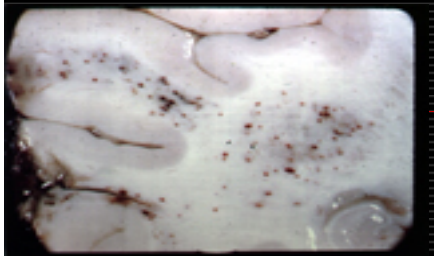
ANESTH ANALG 1999;88:1421–9

Rottenberg DA, Bennett WM, Wolpow ER. **Transient diabetes insipidus complicating systemic fat embolization.** *J Trauma* 1972;12:731–733

Embolization of the Meningohypophyseal Trunk as a Cause of Diabetes Insipidus

Constantine C. Phatouros, Randall T. Higashida, Adel M. Malek, Wade S. Smith, Christopher F. Dowd, and Van V. Halbach

AJNR Am J Neuroradiol 20:1115–1118, June/July 1999



Manifestaciones neurológicas del embolismo graso

Buen pronóstico:

- ROTs presentes
- Respuesta N al dolor

Mal pronóstico:

- Brusquedad en el coma

Pronóstico:

- 25% discapacidad neurológica permanente.

Disfunción neurológica Fisiopatologia

- Moldeamiento del émbolo a través del capilar pulmonar
- Shunts AV pulmonares
- FOP
- Aneurisma septo auricular (ASA)

Shunts AV pulmonares

Asociaciones:

- Cirrosis hepática 60%
- Ejercicio físico

Diagnóstico:

- ETE con contraste
- Angiografia pulmonar

AJNR 99 20(10) 1881-4

ASA

- Prevalencia por ETT: 0.12-1.7%
- Prevalencia por ETE: 2.1-10%
- Prevalencia FOP-ASA: 50-89%

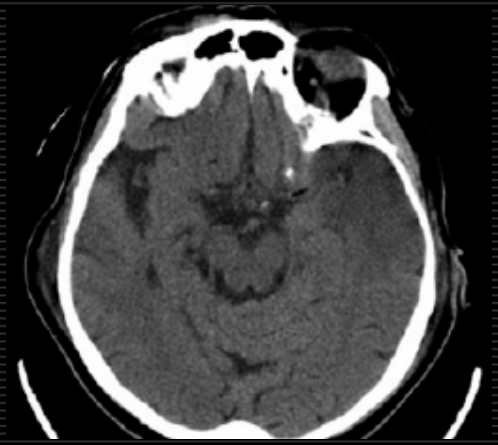
Tipos:
- 1 AD
- 1A < 0,5 cm
- 1B > 0,5 cm
- 2 AI

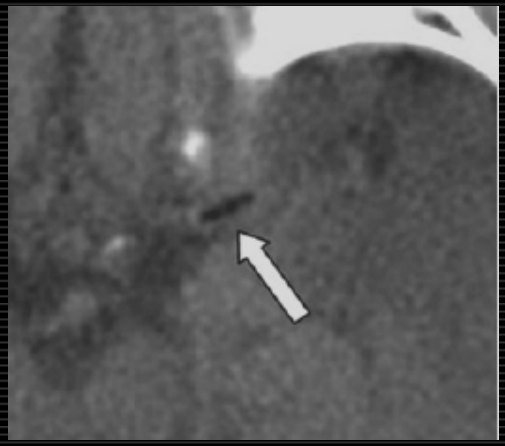
Hanley et al. J Am Coll Cardiol 1985

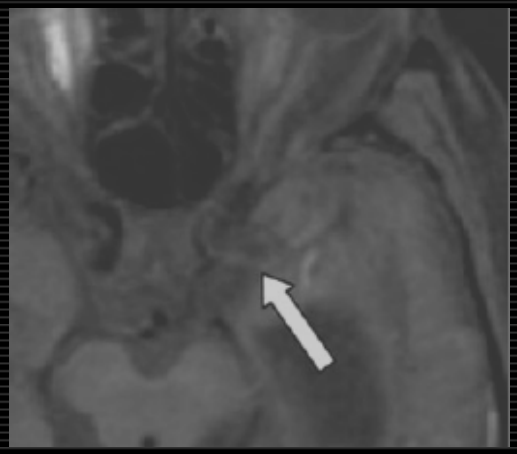
The Hypodense Artery Sign

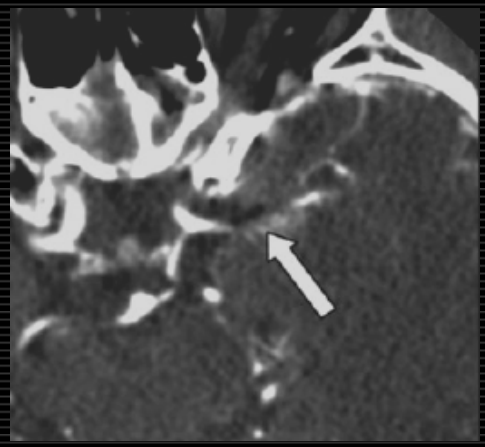
Thomas C. Lee, Eric S. Bartlett, Allan J. Fox, and Sean P. Symons

AJNR Am J Neuroradiol 26:2027–2029, September 2005









TC cerebral y SEG

- Embolos grasos - Sustancia gris

- Isquemia

- Hemorragias
  - Centro semioval
  - Sustancia blanca cerebral
  - Sustancia blanca cerebelosa

Edema cerebral difuso
Borramiento surcos
Ventriculos pequeños

↓ 7 dias

Hipoatenuación
Atrofia cerebral

RMN Y SEG

- TÉCNICA + SENSIBLE
- DIFUSIÓN
- T2
- DIAGNÓSTICO PRECOZ
- PRONÓSTICO
- EVOLUCIÓN

CASE REPORT

D.J.A. Butteriss
D. Mahad
C. Soh
T. Walls
D. Weir
D. Birchall

Reversible Cytotoxic Cerebral Edema in Cerebral Fat Embolism

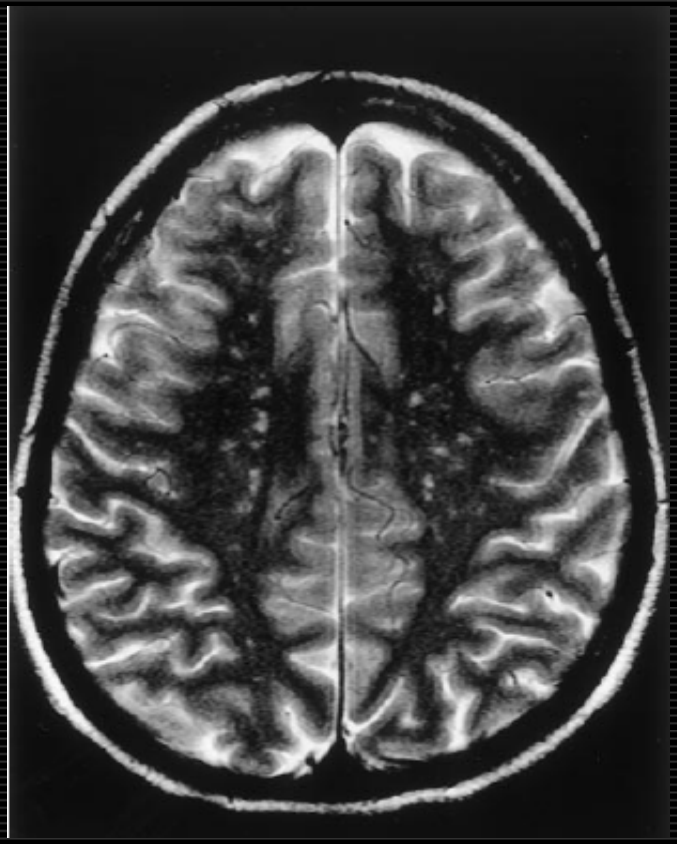
SUMMARY: We present a case of cerebral fat embolism (CFE) that demonstrated evidence of diffuse white matter cytotoxic edema on diffusion-weighted magnetic resonance imaging, in addition to punctate hyperintensities on T2-weighted and diffusion-weighted imaging. The case suggests that CFE represents a combination of occlusive arteriolar disease and secondary neurotoxicity.

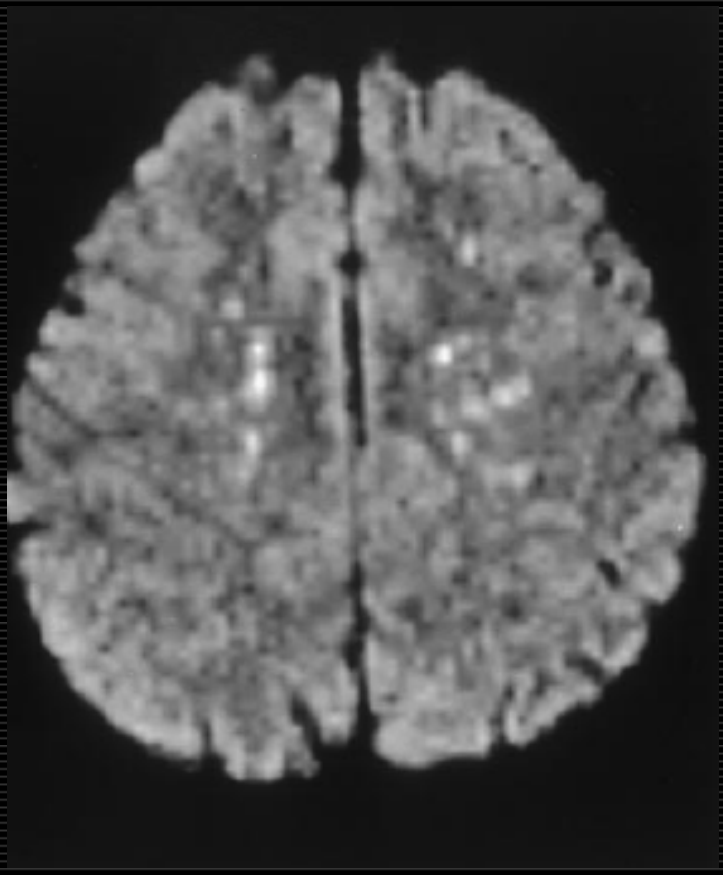
AJNR Am J Neuroradiol 27:620–23 | Mar 2006 | www.ajnr.org

Early Diagnosis of Cerebral Fat Embolism Syndrome by Diffusion-Weighted MRI (Starfield Pattern)

P.M. Parizel, H.E. Demey, G. Veeckmans, F. Verstreken, P. Cras, P.G. Jorens and A.M. De Schepper

Stroke 2001;32;2942-2944



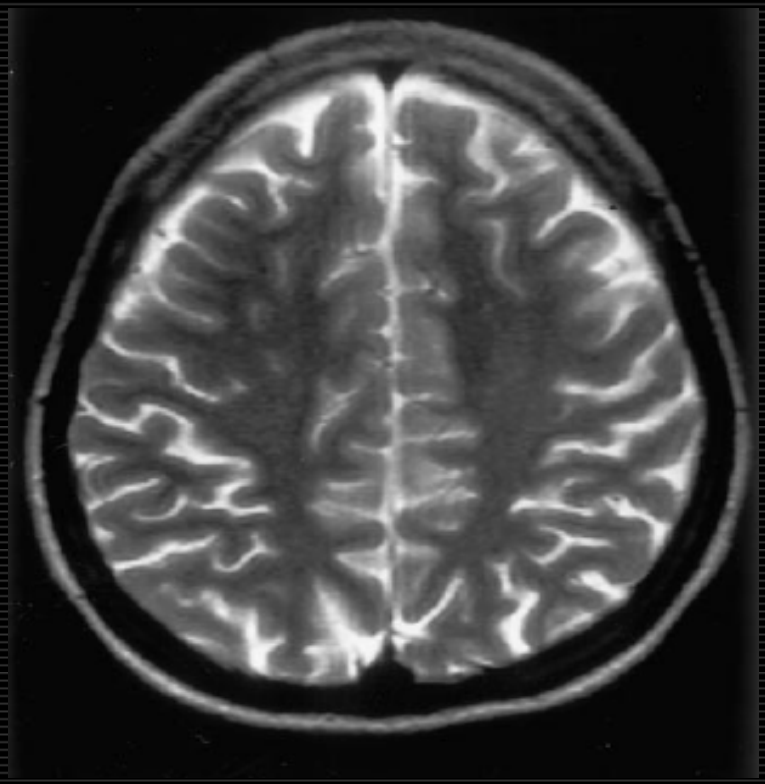


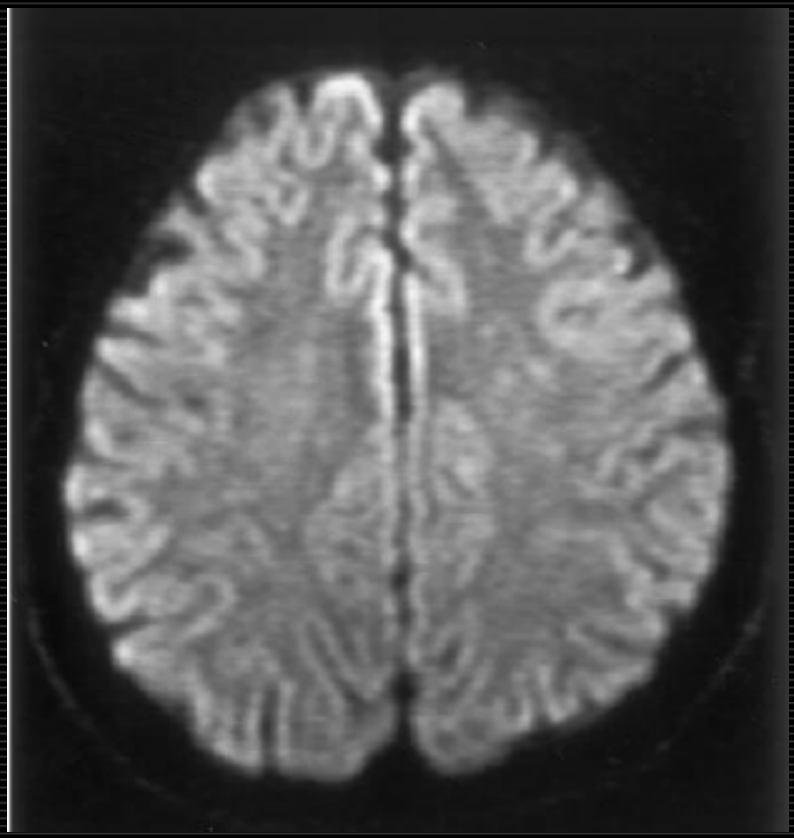
Early Diagnosis of Cerebral Fat Embolism Syndrome by Diffusion-Weighted MRI (Starfield Pattern)

P.M. Parizel, H.E. Demey, G. Veeckmans, F. Verstreken, P. Cras, P.G. Jorens and A.M. De Schepper

Stroke 2001;32;2942-2944

4 SEMANAS



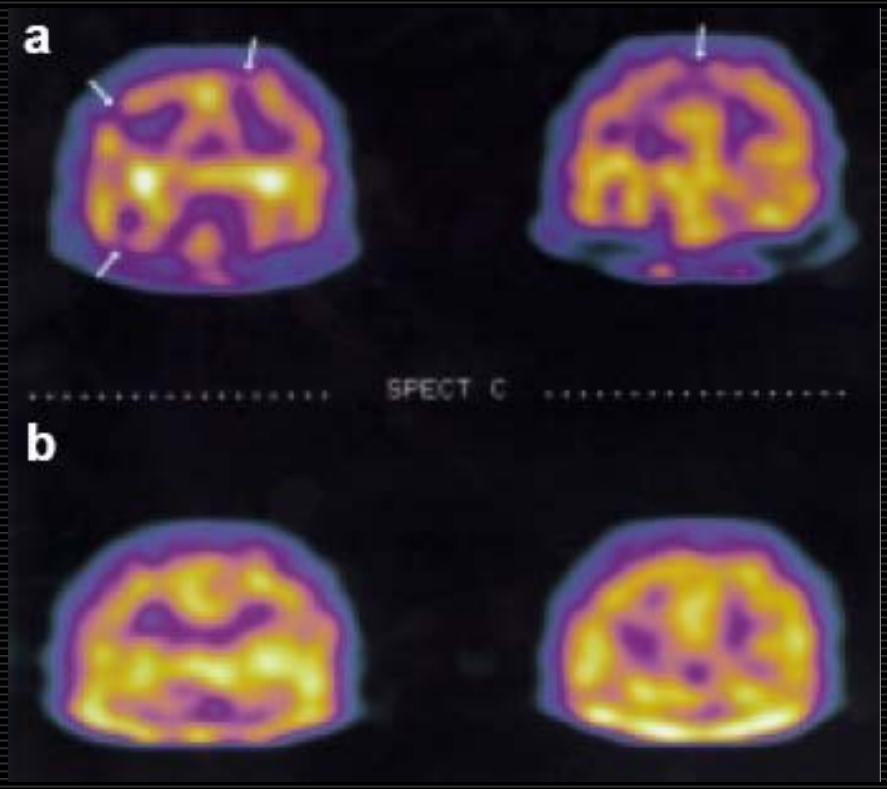


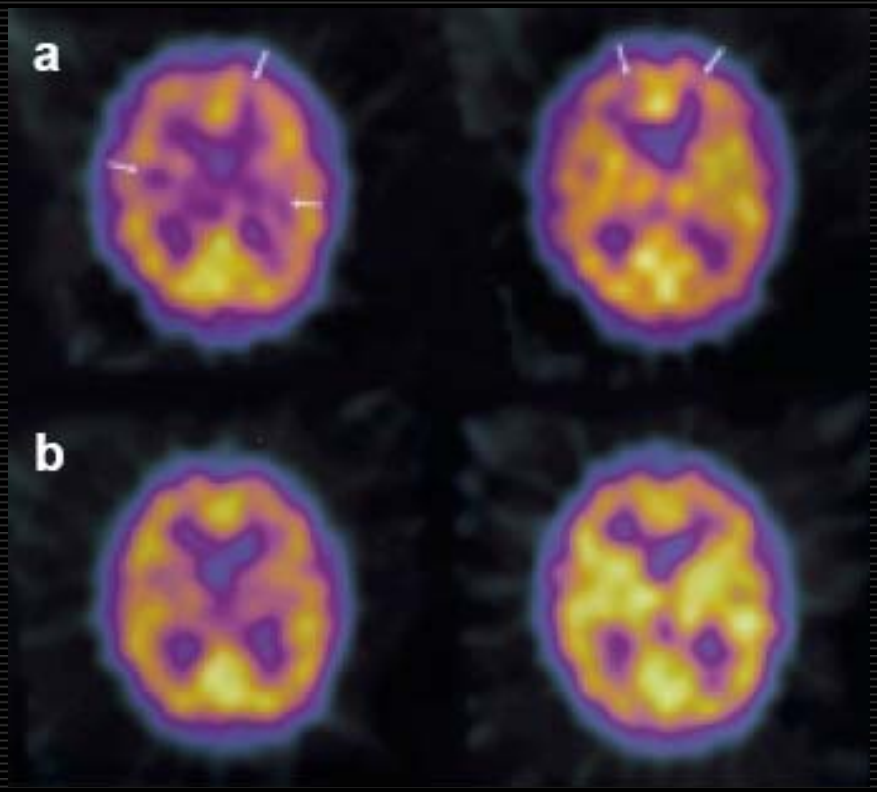
Utilidad del SPECT en la embolia grasa cerebral

T.M. TOMASA IRRIGUIBLE, X. SARMIENTO MARTÍNEZ, R.M. CATALÁN IBARS,
M. FRAILE LÓPEZ-AMOR*, P. TORRABADELLA DE REYNOSO Y M. SOLER OBRADORS

Servicio de Medicina Intensiva. *Servicio de Medicina Nuclear. Hospital Universitari Germans Trias i Pujol. Badalona. Barcelona.

Med Intensiva 2000; 24: 233-236





Trazador: exametazima o HMPAO

Disfunción cardiaca SEG

Taquicardia sinusal 100%
Depresión segmento ST
Aplananamiento onda T
Bloqueos AV
Bloqueos de rama

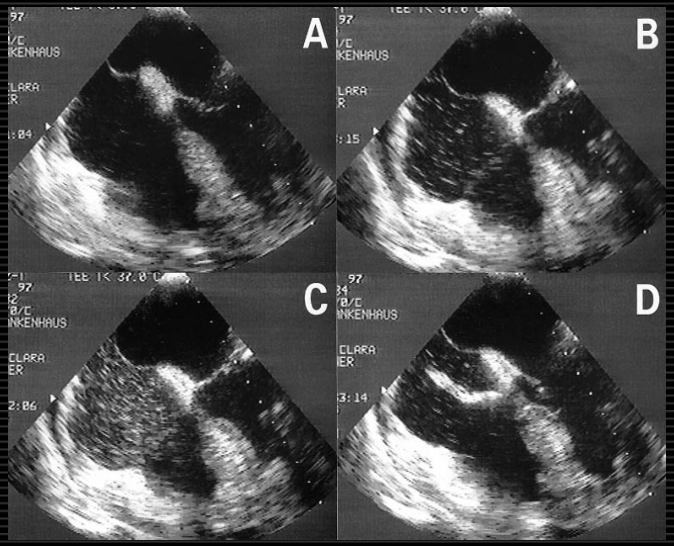
Arch Orthop Trauma Surg (2000) 120:53–58

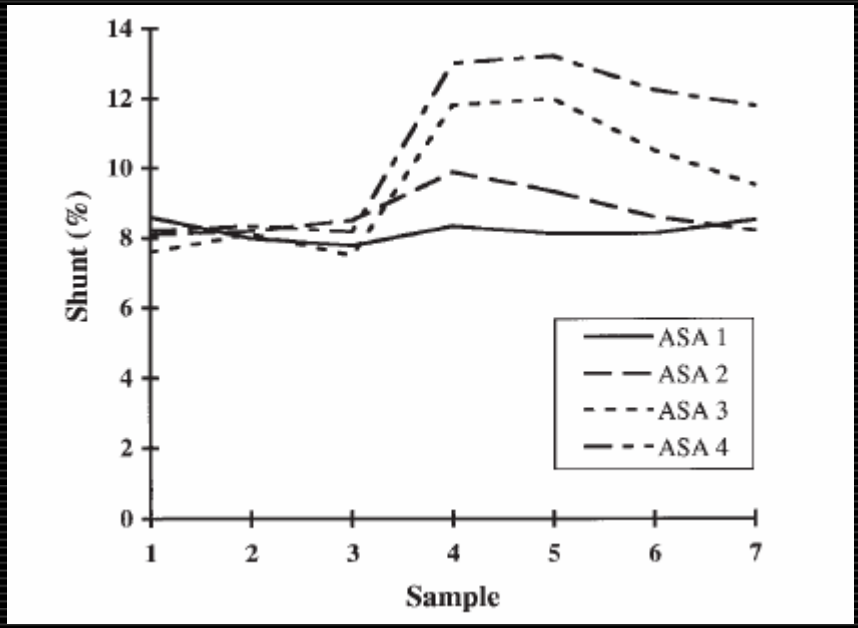
ORIGINAL ARTICLE

R. P. Pitto · J. Blunk · M. Kößler

Transesophageal echocardiography and clinical features of fat embolism during cemented total hip arthroplasty

A randomized study in patients with a femoral neck fracture





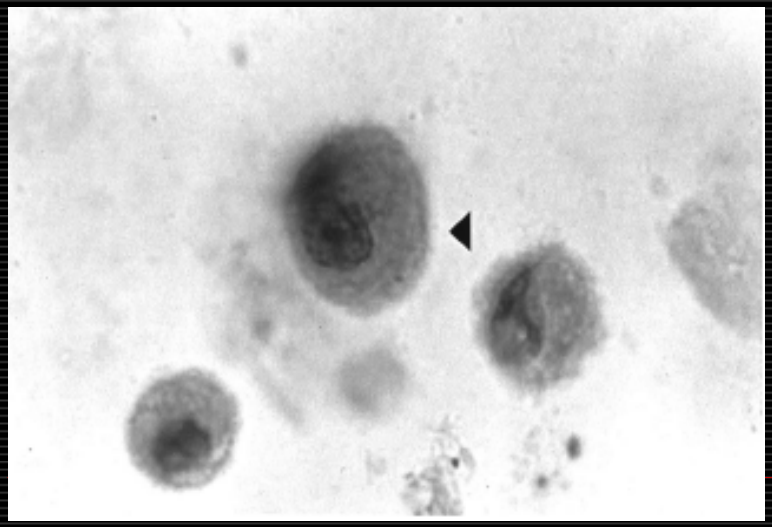
DIAGNÓSTICO

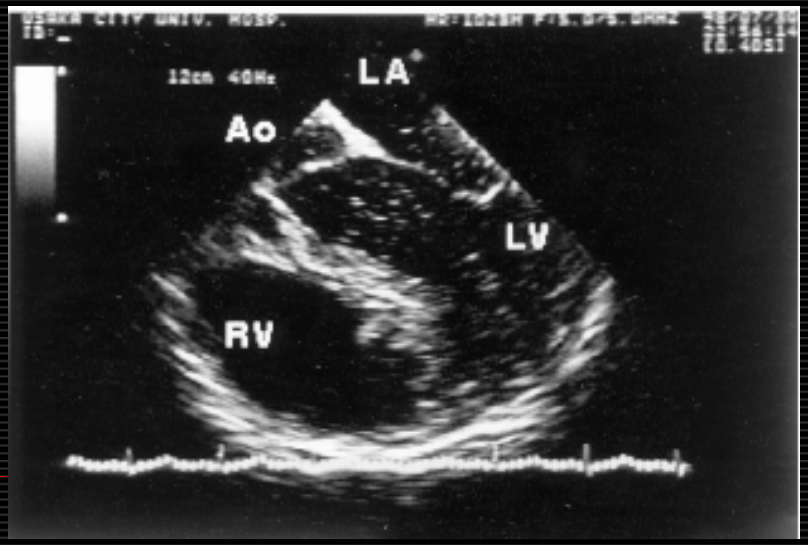
A Case of Fatal Paradoxical Fat Embolism Syndrome Detected by Intraoperative Transesophageal Echocardiography

Nobutaka Kariya, MD*, Mitsuo Shindoh, MD*, Yukako Hayashi, MD*, Masato Nakasuji, MD*, Shinichi Nishi, MD†, Kiyonobu Nishikawa, MD*, and Akira Asada, MD*

*Department of Anesthesiology and Intensive Care Medicine, and †Intensive Care Unit, Osaka City University Medical School, Osaka, Japan

Anesth Analg 2001;92:688–9



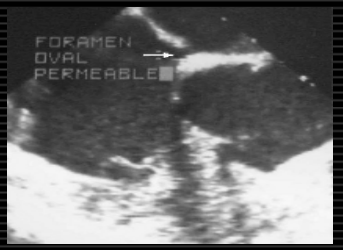


FOP

- 25% población
- 10% embolismos paradójicos
- Asociaciones:
  - Ictus criptogenético
  - EPOC grave 70%
  - OSAS 69%
  - Migraña 60%
  - Amnesia global 55%
  - Embolia grasa 40%
  - Enfermedad descompresiva
- EKG:
  - P bifásicas en DIII
  - BRDHH
  - Patrón de "croquetage"[1]
  - ACxFA paroxística

Diagnóstico:

- DTC
  - Contraste salino
  - Criterios de Jauss[2]
  - Dupplex
  - Power
- ETT
- ETE- Criterios de Cabanes[3]



Rango: 1-22 mm

1.Cohen et al.Can J Card 2000
2.Serena et al.Cerebrovascul Dis 00
3.Cabanes et al.J Am Soc Echocardiogr 02

Patent Foramen Ovale Closure Before Orthopedic Trauma Surgery to Reduce Risk of Recurrent Systemic Fat Embolism

Paul Leeson, PhD, MRCP, Ahmed Al-Mousawi, MB, Jonathan Timperley, MRCP, Andrew R. Mitchell, MRCP, Keith Willett, FRCS, Neil Wilson, FRCP, and Oliver J. Ormerod, FRCP

J Trauma. 2007.

Endovascular Closure of a Patent Foramen Ovale in the Fat Embolism Syndrome

Changes in the Embolic Patterns as Detected by Transcranial Doppler

Alejandro M. Forteza, MD; Alejandro Rabinstein, MD; Sebastian Koch, MD; Gregory Zych, DO; Jay Chandar, MD; Jose G. Romano, MD; Iszet Campo Bustillo, MD

Arch Neurol. 2002;59:455-459

Cierre percutáneo de foramen oval permeable en el síndrome platipnea-ortodesoxia

José R. Ortega Trujillo[a], Javier Suárez de Lezo Herreros de Tejada[a], Antonio García Quintana[a], Francisco Melián Nuez[a], Raquel Rodríguez Delgado[b] y Alfonso Medina Fernández-Aceytuno[a]

Rev Esp Cardiol. 2006;59(1):78-81

Percutaneous Closure of Patent Foramen Ovale in Patients With Paradoxical Embolism

Long-Term Risk of Recurrent Thromboembolic Events

Stephan Windecker, MD; Andreas Wahl, MD; Tushar Chatterjee, MD; Ali Garachemani, MD; Franz R. Eberli, MD; Christian Seiler, MD; Bernhard Meier, MD

Circulation 2000;101;893-898

Endovascular Closure of a Patent Foramen Ovale in the Fat Embolism Syndrome

Changes in the Embolic Patterns as Detected by Transcranial Doppler

Alejandro M. Forteza, MD; Alejandro Rabinstein, MD; Sebastian Koch, MD; Gregory Zych, DO; Jay Chandar, MD; Jose G. Romano, MD; Iszet Campo Bustillo, MD

Arch Neurol. 2002;59:455-459

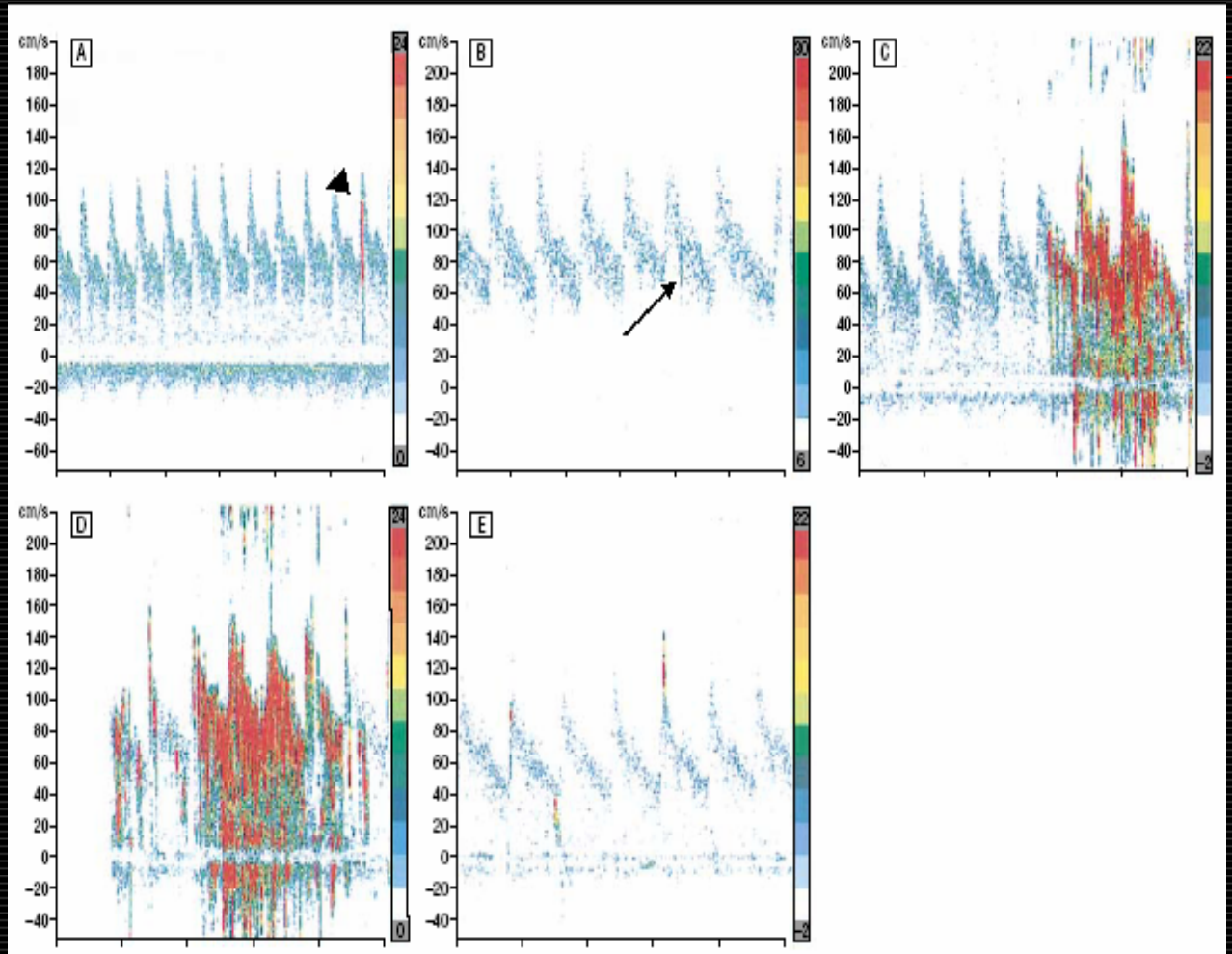
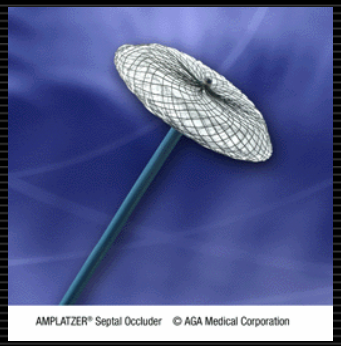
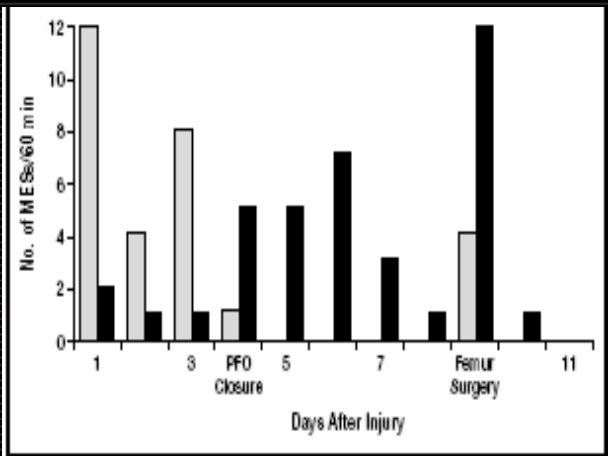


Figure 1. Microembolic signal (MES) characteristics. A, Large MES with an intensity greater than 12 dB (arrowhead) detected in the left middle cerebral artery. B, Small MES of less than 12 dB (long arrow) detected in the left middle cerebral artery. C-E, Multiple MES over the right middle cerebral artery observed within seconds of intravenous injection of aerated, agitated isotonic sodium chloride solution.



AMPLATZER® Septal Occluder © AGA Medical Corporation



Disfunción hematológica SEG

- Petequias
  - Tronco anterior
  - Patogneumónico o síntoma 2°
  - Oclusión de capilares dérmicos
  - Incremento de fragilidad capilar
  - 50%
  - Inicio 2°-3° dia
  - Resolución 5-7 dias
- Heparina
- Alteraciones de la coagulacion
- D-dímero
- Trombocitopenia
- Anemia

EMBOLIA GRASA Y AUTOTRASFUSIÓN

Fat Elimination from Autologous Blood

Michael Booke, MD, PhD, Hugo Van Aken, MD, PhD, Martin Storm, Florian Fritzsche, Stefan Wirtz, and Frank Hinder, MD, PhD

Klinik und Poliklinik für Anästhesiologie und operative Intensivmedizin, University of Münster, 48129 Münster, Germany

Anesth Analg 2001;92:341–3

Fat Elimination During Intraoperative Autotransfusion: An *In Vitro* Investigation

Michael Booke, MD*, Manfred Fobker, MD‡, Detlef Fingerhut, MD*, Martin Storm*, Yves Mortlemans, MD†, and Hugo Van Aken, MD, PhD*

Departments of *Anesthesiology and Intensive Care and †Transfusionsmedicine and Immune-Hematology, and ‡Institute of Clinical Chemistry and Laboratory Medicine, University of Münster, Münster, Germany

Anesth Analg 1997;85:959–62

Fat embolism and autologous blood transfusions in orthopaedic surgery

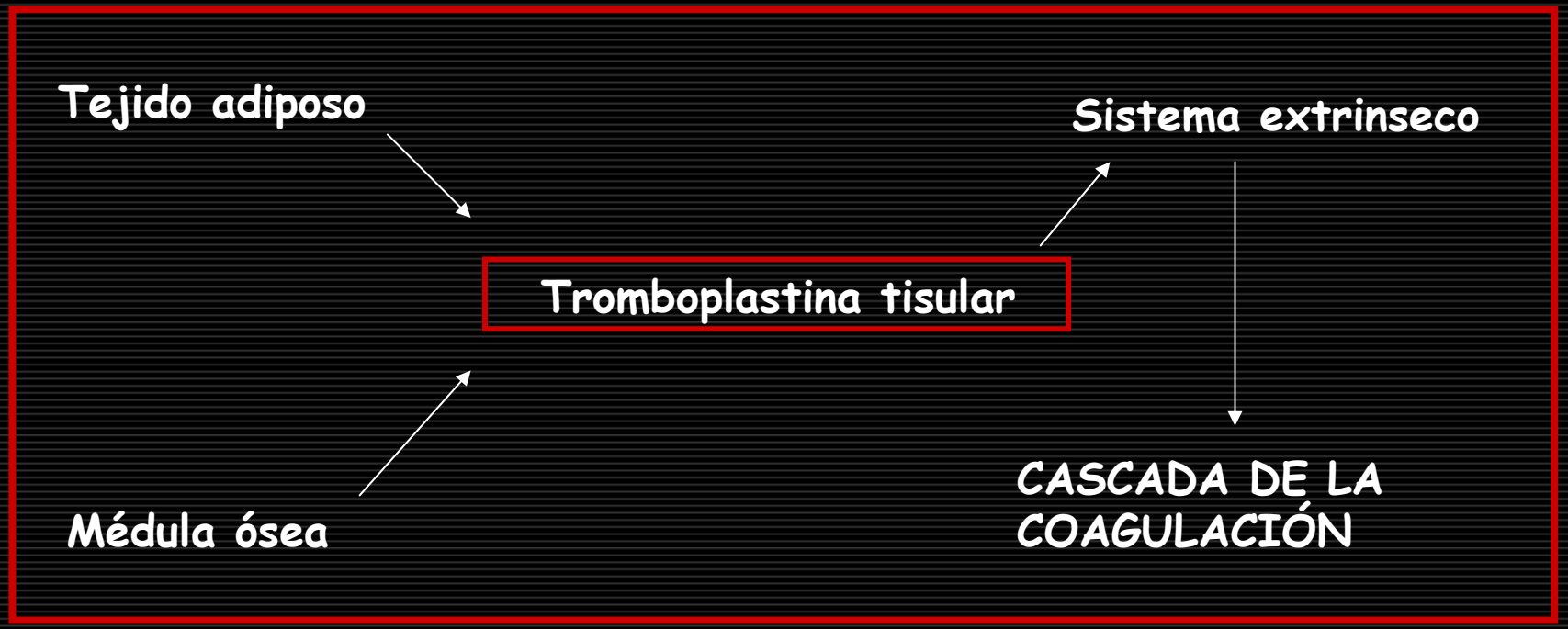
Magnus Jacobsson[a,*], Anders Bengtsson[b]

Current Anaesthesia & Critical Care (2004) 15, 87–93

Editorials

Fat embolism and postoperative coagulopathy

Robert J. Byrick MD



Can J Anaesth. 2001 Jul-Aug;48(7):618-21

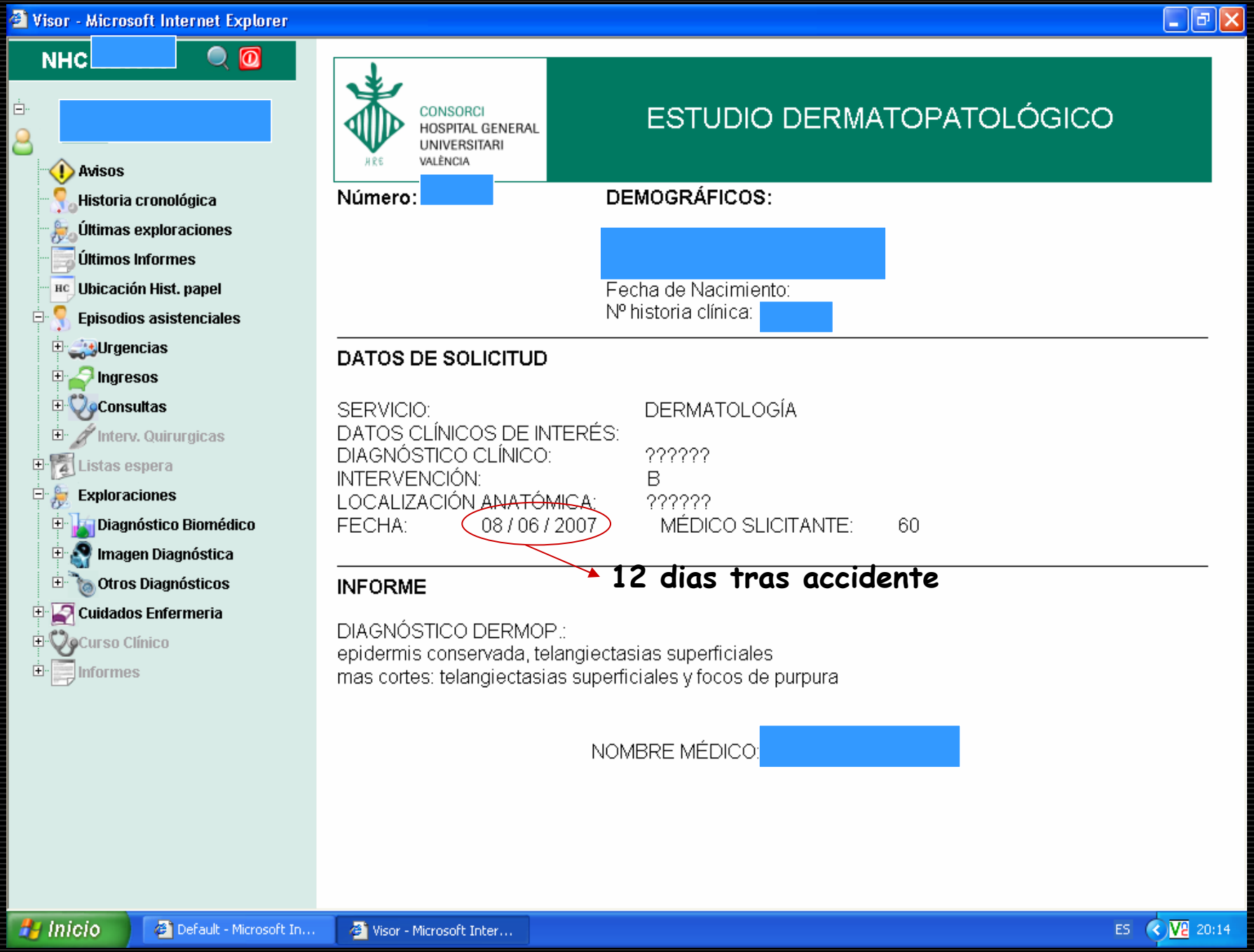

Visor - Microsoft Internet Explorer
NHC
Avisos
Historia cronológica
Últimas exploraciones
Últimos Informes
Ubicación Hist. papel
Episodios asistenciales
Urgencias
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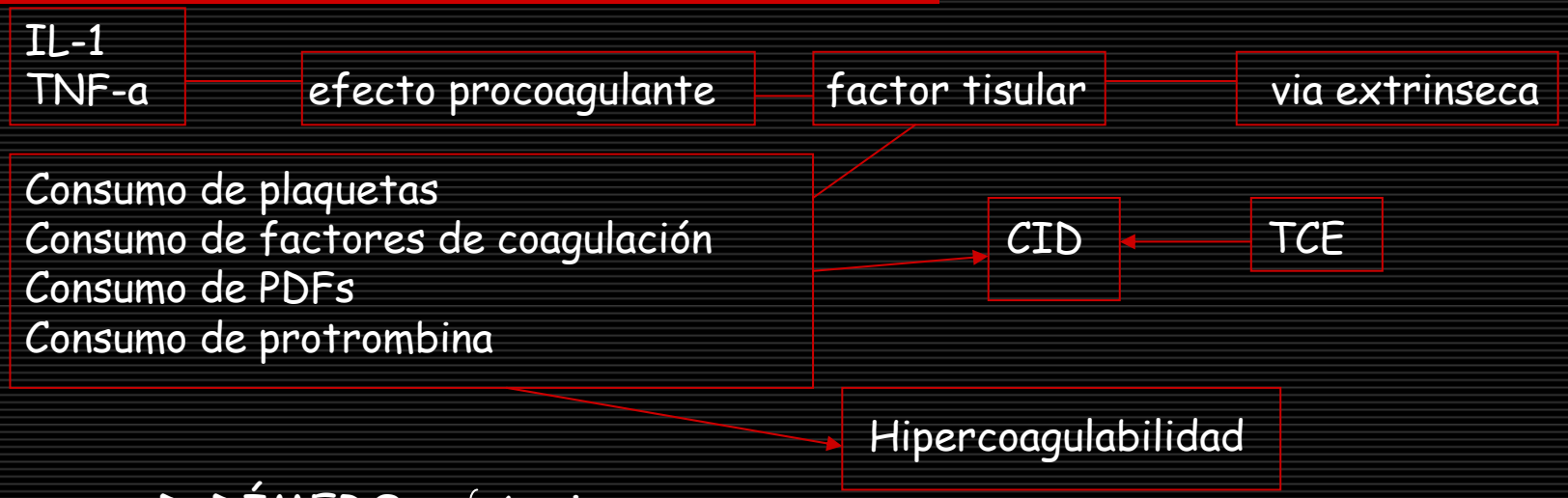
Imagen Diagnóstica
Otros Diagnósticos
Cuidados Enfermeria
Curso Clínico
Informes
CONSORCI
HOSPITAL GENERAL
UNIVERSITARI
VALÈNCIA
ESTUDIO DERMATOPATOLÓGICO
Número:
DEMOGRÁFICOS:
Fecha de Nacimiento:
Nº historia clínica:
DATOS DE SOLICITUD
SERVICIO: DERMATOLOGÍA
DATOS CLÍNICOS DE INTERÉS:
DIAGNÓSTICO CLÍNICO: ??????
INTERVENCIÓN: B
LOCALIZACIÓN ANATÓMICA: ??????
FECHA: 08 / 06 / 2007
MÉDICO SLICITANTE: 60
12 dias tras accidente
INFORME
DIAGNÓSTICO DERMOP.:
epidermis conservada, telangiectasias superficiales
mas cortes: telangiectasias superficiales y focos de purpura
NOMBRE MÉDICO:
Inicio
Default - Microsoft In...
Visor - Microsoft Inter...
ES
20:14

Disfunción hematológica en el paciente politraumático



D-DÍMERO:
- Ancianos
- Estados inflamatorios
- Intervenciones recientes

Disfunción plaquetar:
- Adhesividad plaquetar
- Agregación plaquetar
- Gránulos alfa y beta
- Receptores de membrana

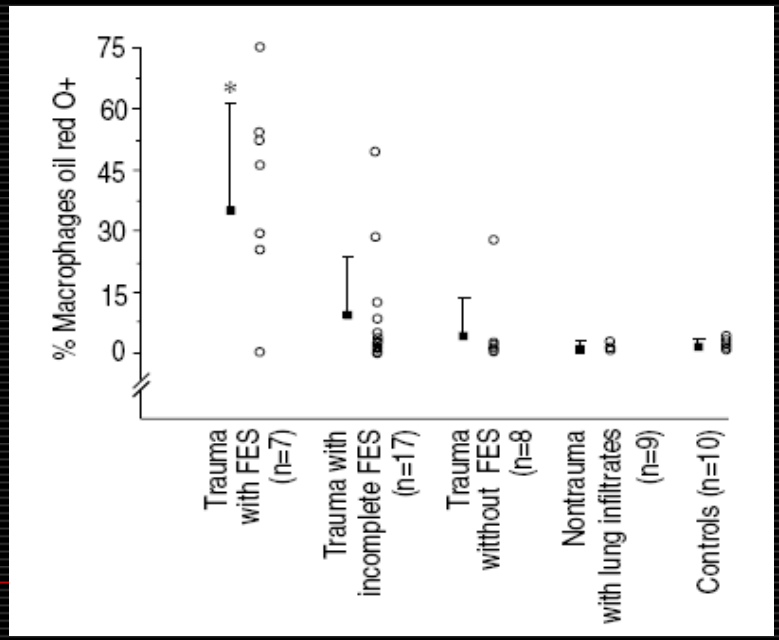
Laboratorio

- Ninguna específica
- Lipasa sérica
- Fosfolipasa
- Endotelina-1
- BAL
- Biopsia transbronquial
- Depósitos grasos en esputo/ orina/sangre
- Hipocalcemia

Role of bronchoalveolar lavage in the diagnosis of fat embolism syndrome

N. Roger*, A. Xaubet*, C. Agustí*, E. Zabala**, E. Ballester*, A. Torres*, C. Picado*, R. Rodriguez-Roisin*

Eur Respir J, 1995, 8, 1275–1280



Intensive Care Med (2006) 32:116–123
DOI 10.1007/s00134-005-2868-x

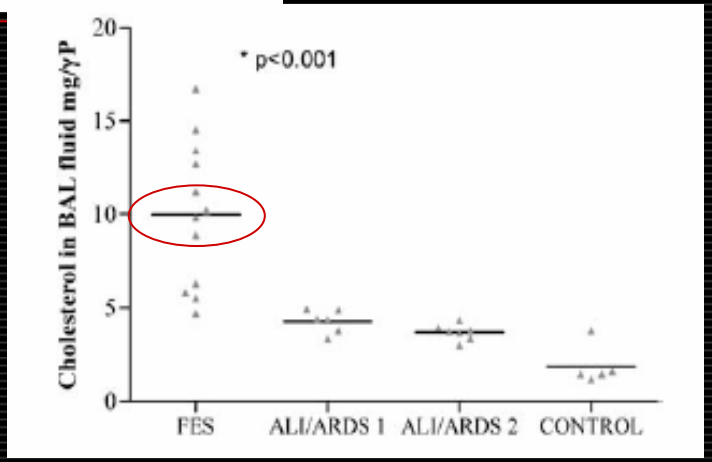
ORIGINAL

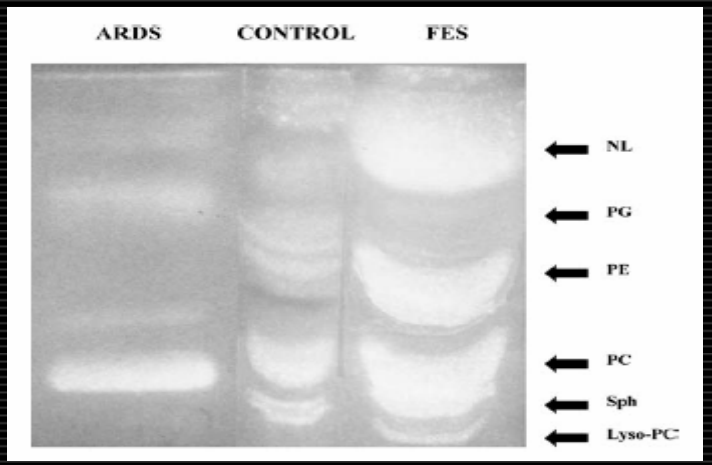
Georgia Karagiorga
George Nakos
Eftychia Galiatsou
Marilena E. Lekka

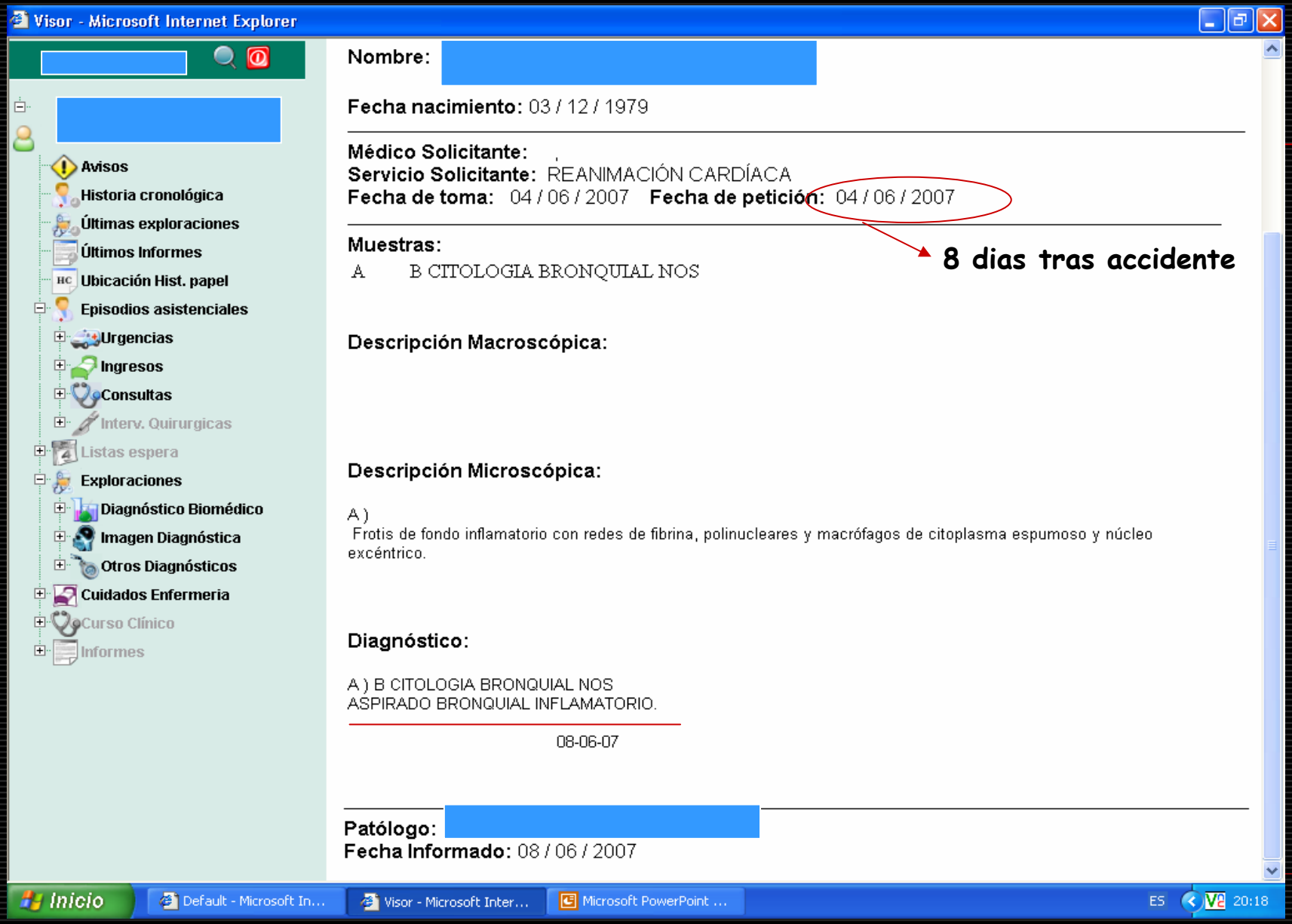
Biochemical parameters of bronchoalveolar lavage fluid in fat embolism

	FES	ALI/ARDS 1	ALI/ARDS 2	Control
Total phospholipids (μg/ml BAL)	3.7±1.2*,**	2.7±0.9*	2.46±0.8*	2.28±0.5
Phosphatidylcholine (%)	53.7±8.3**	50.9±6.6**	44.6±6**	76±5.2
Phosphatidylglycerol (%)	5.1±1.8**	5.3±1.1**	4.2±1.9**	8.7±2.1
Sphingomyelin (%)	13.7±5.9**	15.7±5.9**	11.4±6.7**	2.3±0.8
Phosphatidylethanolamine (%)	5.7±2.2	6.9±1.2	4.6±1.4	4.2±0.6
Phosphaditylserine (%)	6.9±2.9	7.7±2.8	6.7±3.1	4.4±0.6
Phosphatidylinositol (%)	7.5±2.2**	8.3±3.2**	9.1±2.5**	4.1±1.1
Lyso-phosphatidylcholine (%)	2.8±1.6*,**	1.9±1.5**	0.8±0.9***,***	–
Total protein (μg/ml)	1911±1395*,**	876.7±276*,**	1210±1250**	137±32
PAF (pg/9 ml)	1089±364*,**	189±38**	27±19	28±49
PAF-AcH (nmol m^{-1} min^{-1})	0.06±0.04*,**	5.45±3.1**	8.9±4.5**	1.2±0.6
PLA_2 (nmol ml^{-1} min^{-1})	0.51±0.13**	0.65±0.32**	0.9±0.3**	0.01±0.01
Esters (μg/μphospholipid) 0.035±0.07	0.44±0.22*,**	0.015±0.01*	0.012±0.026*	
Cholesterol (μg/μg phospholipid)	10.2±5.3*,**	4.38±0.75*,**	3. 7±0.5*,**	1.97±1.22

*$p \leq 0.05$ FES vs. ALI/ARDS group 1 and/or 2, **FES vs. Control, ***ALI/ARDS groups 1 vs. 2






Visor - Microsoft Internet Explorer
Avisos
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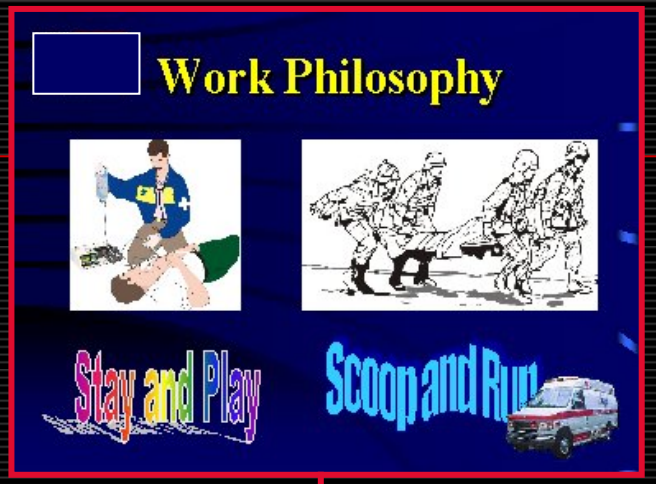
Interv. Quirurgicas
Listas espera
Exploraciones
Diagnóstico Biomédico
Imagen Diagnóstica
Otros Diagnósticos
Cuidados Enfermeria
Curso Clínico
Informes
Nombre:
Fecha nacimiento: 03 / 12 / 1979
Médico Solicitante:
Servicio Solicitante: REANIMACIÓN CARDÍACA
Fecha de toma: 04 / 06 / 2007 Fecha de petición: 04 / 06 / 2007
Muestras:
A B CITOLOGIA BRONQUIAL NOS
8 dias tras accidente
Descripción Macroscópica:
Descripción Microscópica:
A)
Frotis de fondo inflamatorio con redes de fibrina, polinucleares y macrófagos de citoplasma espumoso y núcleo excéntrico.
Diagnóstico:
A) B CITOLOGIA BRONQUIAL NOS
ASPIRADO BRONQUIAL INFLAMATORIO.
08-06-07
Patólogo:
Fecha Informado: 08 / 06 / 2007
Inicio
Default - Microsoft In...
Visor - Microsoft Inter...
Microsoft PowerPoint ...
ES
20:18

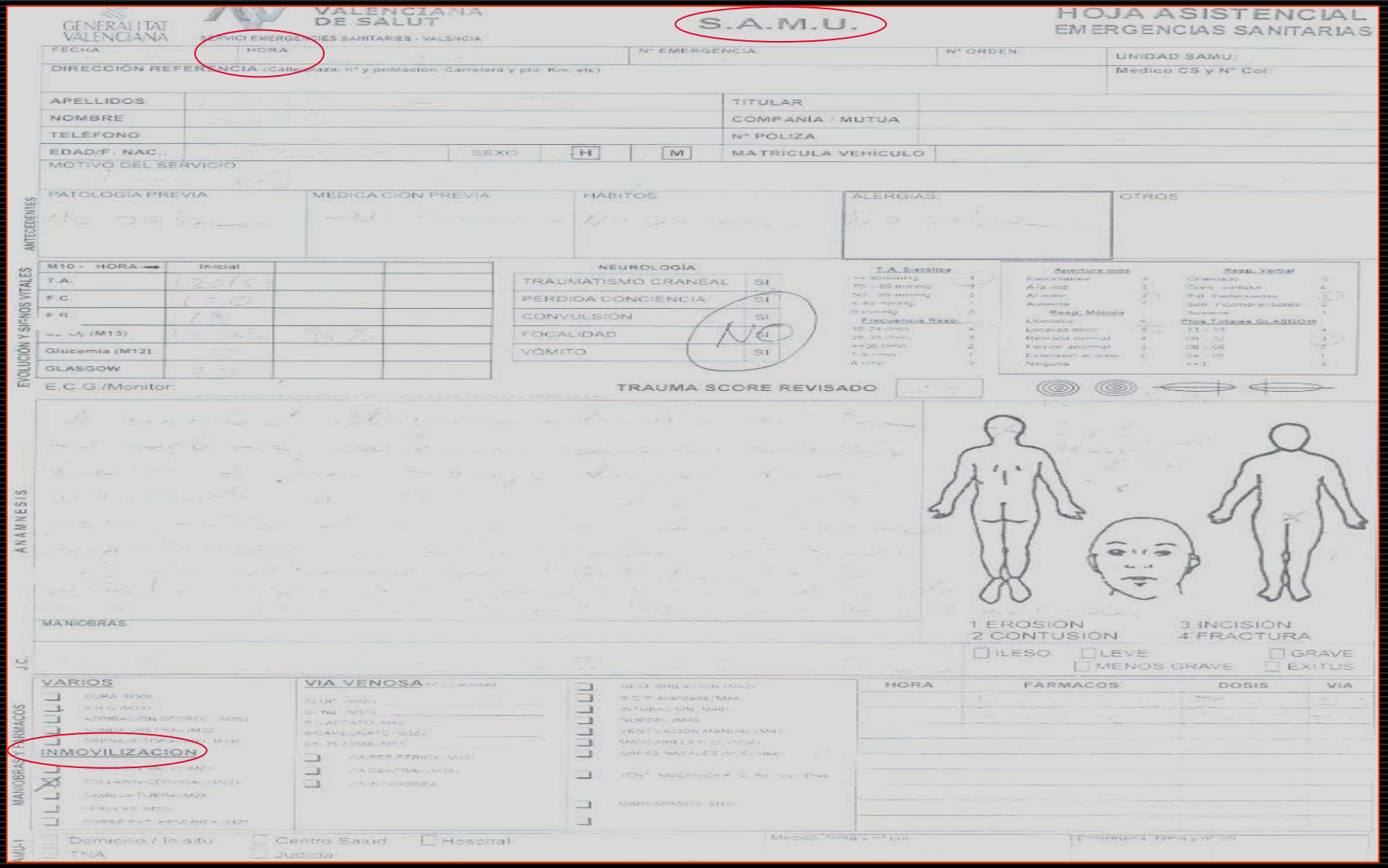
PROFILAXIS SEG

- Sildenafilo[1]
- Oxigenación hiperbárica[2]
- Heparina
- Etanol
- Dextrosa
- Corticoides
- Seroalbúmina
- Inmovilización precoz
- Evitar hipovolemia

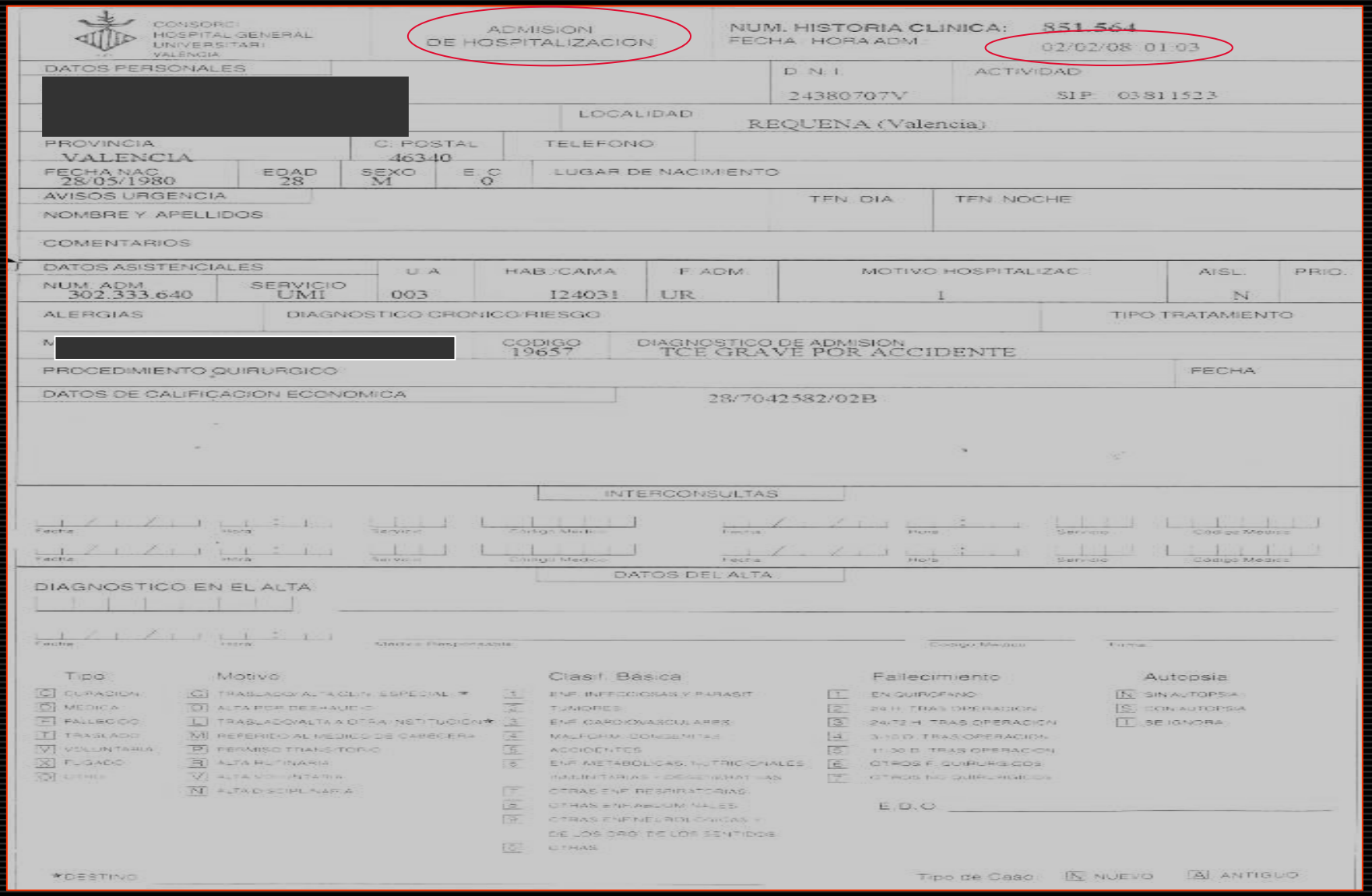


1.Sildenafil prevents cardiovascular changes after bone marrow fat embolization in sheep Anesthesiology 2007 Jul; 107(1): 75-81
2. Hyperbaric oxygenation in fluid microembolism.Neurol Res 2007 Mar;29(2):156-61

STAY AND PLAY vs SCOOP AND RUN



STAY AND PLAY vs SCOOP AND RUN



CONSORCI HOSPITAL GENERAL UNIVERSITARI VALENCIA

ADMISION DE HOSPITALIZACION

NUM. HISTORIA CLINICA: 851.564
FECHA HORA ADM.: 02/02/08 01:03

DATOS PERSONALES

[redacted]

D.N.I.: 24380707V
ACTIVIDAD
SIP: 03811523

LOCALIDAD: REQUENA (Valencia)
PROVINCIA: VALENCIA
C. POSTAL: 46340
TELEFONO:
FECHA NAC.: 28/05/1980
EDAD: 28
SEXO: M
E. C.: 0
LUGAR DE NACIMIENTO:
AVISOS URGENCIA
NOMBRE Y APELLIDOS:
TFN. DIA:
TFN. NOCHE:
COMENTARIOS:

DATOS ASISTENCIALES

NUM. ADM.	SERVICIO	U.A.	HAB./CAMA	F. ADM.	MOTIVO HOSPITALIZAC.	AISL.	PRIO.
302.333.640	UMI	003	I24031	UR	1	N	

ALERGIAS
DIAGNOSTICO CRONICO/RIESGO
TIPO TRATAMIENTO
N[redacted]
CODIGO: 19657
DIAGNOSTICO DE ADMISION: TCE GRAVE POR ACCIDENTE
PROCEDIMIENTO QUIRURGICO
FECHA
DATOS DE CALIFICACION ECONOMICA
28/7042582/02B

INTERCONSULTAS

Fecha	Hora	Servicio	Codigo Medico	Fecha	Hora	Servicio	Codigo Medico
Fecha	Hora	Servicio	Codigo Medico	Fecha	Hora	Servicio	Codigo Medico

DATOS DEL ALTA

DIAGNOSTICO EN EL ALTA

Fecha | Hora | Medico Responsable | Codigo Medico | Firma

Tipo:
- C CURACION
- D MEDICA
- F FALLECIDO
- T TRASLADO
- V VOLUNTARIA
- X FUGADO
- O OTRAS

Motivo:
- C TRASLADO ALTA CLIN. ESPECIAL *
- D ALTA POR DESHAUCIO
- L TRASLADO/ALTA A OTRA INSTITUCION *
- M REFERIDO AL MEDICO DE CABECERA
- P PERMISO TRANSITORIO
- R ALTA RUTINARIA
- V ALTA VOLUNTARIA
- N ALTA DISCIPLINARIA

Clasf. Basica:
- 1 ENF. INFECCIOSAS Y PARASIT.
- 2 TUMORES
- 3 ENF. CARDIOVASCULARES
- 4 MALFORM. CONGENITAS
- 5 ACCIDENTES
- 6 ENF. METABOLICAS, NUTRICIONALES, ALIMENTARIAS Y DESESTIMAT. INM.
- 7 OTRAS ENF. RESPIRATORIAS
- 8 OTRAS ENF. ABDOMINALES
- 9 OTRAS ENF. NEUROLOGICAS Y DE LOS OAG. DE LOS SENTIDOS
- 0 OTRAS

Fallecimiento:
- 1 EN QUIROFANO
- 2 24 H. TRAS OPERACION
- 3 24/72 H. TRAS OPERACION
- 4 3/10 D. TRAS OPERACION
- 5 11/30 D. TRAS OPERACION
- 6 OTROS F. QUIRURGICOS
- 7 OTROS NO QUIRURGICOS

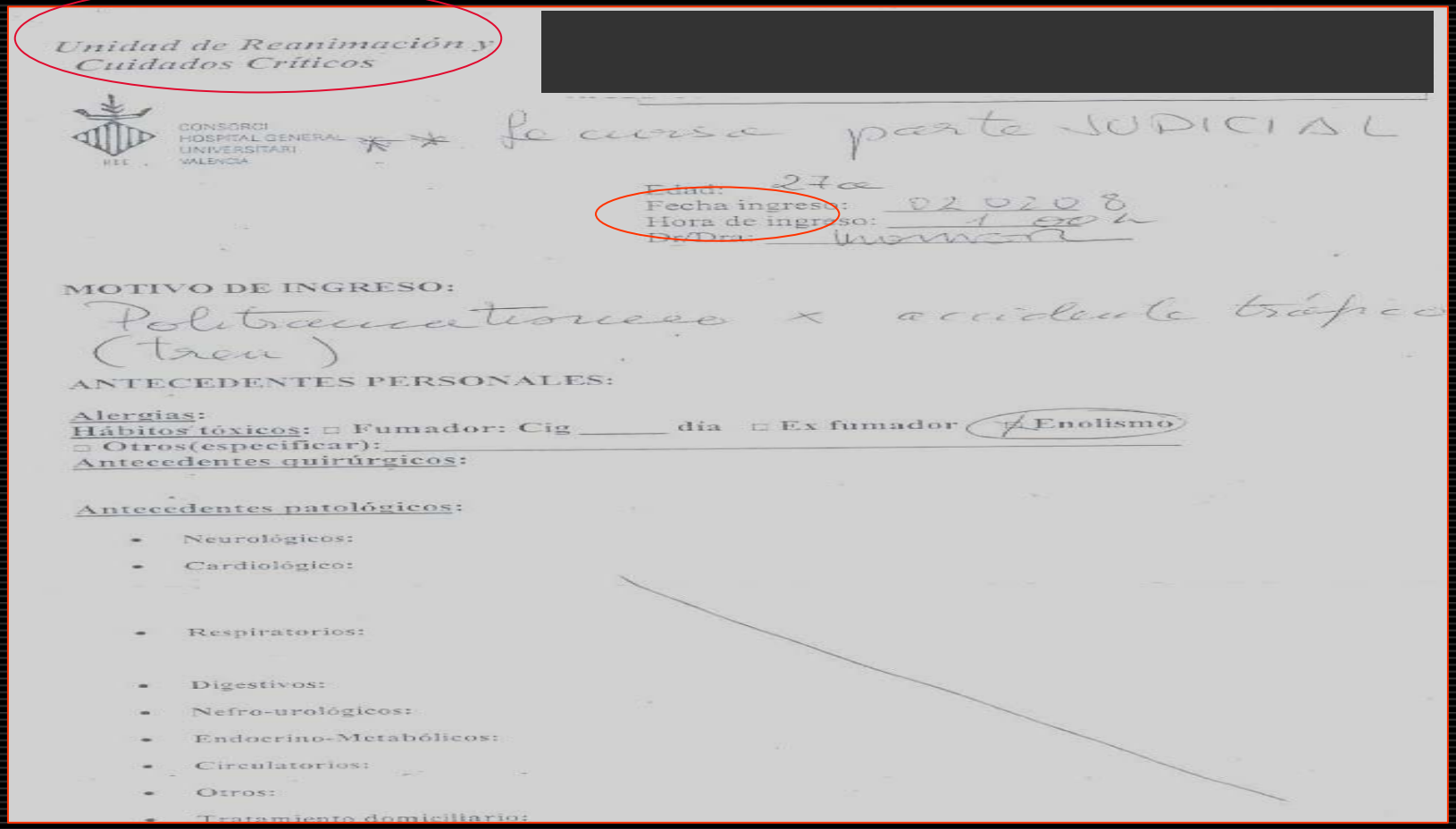
E.D.O.

Autopsia:
- N SIN AUTOPSIA
- S CON AUTOPSIA
- I SE IGNORA

*DESTINO

Tipo de Caso: N NUEVO A ANTIGUO

STAY AND PLAY vs SCOOP AND RUN



Unidad de Reanimación y Cuidados Críticos

CONSORCI HOSPITAL GENERAL UNIVERSITARI VALENCIA

[redacted]

Parte JUDICIAL

Edad: 27a
Fecha ingreso: 02/02/08
Hora de ingreso: 1 00 h
Dr/Dra:

MOTIVO DE INGRESO:

Politraumatismos x accidente tráfico (tren)

ANTECEDENTES PERSONALES:

Alergias:
Hábitos tóxicos: □ Fumador: Cig _____ día □ Ex fumador ✓ Enolismo
□ Otros(especificar):
Antecedentes quirúrgicos:

Antecedentes patológicos:

- Neurológicos:
- Cardiológico:
- Respiratorios:
- Digestivos:
- Nefro-urológicos:
- Endocrino-Metabólicos:
- Circulatorios:
- Otros:
- Tratamiento domiciliario:

Corticoides en SEG

- Estabilizadores de mb de granulocitos
- Reduce niveles catecolaminas
- Retardan la agregación plaquetaria
- Inhiben activación del complemento
- Protegen endotelio capilar
- Indicado si alto riesgo entonces metilprednisolona 6 mg/kg/ 8h/ 48 h
- NNT: 7.4

•Prevention of posttraumatic hypoxaemia in isolated lower limb long bone fractures with a minimal prophylactic dose of corticosteroids.
Babalis GA, Yiannakopoulos CK, Karliaftis K,Antonogiannakis E.
Injury mar 2004

•Glucocorticoid induces micro-fat embolism in the rabbit: a scanning electron microscopic study .Journal of Orthopaedic Research. 24(4):675-83, 2006 Apr.

TÉCNICAS EXPERIMENTALES

- ANTAGONISTAS RECEPTOR IL-1
- INHIBIDORES DE CICLOOXIGENASA
- ANTAGONISTAS DEL FACTOR VII
- ANTITROMBINA III
- PROTEINA C ACTIVADA
- POLIMORFISMO GENETICO
- FILTROS VENA CAVA
- ECMO

Prevention of fat embolism syndrome

Timothy White[1], Brad A Petrisor[2], Mohit Bhandari[2]

Injury, Int. J. Care Injured (2006) 375, S59–S67

Technique	Description	Level of evidence/notes
Prediction of susceptibility	Trauma scores	Level 1 Poor positive predictive value.
	Thoracic injury	Level 1 An isolated thoracic injury carries greater risk than isolated femoral fractures, but combined injuries do not raise risk further.
	Pathological fracture	Level 1 Higher risk
	Inflammatory markers	Il-6 level correlates with outcome, but it is not yet clear whether this is independently predictive.
	Genetic predisposition	Certain polymorphisms correlate with outcome, may allow targeted therapy in future
Resuscitation	BP < 90	Observational studies suggest a correlation between BP <90mm Hg on admission and adverse outcome.
	Transfusion requirements	Observational studies have found massive transfusion requirements to be associated with adverse outcomes (eg, multiple organ dysfunction)—however, definitions of massive transfusion remains controversial.
Timing of surgery	Long-bone surgical stabilization within 24 hours	Level 1 Surgical stabilization reduces ARDS rate by factor 5:1 compared with late.
Type of surgery	DCO	Observational and retrospective studies may suggest that damage control orthopedics reduces the insult of initial operative long bone stabilization but level 1 evidence is awaited.
Adjuvants	Prednisolone	Early papers suggest reduction in incidence of respiratory insufficiency but at expense of increased infection rate.
	Heparin	Empirical
	Ethanol	Empirical
	Dextrose	Empirical
Experimental	Specific antibody treatments	Not currently available. Some animal studies exciting and success of APC in sepsis raises possibility of therapeutic options in future.
	Filters	Experimental studies suggest that inferior vena cava filters decrease the embolic load to the heart during femoral nail insertion

Table 2: Summary of recommendations.

PROFILAXIS QUIRÚRGICA

- Técnicas intramedulares aumentan FES.
- Elegir técnicas menos agresivas como yesos o fijadores externos.
- Clavos no fresados.
- Ventilar canal antes de cementar.
- Cemento preparado en vacío.
- Irrigación y aspiración del canal medular pre cementación.

DAMAGE CONTROL
J. TRAUMA. 2000; 49: 969-978

TIPOS DE ENCLAVADOS INTRAMEDULARES

- **CLAVO CADERA TIPO GAMMA:**

Fx proximales de fémur

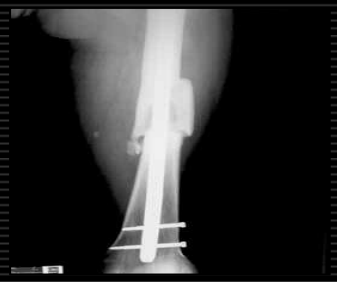
- intertrocantéreas
- pertrocantéreas
- subtrocantéreas

- **CLAVO FÉMUR TIPO GROSSE & KEMPF**

Fx diafisarias de fémur

- **CLAVO RETRÓGRADO:**

Fx supracondíleas de fémur



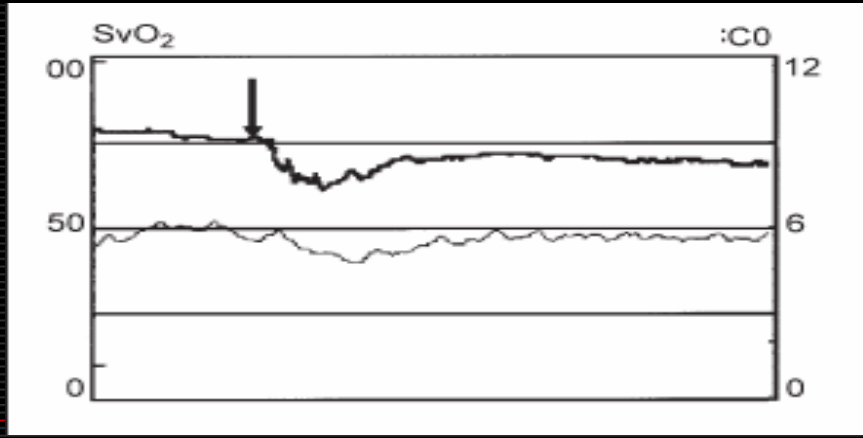
EFECTOS BIOLÓGICOS Y FISIOLÓGICOS DEL FRESADO MEDULAR

- Alteración del sistema arterial medular
- ↑ presión intramedular durante el fresado
- No incremento durante la colocación del enclavado medular
- Depende de la velocidad de la colocación
- ↑ volumen de 20-50 ml / 15 mm de diámetro
- Daños vasculares transitorios a nivel pulmonar SÓLO si existe previamente lesión.
- Generación de calor
- Incremento liberación de factores de crecimiento
- Efecto osteogénico del fresado

El enclavado medular sin fresar

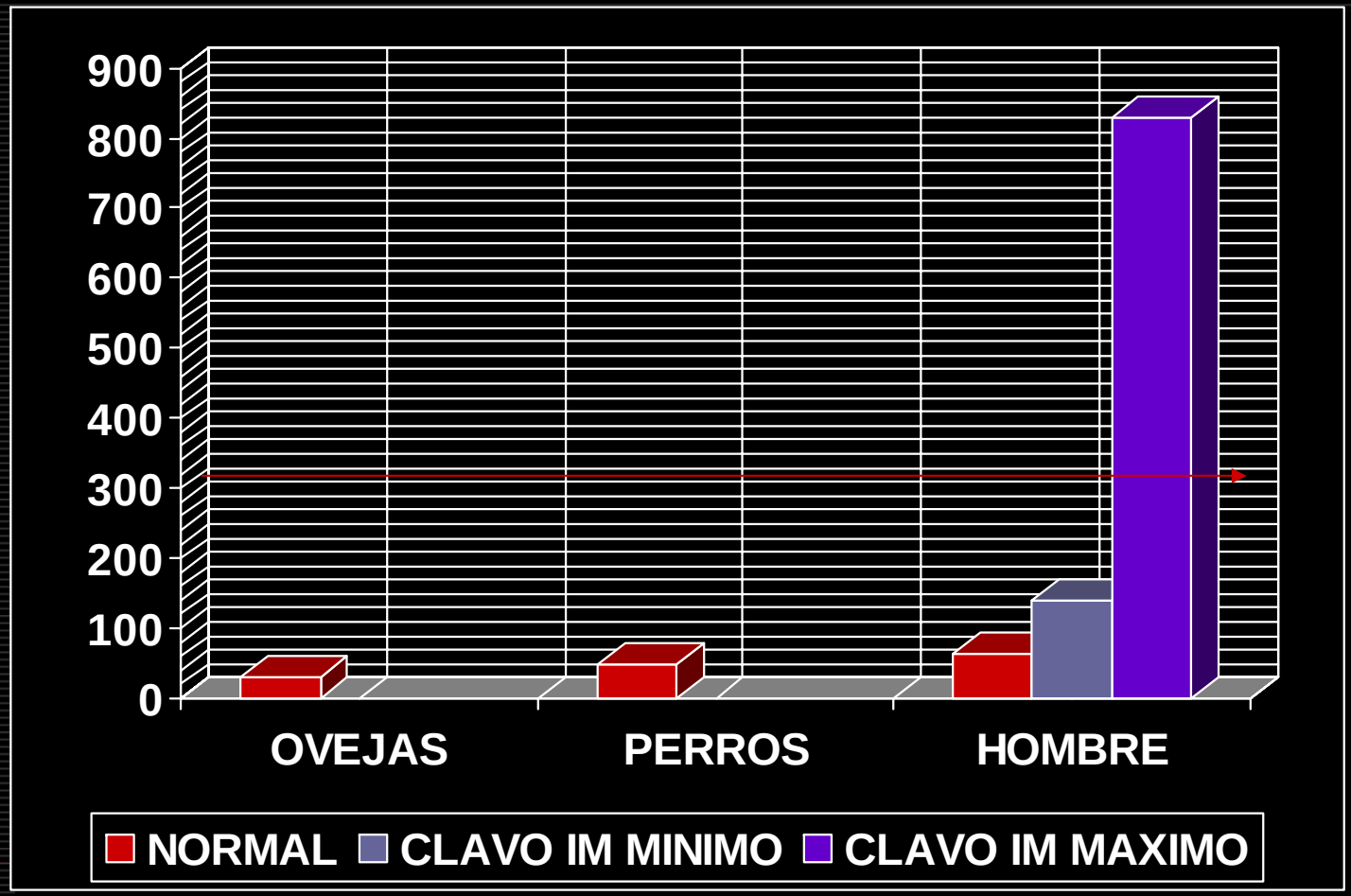
Unreamed intramedullary nailing

DÍEZ ULLOA, M. A., y COUCEIRO, J.
REVISTA DE ORTOPEDIA Y TRAUMATOLOGÍA
Volumen 45, pp 299-306



Pape H.C. et al.J Bone Joint Surg (Br) 2007;89-B:1421-6

NIVELES PRESIÓN INTRAMEDULAR ESTUDIOS EXPERIMENTALES



¿FIJACIÓN PRECOZ O TARDÍA?

	INCIDENCIA	
	SEG	SDRA
1970 Tracción y yeso	20%	-
1977 Técnicas de fijación intramedulares[1,2,3]	5%	7-39%

1. Riska EB et al (1976).Prevention of fat embolism by early internal fixation of fractures in patients with multiple injuries.Injury 8(2);110-116
2. Küntscher G.Die Marknagelung von Knochenbrüchen Arch F Kin 1940;200:443
3. Peltier et al. (1957) An appraisal of the problem of fat embolism. Surg Gynecol Obst;104(4):313-324

CLAVOS INTRAMEDULARES
¿FRESAR O NO FRESAR?

AUTOR	N	LPA	FRESAR †	NO FRESAR †	AÑO
Pape	766 (H)	SI	21%	4%	1993
Pape	31 (H)	NO	⬆		1994
Wolinsky	ovejas	SI	=	=	1996
Willis	ratas	NO	=	=	1999
Elmaraghay	perros	SI	⬆		1999
Schemitsch	conejos	NO	=	=	1997
Bosse	453 (H)	NO	=	=	1997
Buckley	153 (H)	NO	=	=	1998
Buttaro	cerdos	NO	=	=	2002
COTS	(H)		=	=	2008

DAMAGE CONTROL

- Descrita por primera vez por ROTONDO M en fracturas pélvicas con sangrado persistente del plexo venoso presacro[1].

- Laparotomia urgente e inserción de packings para controlar la hemorragia y posteriormente realizar un second look con control posterior del sangrado.

1.Rotondo M et al. J Trauma 1993;35:375-382

Damage Control Orthopaedics

Evolving Concepts in the Treatment of Patients Who Have Sustained Orthopaedic Trauma

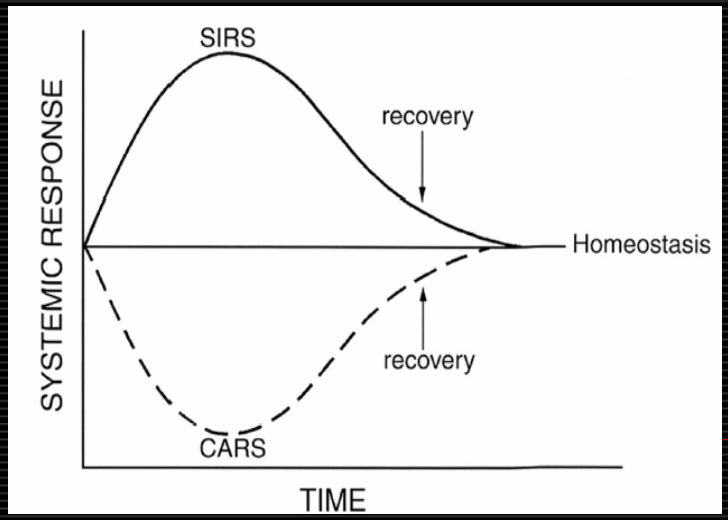
By Craig S. Roberts, MD, Hans-Christoph Pape, MD, Alan L. Jones, MD, Arthur L. Malkani, MD, Jorge L. Rodriguez, MD, and Peter V. Giannoudis, MD

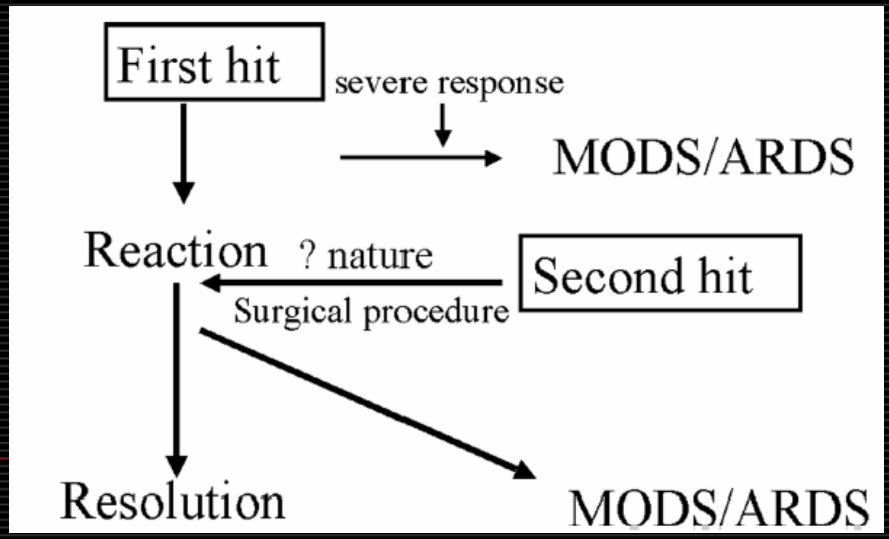
An Instructional Course Lecture, American Academy of Orthopaedic Surgeons

The Journal of Bone & Joint Surgery · jbjs.org
Volume 87-A · Number 2 · February 2005

TABLE I Cytokines That Are Important Inflammatory Mediators

Group	Examples
Interleukins (IL)	IL-1, IL-2, IL-3, IL-4, IL-5, IL-6, IL-7, IL-8, IL-10, IL-11, IL-12, IL-13, IL-18
Tumor necrosis factors (TNF)	TNF, lymphotoxin (LT)
Interferons (IFN)	IFN-alpha, IFN-beta, IFN-gamma
Colony stimulating factors (CSF)	G-CSF, M-CSF, GM-CSF





Excerpta Medica

The American Journal of Surgery

The American Journal of Surgery 183 (2002) 622–629

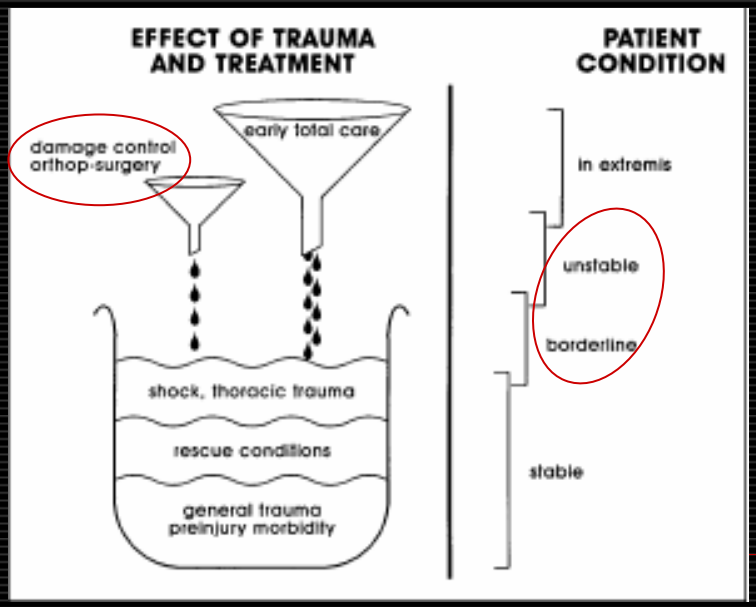
Review

The timing of fracture treatment in polytrauma patients: relevance of damage control orthopedic surgery*

Hans-Christoph Pape, M.D.[a,†], Peter Giannoudis, M.D.[b], Christian Krettek, M.D.[a]

[a]*Department of Trauma Surgery, Hannover Medical School, Carl-Neubergstr. 1, 30625 Hannover, Germany*
[b]*Department of Trauma and Orthopaedics, St James's University Hospital, Leeds, United Kingdom*

Manuscript received July 16, 2001; revised manuscript December 21, 2001



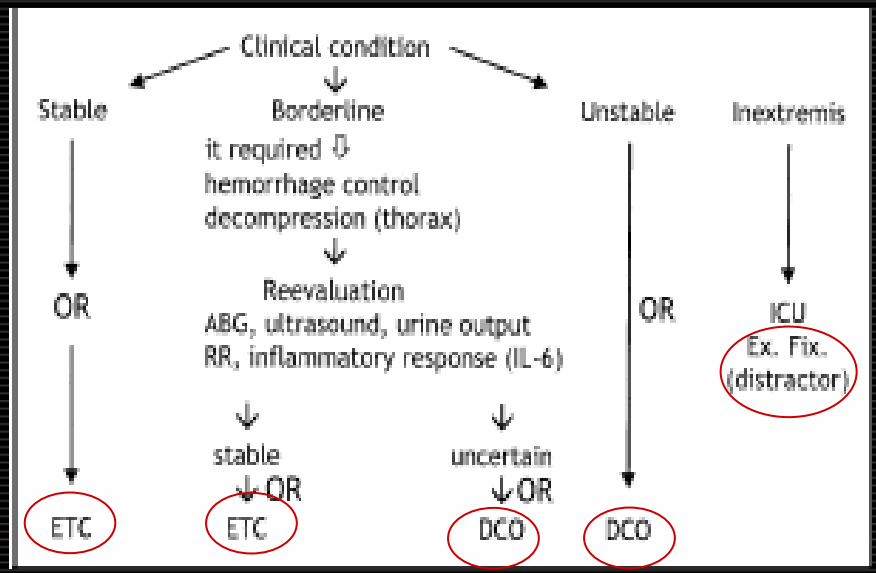


TABLE II Clinical Parameters Used in Hannover, Germany, to Define the "Borderline" Patient for Whom Damage Control Orthopaedics Is Often Preferred

Polytrauma + injury severity score of >20 points and additional thoracic trauma (abbreviated injury score >2 points)
Polytrauma with abdominal/pelvic trauma (Moore score[75] >3 points) and hemorrhagic shock (initial blood pressure <90 mm Hg)
Injury severity score of ≥40 points in the absence of additional thoracic injury
Radiographic findings of bilateral lung contusion
Initial mean pulmonary arterial pressure of >24 mm Hg
Increase of >6 mm Hg in pulmonary arterial pressure during intramedullary nailing

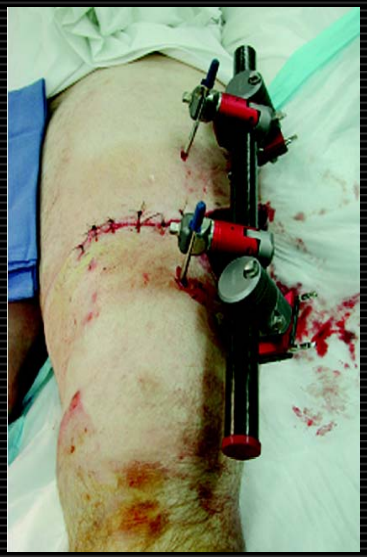
TABLE III Clinical Parameters Associated with Adverse Outcomes in Multiply Injured Patients as Reported in Hannover, Germany

Unstable condition or resuscitation difficult (borderline patient)
Coagulopathy (platelet count <90,000)
Hypothermia (<32°C)
Shock and >25 units of blood needed
Bilateral lung contusion on first plain radiograph
Multiple long-bone injuries and truncal injury; abbreviated injury score of ≥2 points
Presumed operation time >6 hr
Arterial injury and hemodynamic instability (blood pressure <90 mm Hg)
Exaggerated inflammatory response (e.g., IL-6 >800 pg/mL)

Damage Control Orthopedics in Patients With Multiple Injuries Is Effective, Time Saving, and Safe

Georg Taeger, MD, Steffen Ruchholtz, MD, Christian Waydhas, MD, Ulrike Lewan, MD, Boris Schmidt, and Dieter Nast-Kolb, MD

J Trauma. 2005;59:408–415.



External Fracture Fixation Significantly Reduces Primary Operation Time

External Fracture Fixation Does Not Increase Complications

Quality of Definite Osteosynthesis Is Not Impaired by Previous External Stabilization

FIJACION EXTERNA en primeras 8-24 h y fijación definitiva en los siguientes 4 dias es la mejor opción para pacientes de alto riesgo.

Scalea et al. Journal of Trauma Injury April 2000 Vol 48, n 4

¿La fijación medular está contraindicada en un paciente con TCE o trauma torácico?

1362 pacientes

FIJACIONES MEDULARES DE FEMUR ENTRE 2-5 DIAS ↑ ESTANCIA HOSPITALARIA

FIJACIÓN FRESADA EN LAS PRIMERAS 24 H MEJOR OUTCOME INDEPENDIENTEMENTE DEL TIPO DE LESIÓN

Sin diferencias en la mortalidad

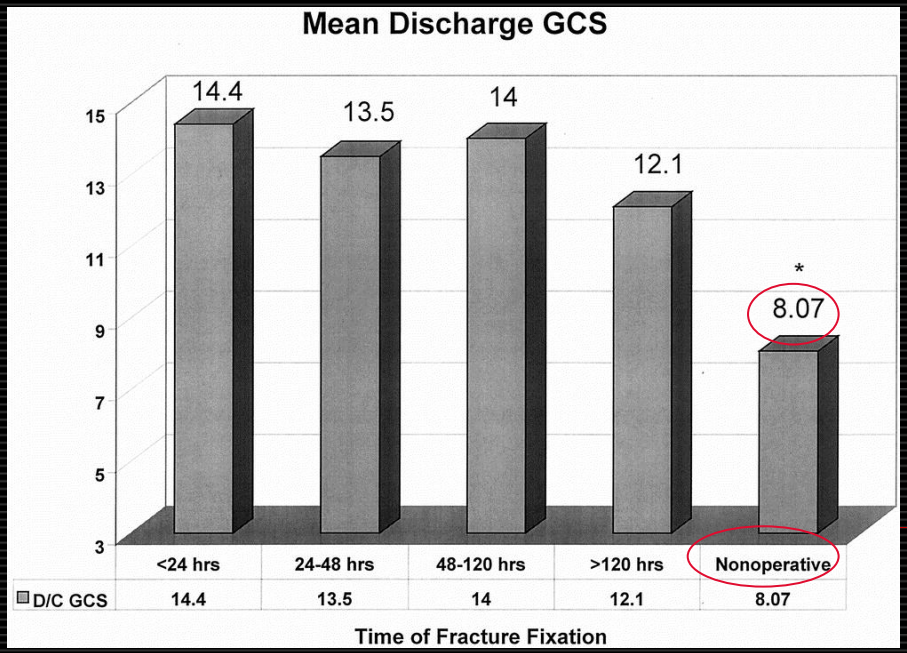
Brundage et al.Timing of femur fracture fixation:Effect on outcome in patients with thoracic and head injuries. Journal of Trauma Vol 52(2), February 2002

Table 4 Head Injury Patients: Head AIS ≥ 2 (n = 512)

	Group				
	1	2	3	4	5
Fixation time	<24 h	24–48 h	48–120 h	>120 h	Nonoperative
no.	283	65	17	13	133
Age (mean)	27.01	27.77	24.94	28.46	22.52
% male	67	68	59	54	76
Head AIS (mean)	3.80	3.05	3.11	2.94	3.46
Chest AIS (mean)	1.12	1.48	1.53	1.54	1.15
Mortality, % (n)	3.9 (11)	4.6 (3)	0.0 (0)	7.7 (1)	50 (67)
ARDS, % (n)	7.4 (21)	12 (8)	29 (5)	7.7 (1)	8.2 (11)
Pneumonia, % (n)	14 (39)	25 (16)	24 (4)	7.7 (1)	6.0 (8)
FES, % (n)	0.4 (1)	1.5 (1)	0.0 (0)	0.0 (0)	0.8 (1)
PE, % (n)	0.0 (0)	1.5 (1)	0.0 (0)	0.0 (0)	0.0 (0)
Discharge GCS score	14.4	13.5	14.0	12.1	8.07
Hospital stay*	15.5	18.8	24.3	23.8	6.07
ICU stay*	4.01	6.47	12.9	7.89	2.04

ISS, Injury Severity Score; AIS, Abbreviated Injury Scale; ARDS, acute respiratory distress syndrome; FES, fat embolism syndrome; PE, pulmonary embolism; ICU, intensive care unit.

* Mean in days.



Brundage et al.
Timing of femur fracture fixation:Effect on outcome in patients with thoracic and head injuries.
Journal of Trauma Vol 52(2), February 2002

¿Fijación precoz DEFINITIVA en paciente politraumático con TCE?

- TCE 17% de politraumáticos

CONTRAINDICACION ABSOLUTA: SWELLING CEREBRAL

OPTIMIZACION DEL MANEJO EN LOS PRIMEROS DIAS ES ESENCIAL

Lehman U et al. Unfallchirurg 2001 Mar;104(3):196-209

Brundage et al. Timing of femur fracture fixation:Effect on outcome in patients with thoracic and head injuries.
Journal of Trauma Vol 52(2), February 2002

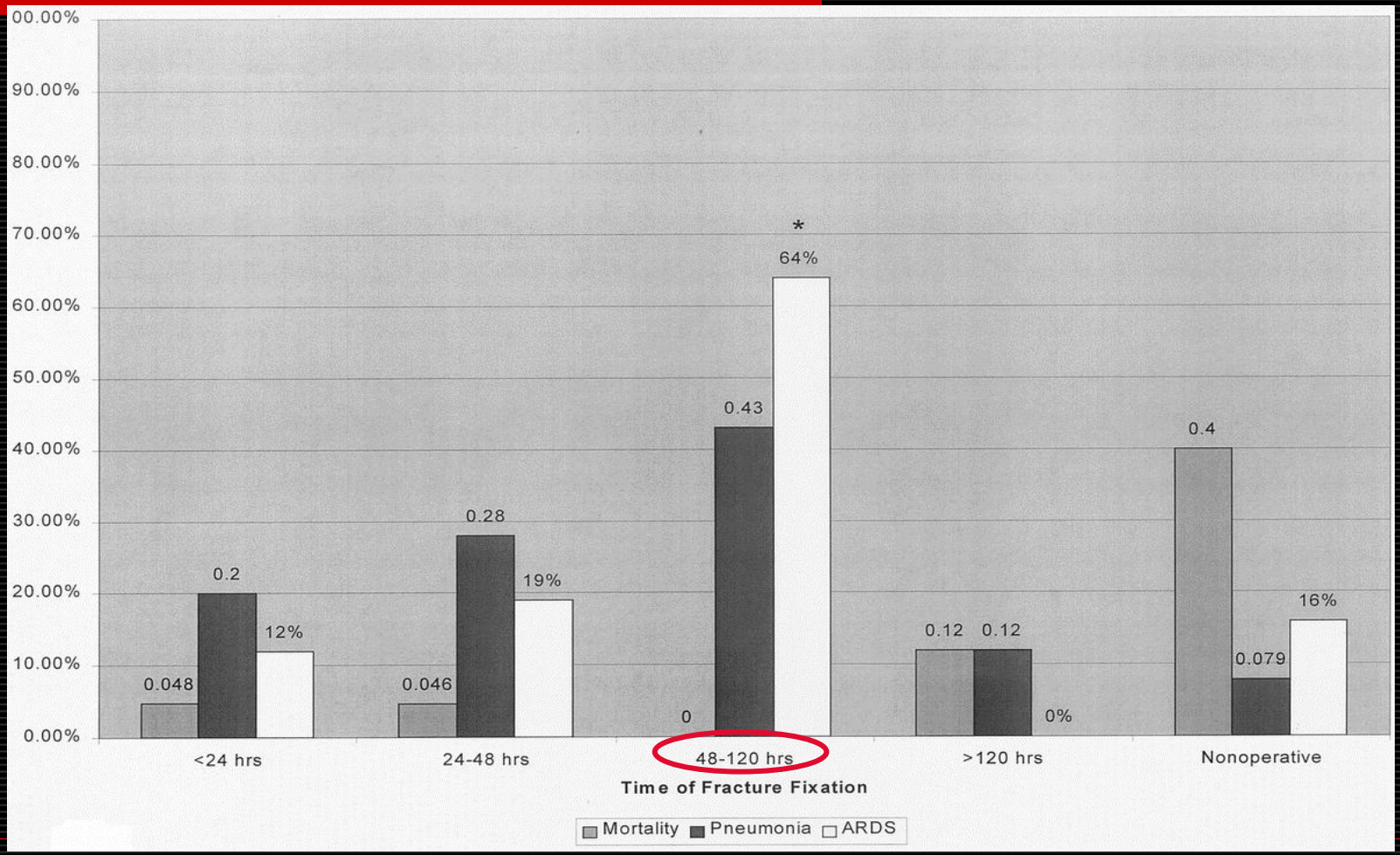
Table 3 Chest Trauma Patients: Chest AIS ≥ 2 (n = 323)

	Group				
	1	2	3	4	5
Fixation time	<24 h	24–48 h	48–120 h	>120 h	Nonoperative
no.	186	43	14	8	77
Age (mean)	31.22	30.98	34.07	27.50	30.95
% male	68	63	64	75	70
ISS (mean)	27.11	28.95	29.71	30.63	36.99
Head AIS (mean)	1.70	2.21	1.64	2.63	2.58
Chest AIS (mean)	3.02	3.14	3.21	3.13	3.25
Mortality, % (n)	4.8 (9)	4.6 (2)	0.0 (0)	12 (1)	40 (31)
ARDS, % (n)	12 (22)	19 (8)	64 (9)	0.0 (0)	16 (12)
Pneumonia, % (n)	20 (37)	28 (12)	43 (6)	12 (1)	7.8 (6)
FES, % (n)	1.1 (2)	0.0 (0)	0.0 (0)	0.0 (0)	2.6 (2)
PE, % (n)	1.6 (3)	0.0 (0)	0.0 (0)	0.0 (0)	0.0 (0)
Hospital stay*	17.7	21.4	42.0	23.1	12.1
ICU stay*	4.51	9.59	27.4	8.17	7.22

ISS, Injury Severity Score; AIS, Abbreviated Injury Scale; ARDS, acute respiratory distress syndrome; FES, fat embolism syndrome; PE, pulmonary embolism; ICU, intensive care unit.

* Mean in days.

Brundage et al.
Timing of femur fracture fixation:Effect on outcome in patients with thoracic and head injuries.
Journal of Trauma Vol 52(2), February 2002



Early Unreamed Intramedullary Nailing of Femoral Fractures is Safe in Patients With Severe Thoracic Trauma

Patrick Weninger, MD, Markus Figl, MD, Ralf Spitaler, MD, Walter Mauritz, MD, PhD, and Harald Hertz, MD, PhD

J Trauma. 2007;62:692–696.

	Study Cohort	Control Cohort	*p* Value
Number of patients	45	107	
Age (mean)	33.4	32.2	NS
Female (n)	14	35	NS
Male (n)	31	72	NS
ISS (range)	39.5 (17–75)	38.3 (17–75)	NS
AIS score, median (range)			
Head/neck	2.2 (0–5)	2.1 (0–5)	NS
Thorax	4.2 (3–5)	4.1 (3–5)	NS
Abdomen	2.1 (0–5)	2.8 (0–5)	NS
Extremity	3.1 (3–5)	1.0 (0–4)	0.002
External	1.2 (0–3)	0.4 (0–3)	0.031

NS, not significant.

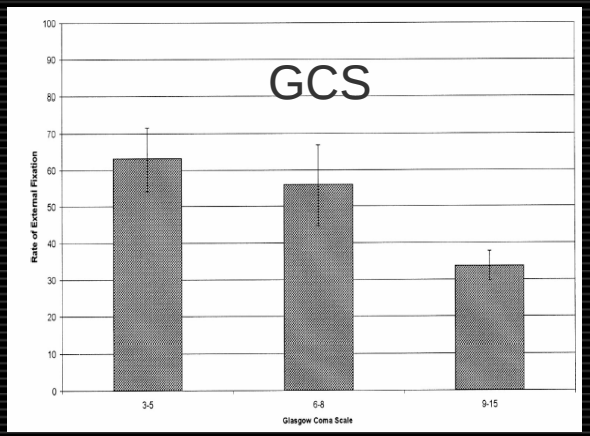
	Study Cohort	Control Cohort
ICU days	11.6 ± 9.2	13.3 ± 10.1
Ventilation days	8.9 ± 7.1	11.0 ± 8.7
Pneumonia	13 (28.9)	32 (29.9)
ARDS	9 (20.0)	26 (24.3)
MOFS	8 (17.7)	19 (17.8)
Early mortality (<24 h after admission)	5 (11.1)	8 (7.5)
TRISS survival	0.81 ± 0.18	0.83 ± 0.20
SAPS II mortality	18.5 ± 14.2	18.1 ± 13.9
ICU mortality	9 (20.0)	22 (20.6)
Observed vs. expected mortality ratio	1.05	1.21

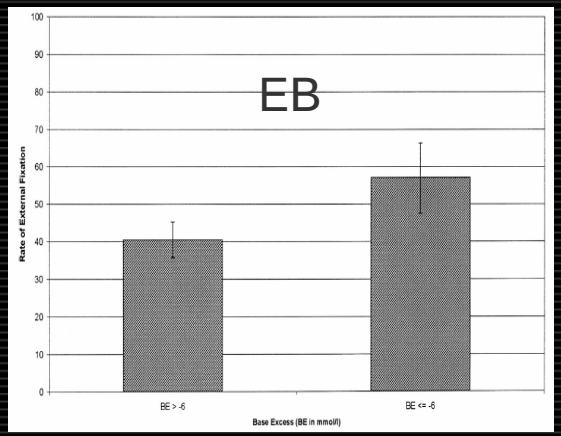
FIJACION QUIRÚRGICA PRECOZ DEPENDIENDO DEL ESTADO PREVIO DEL PACIENTE EN PRIMERAS 24 H.

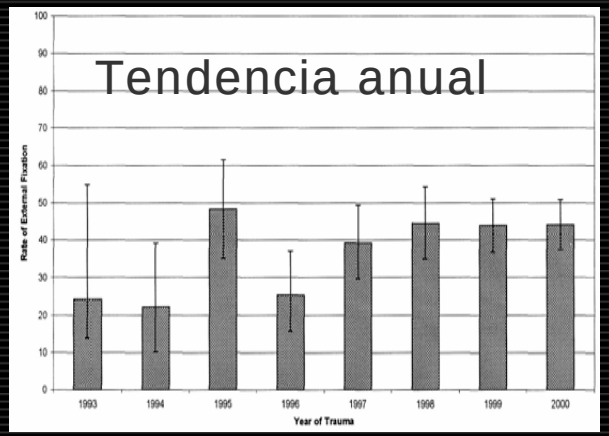
Evaluation of Criteria for Temporary External Fixation in Risk-Adapted Damage Control Orthopedic Surgery of Femur Shaft Fractures in Multiple Trauma Patients: "Evidence-Based Medicine" versus "Reality" in the Trauma Registry of the German Trauma Society

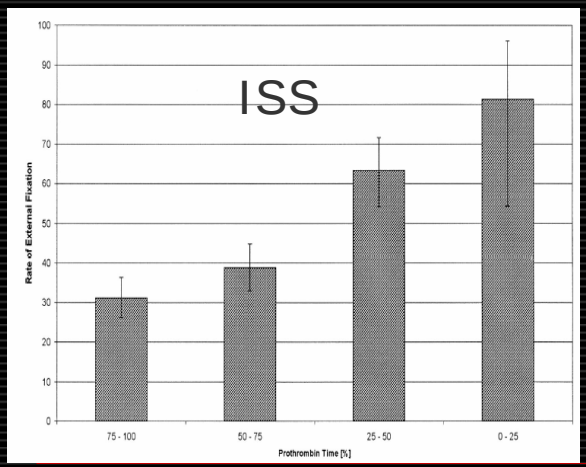
Dieter Rixen, MD, Guido Grass, MD, Stefan Sauerland, MD, MPH, Rolf Lefering, PhD, Marcus R. Raum, MD, Nedim Yücel, MD, Bertil Bouillon, MD, Edmund A. M. Neugebauer, PhD, and the Polytrauma Study Group of the German Trauma Society

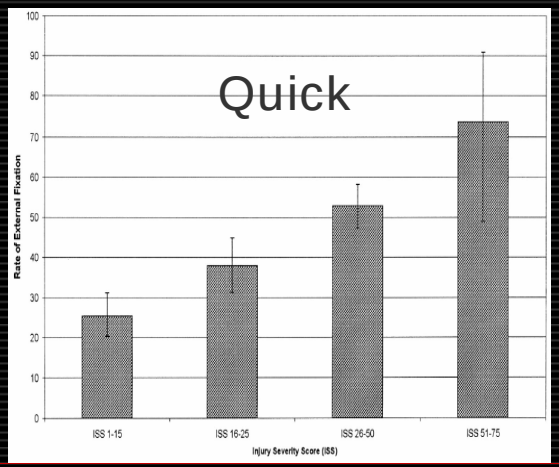
J Trauma. 2005;59:1375–1395.

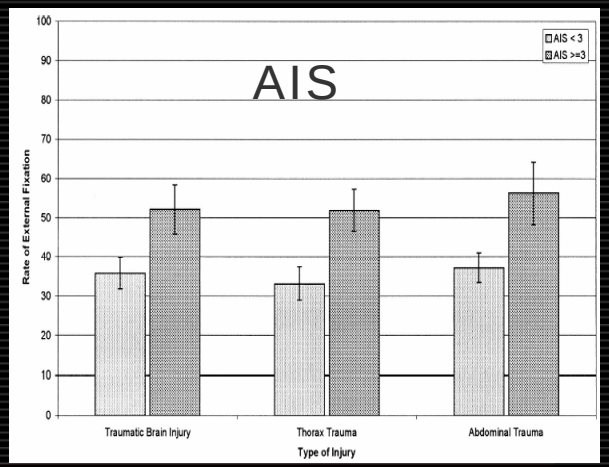








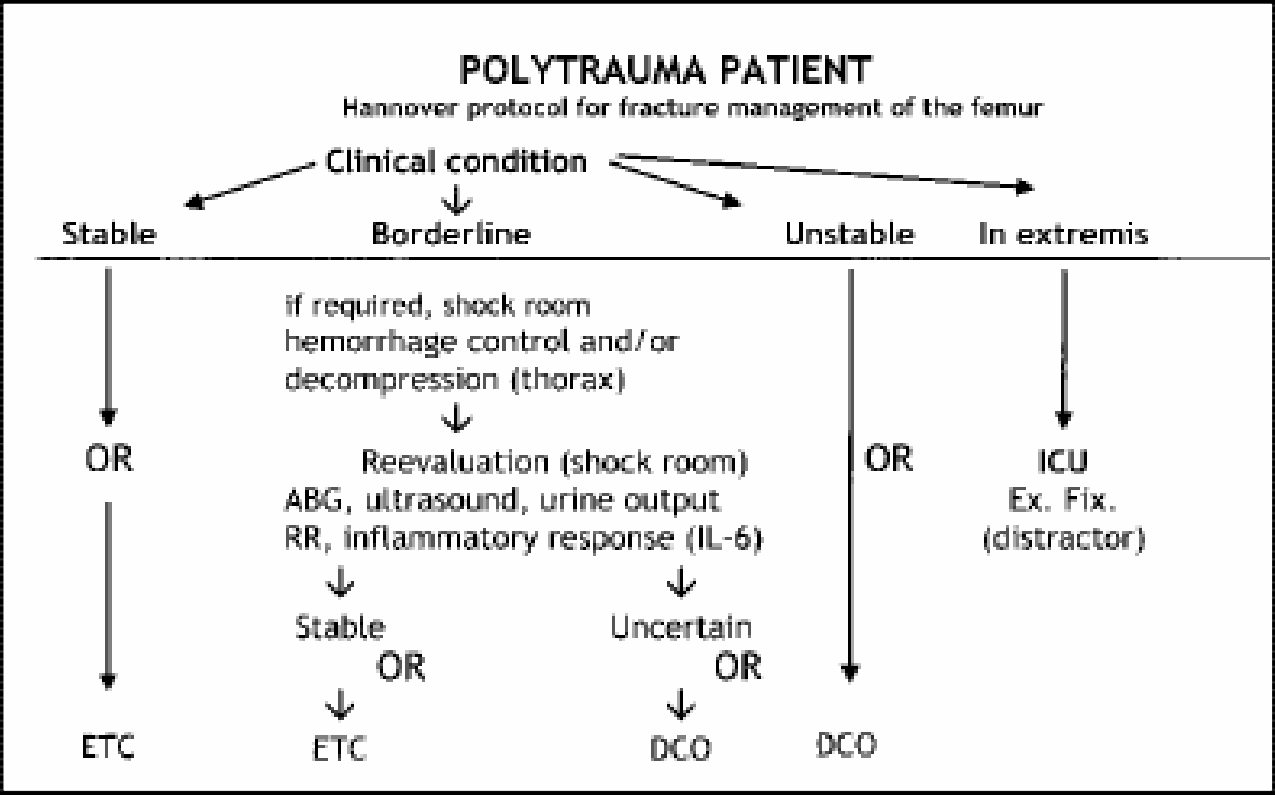




Changes in the Management of Femoral Shaft Fractures in Polytrauma Patients: From Early Total Care to Damage Control Orthopedic Surgery

Hans-Christoph Pape, MD, Frank Hildebrand, MD, Stephanie Pertschy, MD, Boris Zelle, MD, Rayeed Garapati, MD, Kai Grimme, MD, and Christian Krettek, MD

J Trauma. 2002;53:452–462.



CRÍTICAS A LA LITERATURA

- CONCEPTO DE FIJACIÓN PRECOZ
- TIPO DE FIJACIÓN
- LESIÓN PARTES BLANDAS[1]

1.Mudd Kl et al.Analysis of pulmonary fat embolism in blunt force fatalities J Trauma 48 (4):711-715

¿SE PUEDE OBJETIVAR EN EL LABORATORIO EL BENEFICIO DEL TRATAMIENTO QUIRÚRGICO EN FRACTURAS DE FÉMUR?

Impact of Intramedullary Instrumentation versus Damage Control for Femoral Fractures on Immunoinflammatory Parameters: Prospective Randomized Analysis by the EPOFF Study Group

Hans-Cristoph Pape, MD, K. Grimme, Martin van Griensven, PhD, A. H. Sott, MD, P. Giannoudis, MD, J. Morley, MD, Olav Roise, MD, Elisabeth Ellingsen, MD, Frank Hildebrand, MD, B. Wiese, MD, and Christian Krettek, MD

J Trauma. 2003;55:7–13.

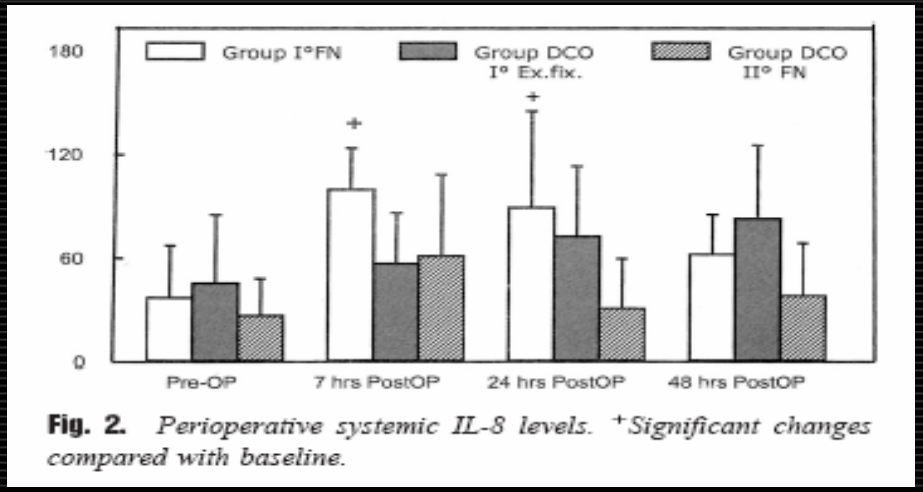


Fig. 2. *Perioperative systemic IL-8 levels. ⁺Significant changes compared with baseline.*

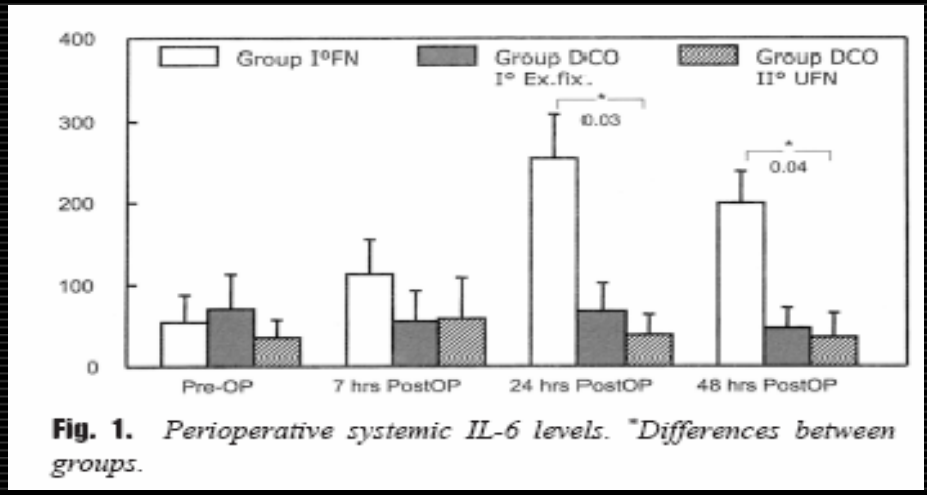


Fig. 1. *Perioperative systemic IL-6 levels. *Differences between groups.*

IL-6 > 800 pg/ml PREDICTOR DE FMO

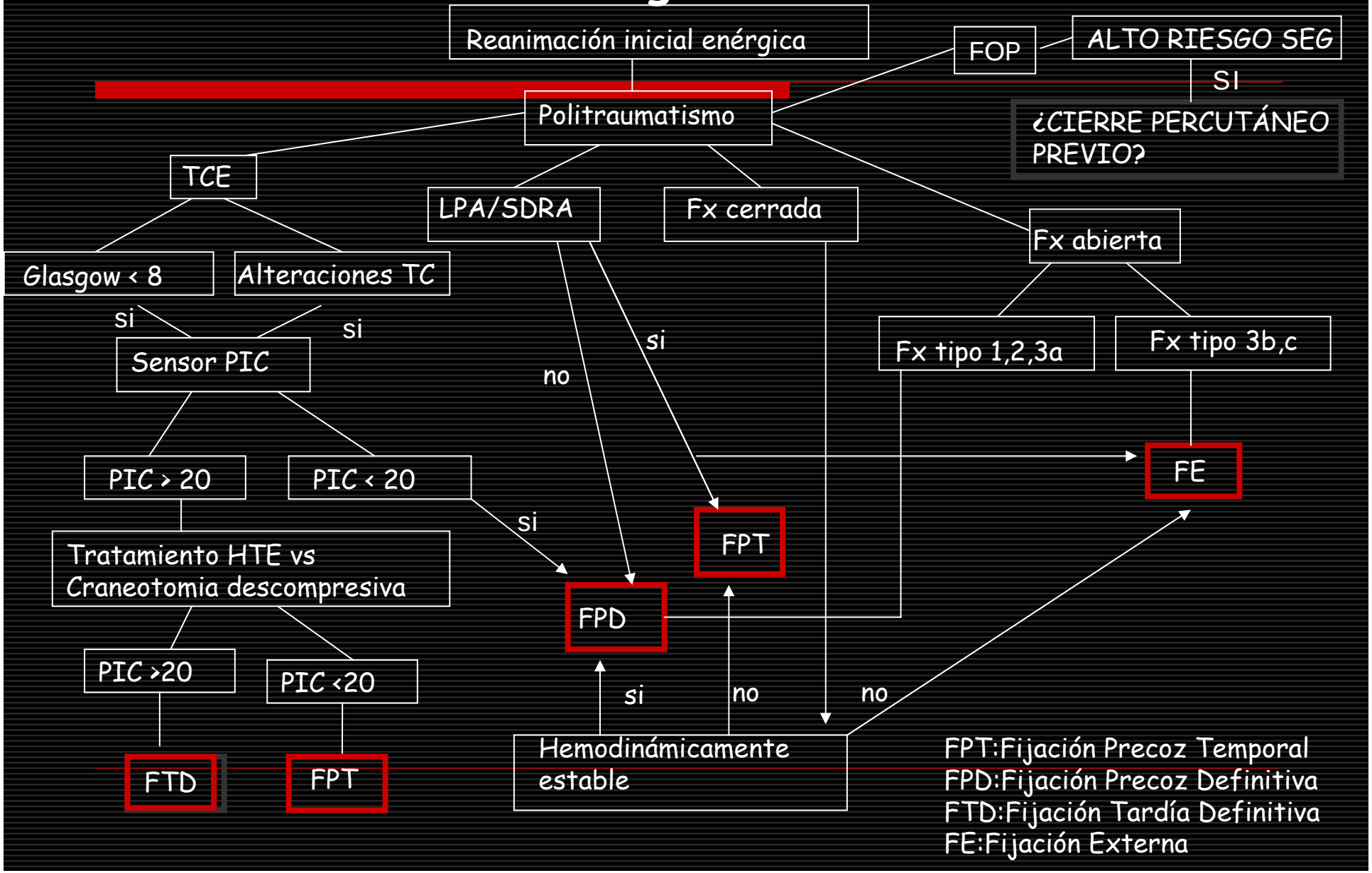
Propuesta de protocolo de tratamiento de fracturas de huesos largos en PPT

Fracturas abiertas según Gustilo

- Tipo I: Tejido cutáneo limpio < 1 cm longitud
- Tipo II: Laceración > 1 cm en ausencia de lesión severa de partes blandas
- Tipo III: Partes blandas extensa
  - A)Adecuado cubrimiento partes blandas
  - B)No cobertura de partes blandas
  - C)Lesión arterial

J Trauma 1984;24:742-746

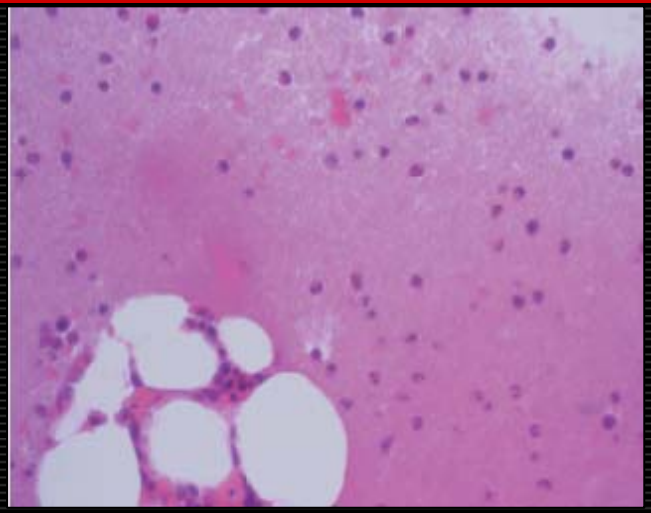
Propuesta de protocolo de tratamiento de fracturas de huesos largos en PPT

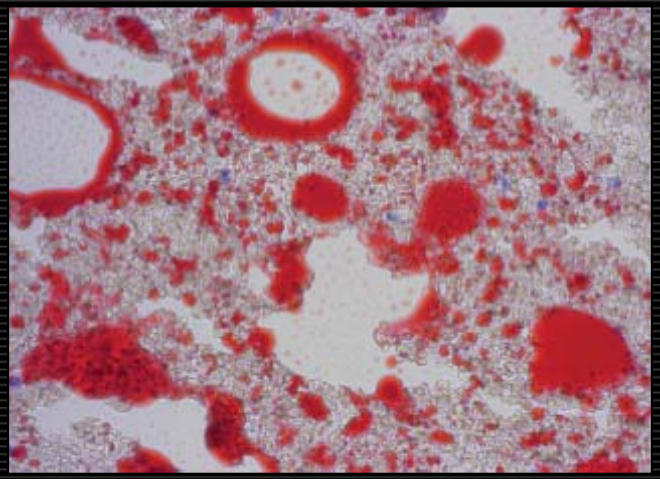


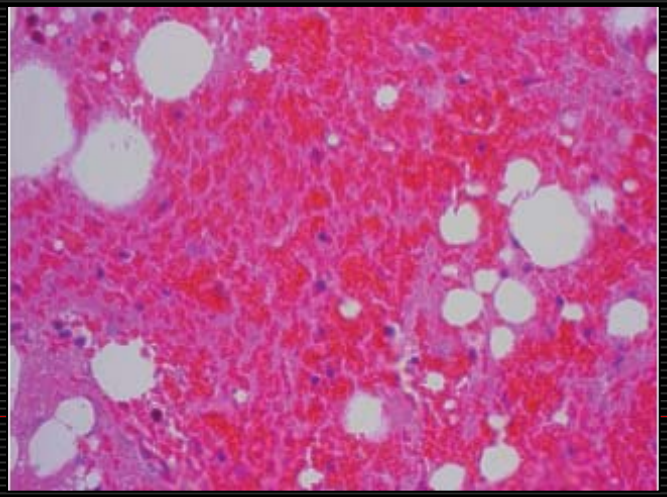
Histopathologic Features of Fat Embolism in Fulminant Fat Embolism Syndrome

Fumio Arai, M.D., Takashi Kita, M.D., Ph.D.,* Takeshi Nakai, M.D., Ph.D.,† Tatsuyuki Hori,‡ Naritoshi Maki, M.D.,* Masaaki Kakiuchi, M.D., Ph.D.,† Shigeta Sasaki, M.D.**

Anesthesiology 2007; 107:509–11







CASO CLINICO

- EDAD:27 A
- SEXO: VARÓN
- AP: ENOLISMO MODERADO.
- CAUSA: ACCIDENTE MOTO SIN CASCO
- LESIONES PRIMARIAS: ESCORIACIONES TORAX Y ESPALDA, FX TERCIO MEDIO FÉMUR DERECHO, FX ESCAFOIDES DERECHO,NO PÉRDIDA DE CONCIENCIA.
- INTERVALO CLÍNICA:48 H
- LESIONES SECUNDARIAS: HIPOXEMIA, PETEQUIAS TRONCO Y DESATURACIÓN BRUSCA COINCIDIENDO CON TRASFUSIÓN DE UN CONCENTRADO DE HEMATIES.
- GSA: HIPOXEMIA, HIPOCAPNIA, ANEMIA, HIPOCALCEMIA
- CLÍNICA POSTERIOR: SDR. CONFUSIONAL Y AGITACIÓN PSICOMOTRIZ

COMPLICACIONES TRANSFUSIONALES

REACCIONES INMUNOLOGICAS

- Inmunomodulacion

POR HEMATIES:

- Reacción hemolítica aguda
- Reacción hemolítica retardada
- Aloanticuerpos eritrocitarios
- Aloinmunización

POR LEUCOCITOS

- Reacción febril
- Edema agudo de pulmón
- Aloinmunización
- Enfermedad injerto contra huésped

POR PLAQUETAS

- Aloinmunizaciones
- Púrpura transfusional
- Leucopenia

POR PROTEINAS PLASMÁTICAS

- Urticaria
- Anafilaxia

REACCIONES NO INMUNOLÓGICAS

- Infecciosas
- Sobrecarga de Fe
- Trombocitopenia por hemodilución
- Déficit de factores por hemodilución
- Reacción embólica (gaseosa o microagregados)

Púrpura postransfusional

- Antecedente de embarazo o transfusión previa
- Etiopatogenia: Ac anti Ag plaquetarios
- Ag + frecuente hallado: Ag PI^{A1}
- Ag PIA1 carece 2% población
- Clínica: Intensa trombocitopenia **6 - 10 dias** tras la transfusión.

Am J Hemat 1996 Jul 52(3):205-11

Exantemas asociados a la transfusión

EICH postransfusional:

- Linfocitos del donante ⇔ Ag del receptor
- Inicio clínica: 2-50 dias
- Alteraciones de la inmunidad celular
- Exantema + diarrea + pancitopenia + hepatopatia

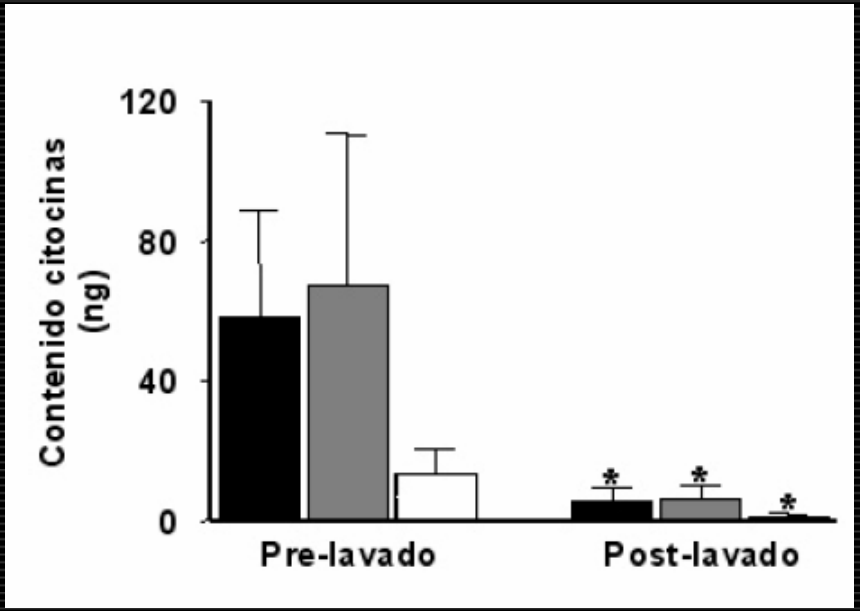
Reacción alérgica-anafilactoide:

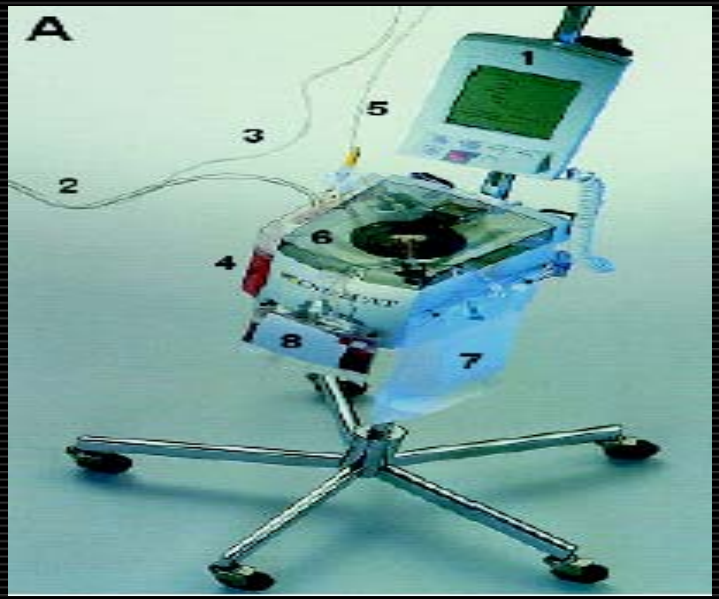
- 3% transfusiones plasmáticas
- Ac Ig E receptor ⇔ proteinas del donante.
- Profilaxis

(Rev. Esp. Anestesiol. Reanim. 2005; 52: 321-327) **ORIGINAL**

Evaluación del sistema de autotransfusión OrthoPAT®, utilizando modelos experimentales de simulación de recuperación de sangre intra y postoperatoria

M. Muñoz Gómez, D. Ariza Villanueva*, A. Romero Ruiz**, E. Muñoz Morán, I. Prat Arrojo***, A. Gómez Luque*
GIEMSA, Facultad de Medicina. Servicios de *Anestesiología y Reanimación y **Hematología, Hospital Clínico Universitario Virgen de la Victoria; y ***Centro Regional de Transfusión Sanguínea. Málaga.





TRALI

- Primer caso descrito en 1951 por Barnard.
- Descrito por Popovosky en 1983
- Incidencia real desconocida
  (0.001-0.16%)
- 2ª causa de mortalidad asociada a la transfusión
- Mortalidad 5-45%

TRALI
EPIDEMIOLOGIA

Cuadro I
Prevalencia porcentual y relativa de TRALI según el número de unidades transfundidas (90 transfusiones)[6]

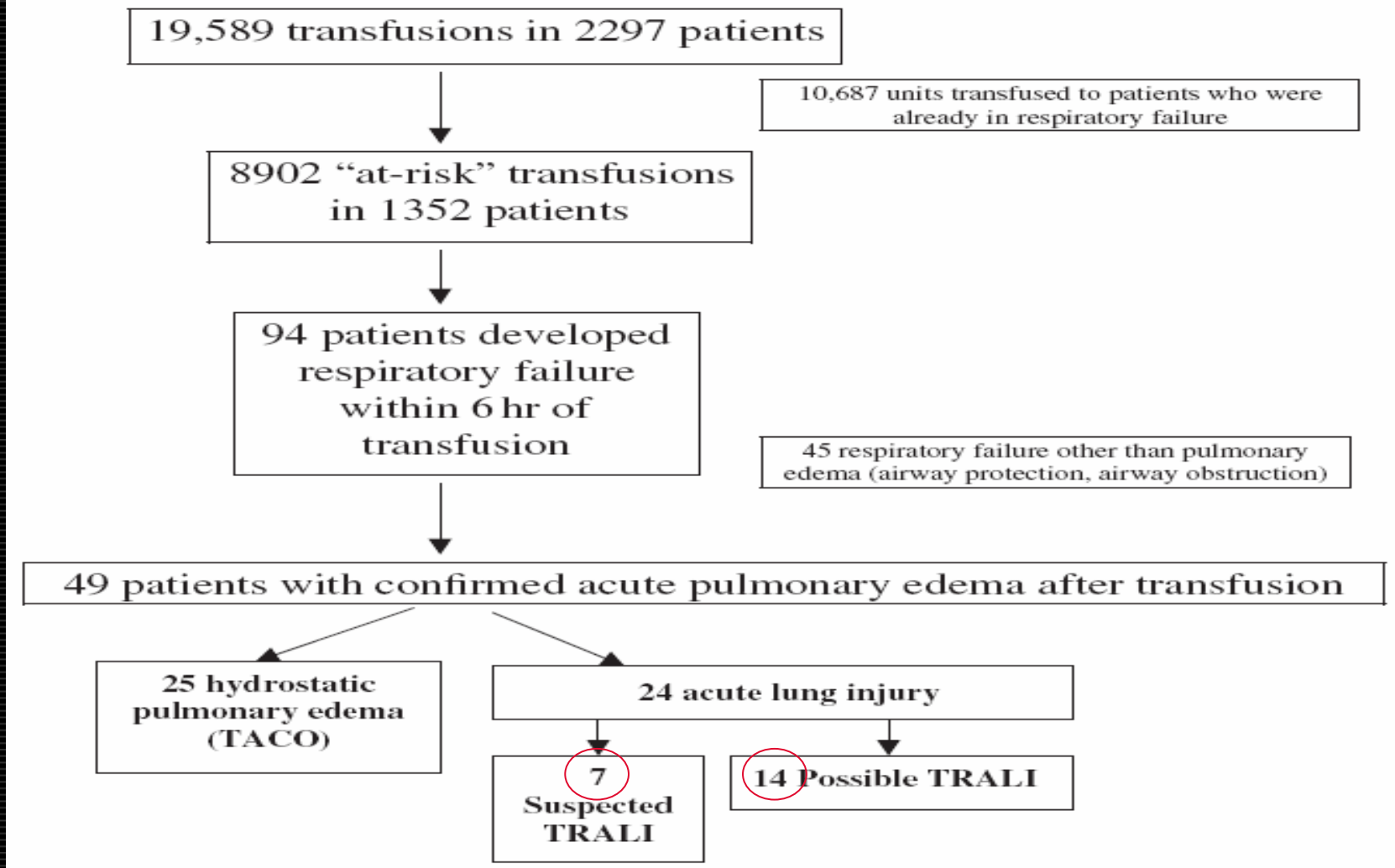
Componente	Número de casos	Total de unidades transfundidas	Prevalencia %	Prevalencia Relativa
Concentrado plaquetas (sangre)	72	31 074	0.23	1/432
Concentrado plaquetas (aféresis)	2	2447	0.082	1/1224
Concentrado de glóbulos rojos	15	66 161	0.023	1/4410
Plasma fresco congelado	1	19 411	0.0052	1/19411
Sólo componente celular	89	99 682	0.089	1/1120

Rev Med IMSS 2004; 42 (5): 501-505

Transfusion-related acute lung injury and pulmonary edema in critically ill patients: a retrospective study

Rimki Rana, Evans R. Fernández-Pérez, S. Anjum Khan, Sameer Rana, Jeffrey L. Winters, Timothy G. Lesnick, S. Breanndan Moore, and Ognjen Gajic

TRANSFUSION Volume 46, September 2006

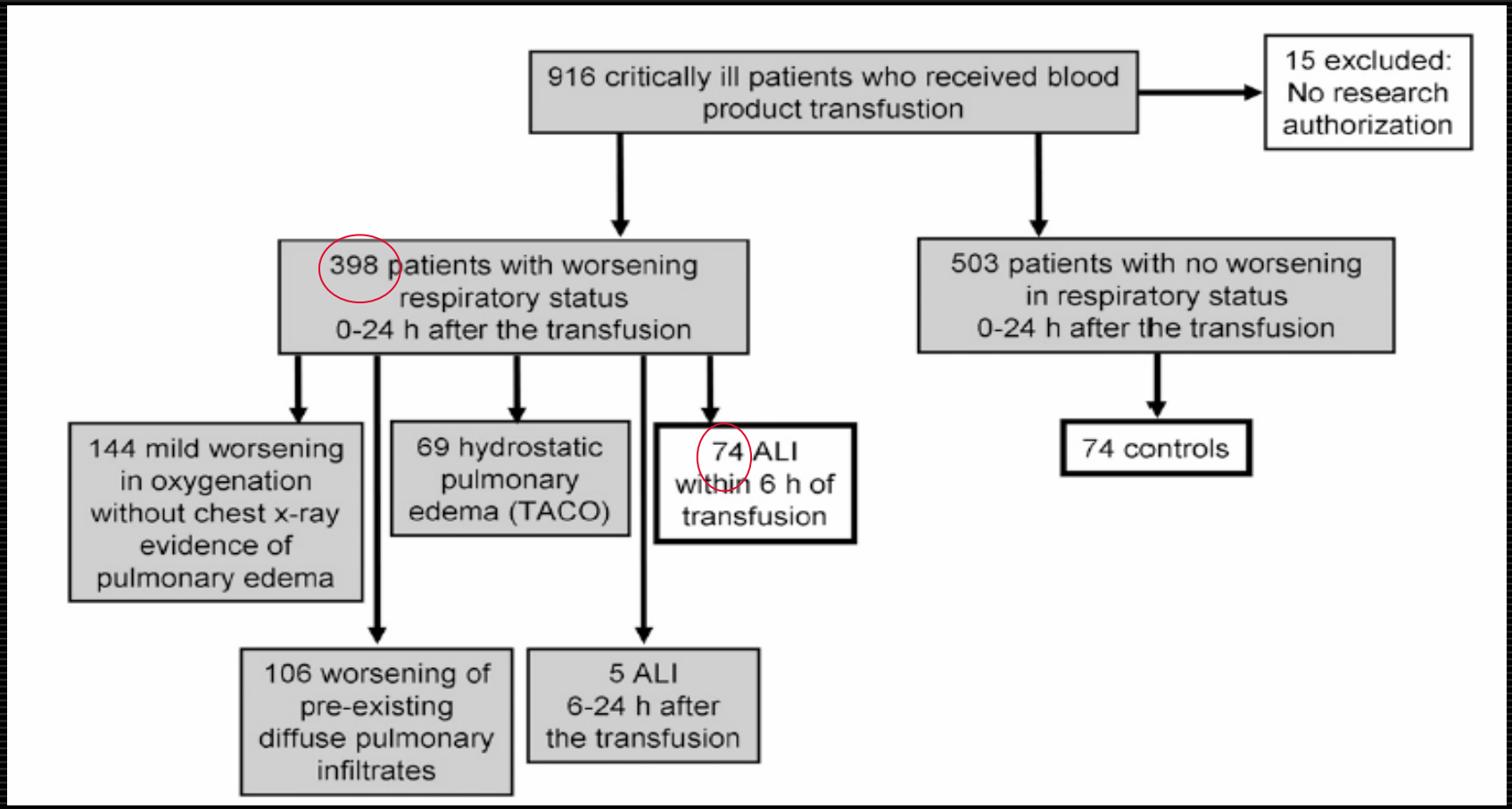


Transfusion-related Acute Lung Injury in the Critically Ill

Prospective Nested Case-Control Study

Ognjen Gajic[1,4], Rimki Rana[1], Jeffrey L. Winters[2], Murat Yilmaz[1], Jose L. Mendez[1], Otis B. Rickman[1], Megan M. O'Byrne[3,4], Laura K. Evenson[1,4,5], Michael Malinchoc[3,4], Steven R. DeGoey[2], Bekele Afessa[1,4], Rolf D. Hubmayr[1,4], and S. Breanndan Moore[2]

AMERICAN JOURNAL OF RESPIRATORY AND CRITICAL CARE MEDICINE VOL 176 2007



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[1]Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, [2]Division of Transfusion Medicine, Department of Laboratory Medicine and Pathology, and [3]Division of Biostatistics, Department of Health Sciences Research, [4]Mayo Epidemiology and Translational Research in Intensive Care (METRIC), and [5]Department of Nursing, Mayo Clinic College of Medicine, Rochester, Minnesota

Am J Respir Crit Care Med Vol 176. pp 886–891, 2007

TABLE 3. TRANSFUSION-RELATED RISK FACTORS FOR ACUTE LUNG INJURY

	Unadjusted*		Adjusted†	
Variable	OR (*95% CI*)	*P* Value	OR (*95% CI*)	*P* Value
Any high plasma volume components (FFP or platelets)	2.55 (1.27–5.11)	0.009	2.78 (1.21–6.38)	0.016
Number of units	1.09 (0.99–1.20)	0.081	1.11 (0.99–1.25)	0.086
Number of units from female donors	1.30 (1.03–1.66)	0.029	1.51 (1.08–2.12)	0.016
Amount of plasma from male donors, L	1.55 (0.79–3.06)	0.202	1.60 (0.76–3.37)	0.215
Amount of plasma from female donors, L	3.23 (1.17–8.91)	0.024	5.09 (1.37–18.85)	0.015
Amount of plasma from female donors with at least one pregnancy, L	4.41 (1.00–19.55)	0.050	9.48 (1.38–65.35)	0.022
Number of pregnancies among donors	1.11 (1.00–1.22)	0.047	1.19 (1.05–1.34)	0.007
Number of HLA class I^{+} units	1.81 (0.97–3.38)	0.061	1.70 (0.94–3.09)	0.098
Number of HLA class II^{+} units	1.93 (0.88–4.28)	0.103	3.08 (1.15–8.25)	0.025
Number of GIF^{+} units	4.19 (1.22–14.32)	0.023	4.85 (1.32–17.86)	0.018
Mean LysoPC 16:0** (per 10-mol/L increase)	1.16 (1.04–1.30)	0.011	1.16 (1.02–1.32)	0.022
Mean LysoPC 18:0** (per 10-mol/L increase)	1.58 (1.10–2.26)	0.013	1.61 (1.08–2.38)	0.018

Understanding the Consequences of Transfusion-Related Acute Lung Injury*

Aryeh Shander, MD, FCCP; and Mark A. Popovsky, MD

CHEST / 128 / 5 / NOVEMBER, 2005 SUPPLEMENT

Table 1—*Incidence/Prevalence of TRALI With Various Blood Transfusion Products*

Product Type	Reported Occurrence	Source
All components	1 in 1,323 U	Silliman et al,[10] 2003
	1 in 5,408 U	Popovsky and Moore,[2] 1985
All cellular components	1 in 1,120 U	Silliman et al,[10] 2003
	1 in 2,000 U	Silliman et al,[18] 1997
Packed RBCs	1 in 4,410 U	Silliman et al,[10] 2003
Plasma (FFP)	1 in 19,411 U	Silliman et al,[10] 2003
	1 in 7,900 U	Wallis et al,[12] 2003
From multiparous donors	1 in 200 U	Palfi et al,[17] 2001
From a single donor implicated in a TRALI fatality	1 in 3 patients exposed	Kopko et al,[4] 2002
Platelets (random donor)	1 in 317 U	Clarke et al,[21] 1994
Whole blood-derived platelets	1 in 432 U	Silliman et al,[10] 2003
Apheresis platelets	1 in 1,224 U	Silliman et al,[10] 2003
Platelet concentrates and/or RBCs		
Preuniversal leukodepletion	Approximately 1 in 4 patients with transfusion reaction	Yazer et al,[20] 2004
Intermediate leukodepletion*	Approximately 1 in 11 patients with transfusion reaction	Yazer et al,[20] 2004
After universal leukodepletion	Approximately 1 in 32 patients with transfusion reaction	Yazer et al,[20] 2004
IVIG	Single case report	Rizk et al,[15] 2001
Cryoprecipitate	Single case report	Reese et al,[16] 1975
Allogeneic bone marrow stem cells	Single case report	Urahama et al,[14] 2003
Granulocytes	2 case reports	O'Connor et al,[22] 1998; Sachs and Bux,[19] 2003

*After universal leukodepletion of platelet concentrates but before universal depletion of RBCs.

A randomized controlled trial of transfusion-related acute lung injury: is plasma from multiparous blood donors dangerous?

Miodrag Palfi, Sören Berg, Jan Ernerudh, and Gösta Berlin

Volume 41, March 2001 TRANSFUSION

TABLE 2. TNFα, IL-1ra, sE-Selectin, and C3d before and after transfusion of control plasma or plasma from multiparous women*

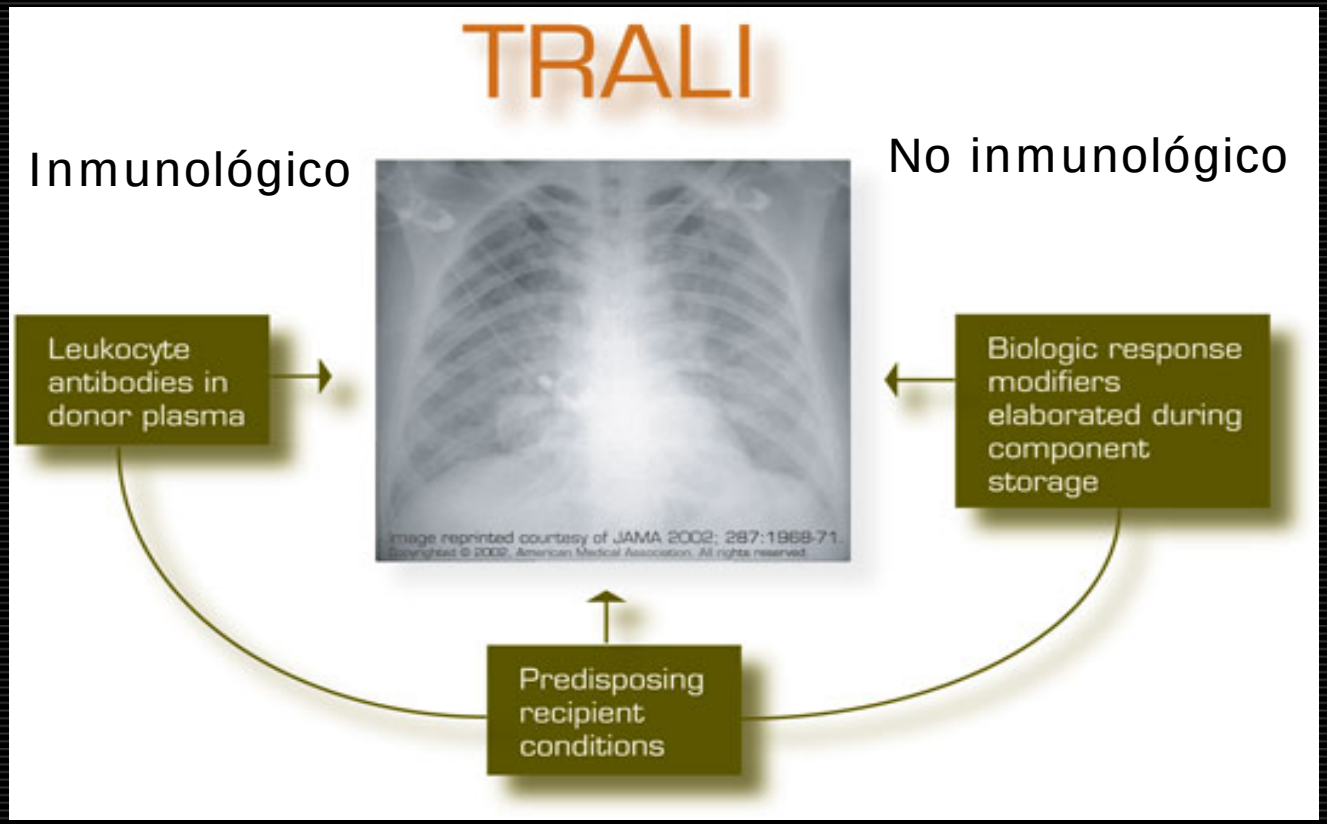
	Plasma	Before	After	Normal range according to the manufacturer
TNFα	Control	12.1 ± 15.6	15.7 ± 26.7	<15.6 pg/mL
(n† = 30)	Multiparous	10.0 ± 23.6	23.6 ± 40.3‡	
IL-1ra	Control	2714 ± 2252	2647 ± 2288	200 pg/mL
(n = 30)	Multiparous	2894 ± 2433	2884 ± 2640	
sE-Selectin	Control	100 ± 77.2	96.5 ± 67.6	46.2 ± 17.1 ng/mL
(n = 20)	Multiparous	77.8 ± 65.3	85.1 ± 59.1	
C3d	Control	9.8 ± 4.0	9.4 ± 3.3	<12 g/L
(n = 30)	Multiparous	9.0 ± 3.5	9.1 ± 2.4	

* The results are expressed as mean ± SD.

† Number of tested patient samples (paired samples, Wilcoxon's signed-rank test).

‡ $p<0.05$ difference in concentration (concentration after transfusion minus concentration before transfusion, paired samples, Wilcoxon's signed-rank test).

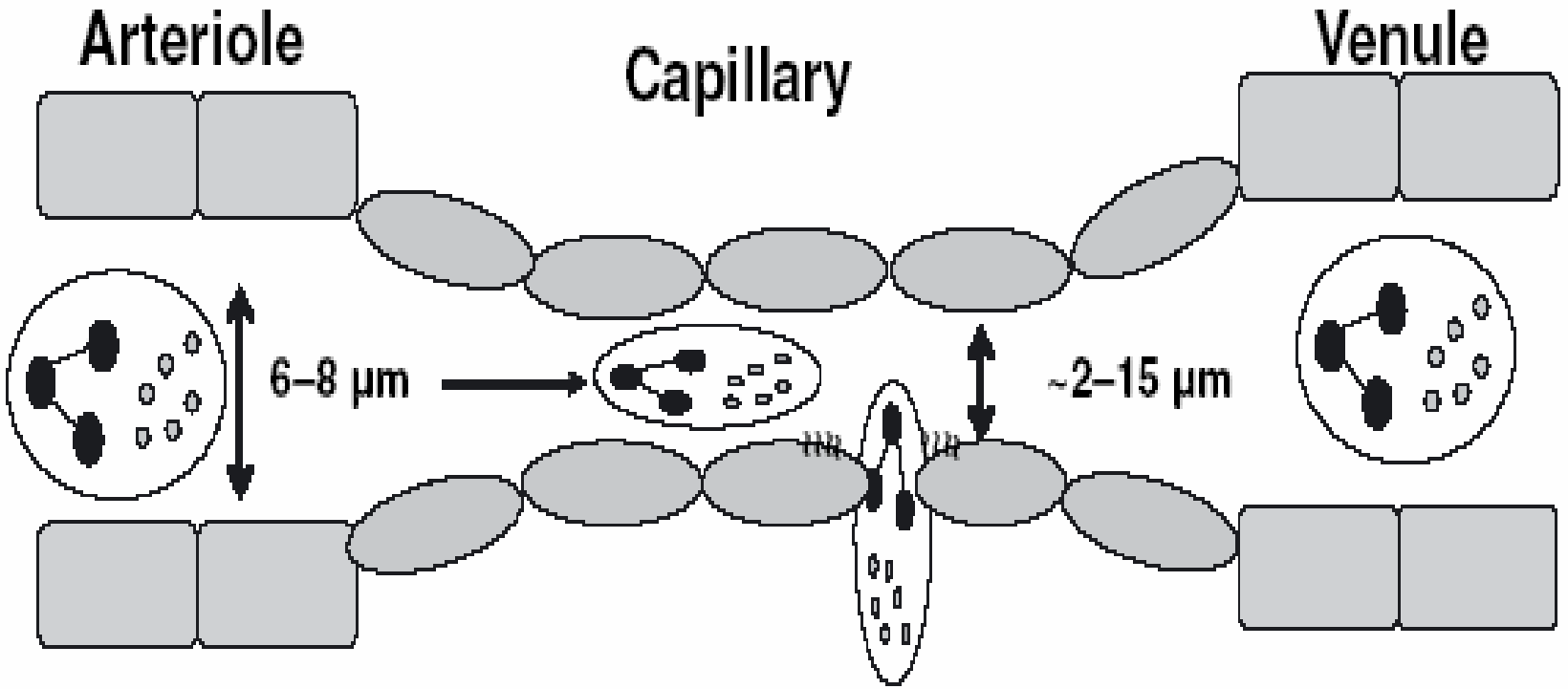
TRALI
FISIOPATOLOGIA



The pathogenesis of transfusion-related acute lung injury (TRALI)

Jürgen Bux[1] and Ulrich J. H. Sachs[2]

[1]DRK-Blood Service West of the German Red Cross, Hagen, and [2]Institute for Clinical Immunology and Transfusion Medicine, Justus Liebig-University, Giessen, Germany



British Journal of Haematology, **136**, 788–799

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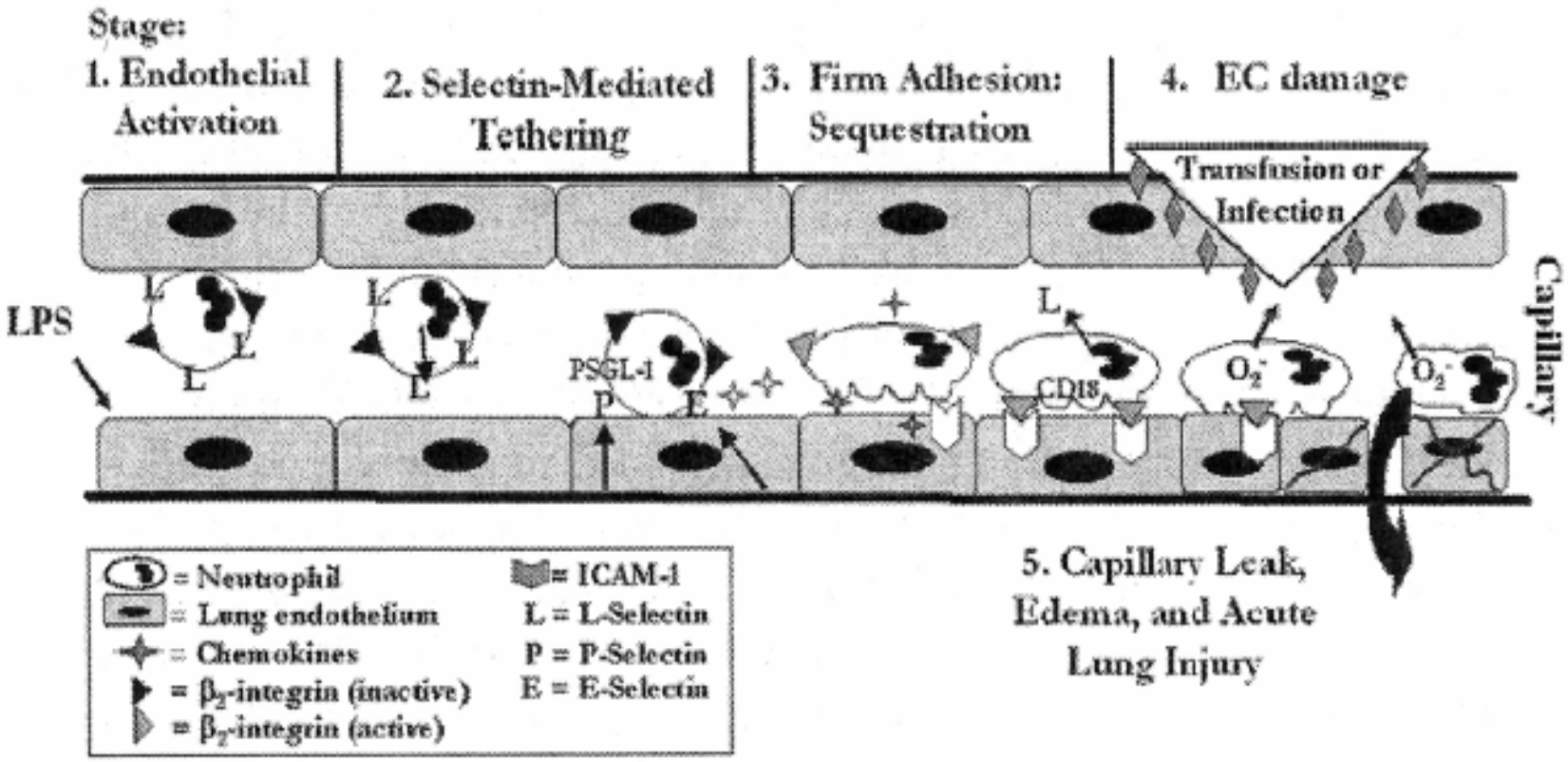


Transfusion. 2005 (suppl);45:109S-16S.

TRALI
FISIOPATOLOGIA

-Laboratory Investigation and Documentation of Suspected TRALI Episode*

CHEST / 128 / 5 / NOVEMBER, 2005 SUPPLEMENT

General
- ABO typing
- CBC count
- Direct antiglobulin test
- Blood cultures to rule out infection
- Biologic response modifiers (cytokines: IL-6, IL-8, others)?
- Plasma lipids?
- Cross-match?

Antibodies (previously implicated in parenthesis)
- Anti-HLA class I (A2, A9, B13, B27)
- Anti-HLA class II (DR3/5/6 CRG, DR1, DR4)†
- Antigranulocyte (anti-HNA1, anti-HNA2, anti-HNA3a, − 5b, anti-HNB1, anti-HNB2, −9a)
- Antimonocyte (HLA-II)
- Anti-IgA (not specified)

Mechanisms of transfusion-related acute lung injury (TRALI): Anti-leukocyte antibodies

Brian R. Curtis, MS, MT(ASCP)SBB; Janice G. McFarland, MD

Crit Care Med 2006 Vol. 34, No. 5 (Suppl.)

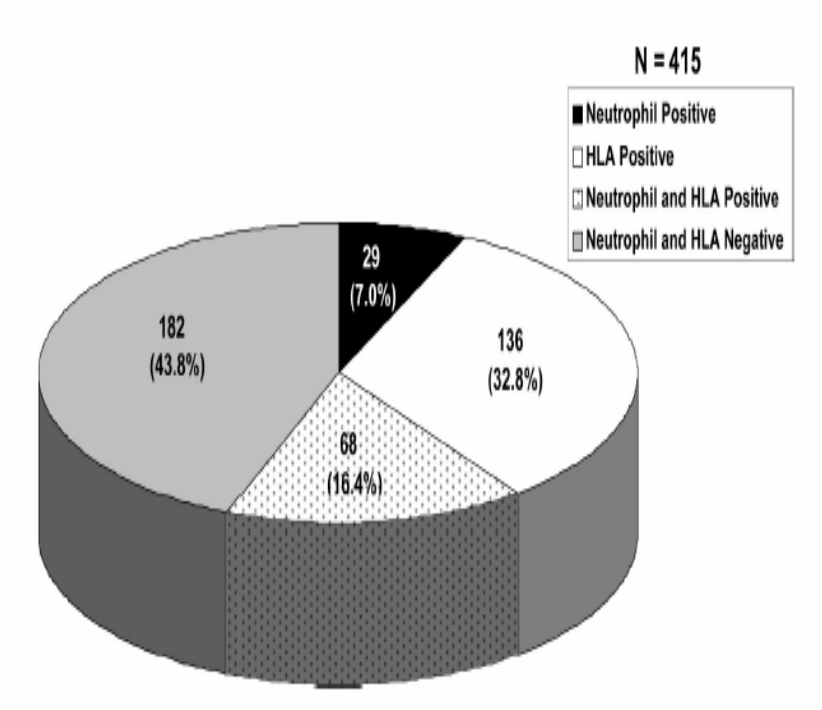


Table 1. Human neutrophil alloantigens

Alloantigen	Antigen Frequency (Caucasian), %	Glycoprotein Location	Alleles
HNA-1a (NA1)	54	FcγRIIIb, CD16	FCGR3B*01
HNA-1b (NA2)	88	FcγRIIIb, CD16	FCGR3B*02
HNA-1c (SH)	5	FcγRIIIb, CD16	FCGR3B*03
HNA-2a (NB1)	97	CD177	CD177*01
HNA-3a (5b)	97	Not known	Not known
HNA-4a(Mart)	92	MAC-1, CD11b	CD11B*1
HNA-5a(OND)	99	LFA-1, CD11a	CD11A*1

Pulmonary edema after transfusion: How to differentiate transfusion-associated circulatory overload from transfusion-related acute lung injury

Ognjen Gajic, MD; Michael A. Gropper, MD; Rolf D. Hubmayr, MD

Crit Care Med 2006 Vol. 34, No. 5 (Suppl.)

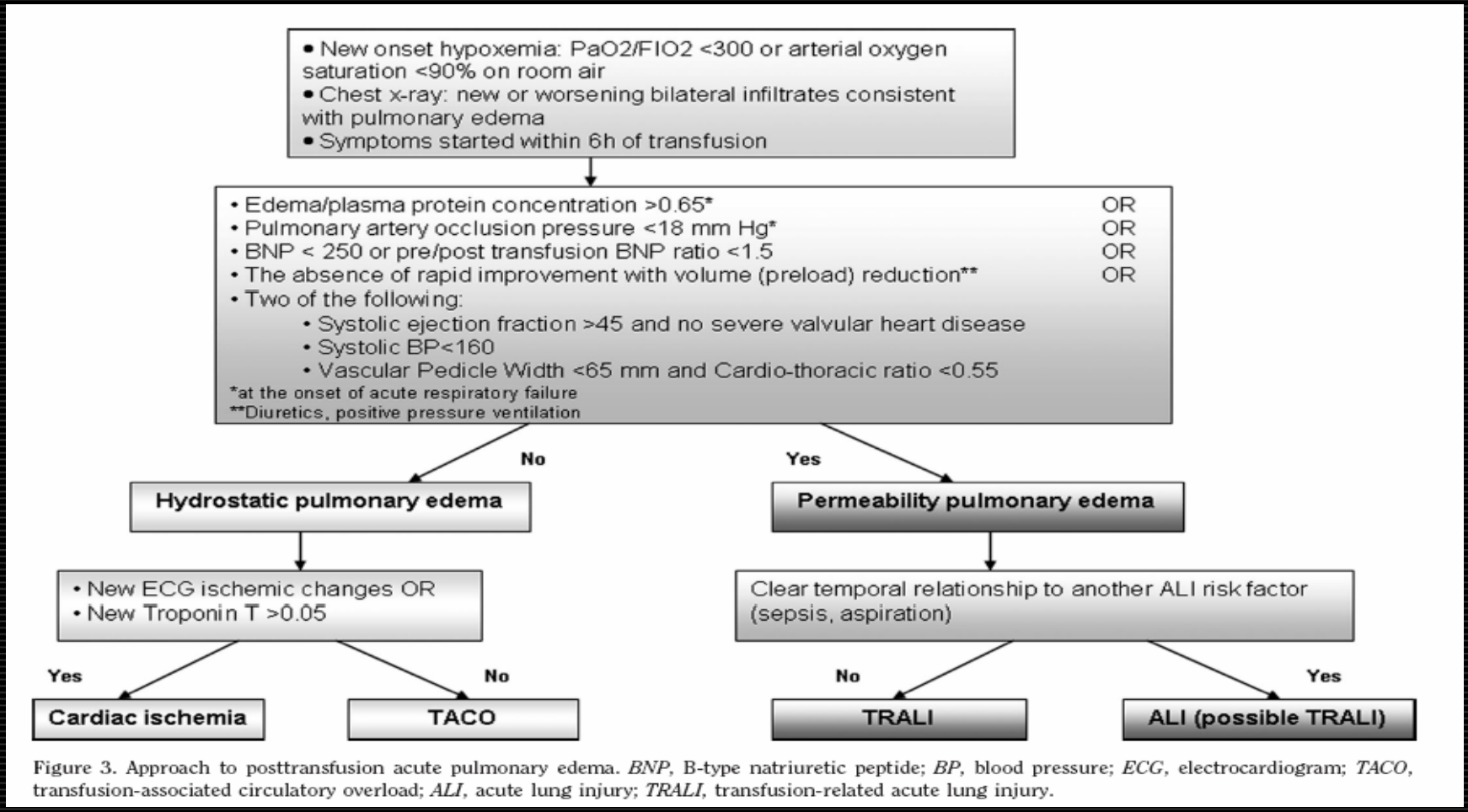


Figure 3. Approach to posttransfusion acute pulmonary edema. *BNP*, B-type natriuretic peptide; *BP*, blood pressure; *ECG*, electrocardiogram; *TACO*, transfusion-associated circulatory overload; *ALI*, acute lung injury; *TRALI*, transfusion-related acute lung injury.

Massive transfusion as a risk factor for acute lung injury: Association or causation?

Avery B. Nathens, MD, PhD, MPH

Crit Care Med 2006 Vol. 34, No. 5 (Suppl.)

Table 1. American-European Consensus Conference definition of acute lung injury (ALI) and acute respiratory distress syndrome (ARDS)

	Timing	Oxygenation	Chest Radiographs	Pulmonary Artery Occlusion Pressure
ALI	Acute onset	Pao_2/Fio_2 <300 mm Hg[a]	Bilateral infiltrates seen on frontal chest radiograph	≤18 mm Hg when measured or no clinical evidence of left atrial hypertension
ARDS	Acute onset	Pao_2/Fio_2 <200 mm Hg[a]	Bilateral infiltrates seen on frontal chest radiograph	≤18 mm Hg when measured or no clinical evidence of left atrial hypertension

Table 2. American-European Consensus Conference clinical risk factors for acute lung injury

Direct Lung Injury	Indirect Injury
Aspiration	Sepsis/septic shock
Diffuse pulmonary infection	Severe nonthoracic trauma
Near-drowning	Massive transfusion
Toxic inhalation	Cardiopulmonary bypass
Lung contusion	

Table 3. Risk factors for acute lung injury in clinical studies

	Randomized Controlled Trials	Cohort Studies	
	ARDS Clinical Trials Network (Refs. 7, 8) (n = 1408)	European Intensive Care Units (Ref. 9) (n = 463)	Massachusetts General Hospital (Mixed Intensive Care Units) (Ref. 11) (n = 221)[a]
Pneumonia	37	46	63
Sepsis	25	24	11
Aspiration	15	16	10
Trauma	10	22	9
Multiple transfusions	4	6	10
Other causes	10		

ARDS, acute respiratory distress syndrome.
[a]ARDS only.

CRITERIOS DIAGNÓSTICOS

Table I. Criteria for the clinical diagnosis of transfusion-related acute lung injury (TRALI).

TRALI Consensus Conference Committee 2004 and European Haemovigilance Network

- Acute respiratory distress
- Bilateral lung infiltrations in the chest radiograph
- Occurrence during or within 6-h after completion of transfusion
- No evidence of transfusion-associated circulatory overload/cardiogenic lung oedema
- Hypoxaemia (PaO_2/FiO_2 <300 mmHg or O_2 saturation <90% or other clinical evidence)
- New acute lung injury (ALI) and no other ALI risk factors present (aspiration, multiple trauma, pneumonia, cardiopulmonary bypass, burn injury, toxic inhalation, lung contusion, pancreatitis, drug overdose, near drowning, shock, severe sepsis)

If one or more ALI risk factors are present, possible TRALI should be diagnosed.

British Journal of Haematology, **136**, 788–799

Table 1. Canadian Consensus Conference proposed criteria for transfusion-related acute lung injury (TRALI).

Criteria for TRALI

- Acute lung injury (ALI)
  - Acute onset
  - Hypoxemia
    - In research setting:
      - Ratio of $PaO_2/FiO_2 \leq 300$ or
      - $SpO_2 < 90\%$ on room air
    - Non-research setting:
      - Ratio of $PaO_2/FiO_2 \leq 300$ or
      - $SpO_2 < 90\%$ on room air
      - Other clinical evidence of hypoxia
  - Bilateral infiltrates on frontal chest radiograph
  - No evidence of left atrial hypertension (i.e., circulatory overload)
- No preexisting ALI before transfusion
- During or within 6 hours of transfusion; and
- No temporal relationship to an alternative risk factor for ALI

Criteria for possible TRALI

- ALI
- No preexisting ALI before transfusion
- During or within 6 hours of transfusion; and
- A clear temporal relationship to an alternative risk factor for ALI

From Kleinman S, et al. Transfusion. 2004;44:1774-1789.

TRALI CLÍNICA

Table 1. Clinical features of transfusion-related acute lung injury

Clinical Features	Frequency
Dyspnea/respiratory distress requiring oxygen support	Virtually all
Requiring mechanical ventilation	~70%
Documented hypoxemia	Virtually all
Cyanosis	Very common
Hypotension	Majority
Fever	Very common
Hypertension	Unusual

TEMPORALIDAD DE LESIONES E IMÁGENES RADIOLÓGICAS

Irwin & Rippe´s Medicina Intensiva.2006.Cap.63

HORA / LESIÓN	0-6	12-72	96	10 D
CONTUSION LEVE/GRAVE	P	P	A/P	A/P
SDRA	A	P	P	P
SEG	A	P	P	A
BRONCO ASPIRACIÓN	A	P	P	A
EAP CARDIOGÉNICO	P	P	P	P
TRALI	P	P	A	A
EAP NEURÓGENO	P	P	A	A

P- PRESENTE
A- AUSENTE

TRALI LABORATORIO

- Neutropenia aguda transitoria
- Plaquetopenia
- Hipoalbuminemia

Leucopenia no explicada tras transfusión

Blood donor and component management strategies to prevent transfusion-related acute lung injury (TRALI)

D. C. Mair, MD; Nora Hirschler, MD; Ted Eastlund, MD

Crit Care Med 2006 Vol. 34, No. 5 (Suppl.)

Table 1. Possible donor-management strategies to prevent transfusion-related acute lung injury (TRALI)

Prevent donation by individuals at risk for developing or known to have leukocyte antibodies (e.g., multiparous females or transfused donors).
Ban further donation by donors who have been previously implicated in any confirmed TRALI case.
Ban future donations by donors who have been previously implicated in two or more cases of suspected TRALI.
Ban future donations by donors who have been previously implicated in a fatal case of TRALI.

Table 2. Blood-component-management strategies to prevent transfusion-related acute lung injury (TRALI)

Products from donors with known leukocyte antibodies or from donors likely to have those (e.g., multiparous females) are diverted to plasma for fractionation or are plasma-reduced and only the cells are transfused.
Leukoreduction of blood components is performed to prevent reactivity with recipient leukocyte antibodies.
Leukoreduction is performed to decrease the development of biologic response mediators in cellular components.
Storage time of cellular components is shortened to reduce accumulation of cytokines and other biologic response mediators.
Cellular components are washed.

TRALI
PROFILAXIS

- EVITAR TRANSFUSIONES INAPROPIADAS E INJUSTIFICADAS
- EVITAR LAS TRANSFUSIONES DE PFC DE DONANTE ÚNICO
- OPTIMIZAR TÉCNICAS DE AHORRO SANGUÍNEO

TRALI
TRATAMIENTO

- Soporte ventilatorio
- Ventilación de protección pulmonar
- Plasmaféresis
- ECMO

NO EVIDENCIA:

- Corticoides

CONTRAINDICADOS:

- Diuréticos

¿A qué es debido el sindrome confusional y la agitación posteriores?

- Hipoxemia
- Tipo anestesia en quirófano
- SEG
- Sindrome de abstinencia alcohólica
- Dolor
- Fármacos

Table 1. Medications associated with agitation in patients in the intensive care unit (8)

Antibiotics	Cardiac Drugs
Acyclovir	Captopril
Amphotericin B	Clonidine
Cephalosporins	Digoxin
Ciprofloxacin	Dopamine
Imipenen—cilastatin	Labetalol
Ketoconazole	Lidocaine
Metronidazole	Nifedipine
Penicillin	Nitroprusside
Rifampin	Procainamide
Trimethoprim—sulfamethoxazole	Propranolol
	Quinidine sulfate
Anticonvulsants	**Corticosteroids**
Phenobarbital	Dexamethasone
Phenytoin	Methylprednisolone
Miscellaneous Drugs	**Narcotic Analgesics**
Hydroxyzine	Codeine
Ketamine	Meperidine
Metoclopramide	Morphine sulfate
Theophylline	
Anticholinergics	
Benzodiazepines	
Nonsteroidal anti-inflammatory agents	

¿Alteraciones residuales cognitivas tras SEG?

- Alteración neurológica vs respiratoria
- No hay artículos que correlacionen la severidad del cuadro con resultados cognitivos postoperatorios.

TABLE II Results of Cognitive Testing Six and Eighteen Months Following Injury*

Test	Description	Results	
		6 Mo (%)	18 Mo (%)
WAIS-III	Full-scale intelligence quotient (four indices)	88	97
	Verbal comprehension	95	98
	Working memory	66	55
	Processing speed	10	39
	Perceptual organization	94	99
WMS-III	Logical memory (verbal) immediate	63	91
	Logical memory (verbal) delayed	84	98
	Family pictures (visual) immediate	63	25
	Family pictures (visual) delayed	63	36
Stroop	Sustaining and switching attention	19	100
Color Trails Test	Perceptual tracking, graphomotor skills, sustained and divided attention, sequencing and self-monitoring		
Part I		73	82
Part II		54	58
COWAT	Verbal initiation, self-monitoring, and speed	15	45

*The results of all tests are expressed as percentiles of the predicted premorbid cognitive score. WAIS-III = Wechsler Adult Intelligence Scale (third edition), WMS-III = Wechsler Memory Scale (third edition), Stroop = Color-Word Task of the Stroop Neuropsychological Screening Test, COWAT = Controlled Oral Word Association Test.

Andrew C.Gray et al.
The cognitive effects of fat embolus syndrome following femoral shaft fracture.
J Bone Joint Surg Am 2007;89:1092-1096

Manifestaciones neurológicas de la hipoxia aguda

SINDROME ABSTINENCIA OH
Diagnóstico

Interrupción o reducción de un consumo fuerte y prolongado de alcohol

Aparición posterior, en unas horas o días, de 2 o más de los siguientes síntomas: hiperactividad autonómica (sudoración, taquicardia, hipertensión); temblor de manos; insomnio; alucinaciones táctiles, auditivas o visuales transitorias; náuseas o vómitos; agitación psicomotriz; ansiedad; y crisis convulsivas de gran mal.

Los síntomas del criterio anterior producen alteraciones clínicamente significativas en la esfera cognitiva, social u ocupacional.

Los síntomas no son debidos a otra enfermedad médica o psiquiátrica definidas.

SINDROME ABSTINENCIA OH
Epidemiologia y Clínica

- Incidencia 13-70%
- Inicio:6-24 h
- Resolución: horas-dias
- Variabilidad interindividual
- Temblor
- Ansiedad
- Hipervigilancia
- Pérdida apetito
- Emesis
- Cefalea
- Diaforesis
- Alucinaciones
- Convulsiones

Delirium tremens

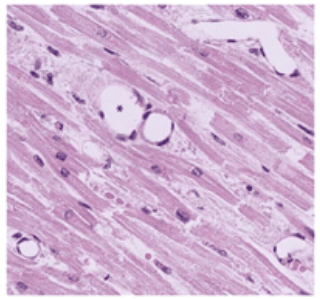
- Inicio: 1-4 dias
- Pico: 3- 4 dias
- Incidencia: 10%
- Duración: 5-10 dias
- Mortalidad:5-15%
- Desorientación
- Signos autonómicos
- Agitación grave
- Taquicardia
- HTA
- Fiebre

SINDROME ABSTINENCIA OH
Tratamiento

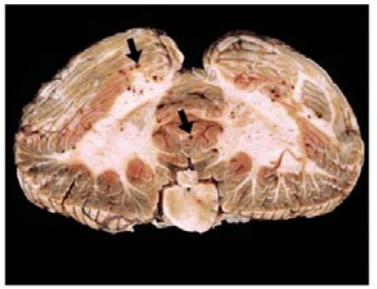
- Clordiazepóxido
- Diazepán
- Lorazepán
- Propanolol
- Carbamacepina
- Alcohol etílico
- Clonidina
- Ac.valproico
- Haloperidol
- Fenobarbital
- Fenitoina
- Clometiazol

RESUMEN

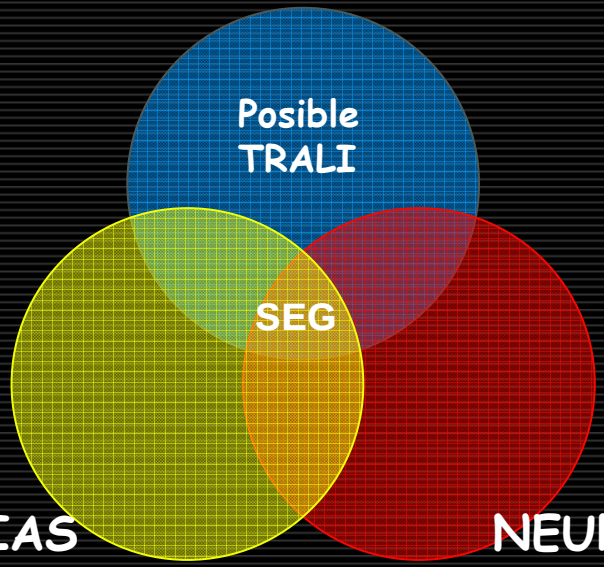
- Paciente joven,
- accidente de moto,
- fx fémur diafisaria,
- traumatismo torácico,
- sospecha broncoaspiracion,
- intervenido a las 48 h,
- politrasfundido o trasfundido,
- Hipoxemia a las 6 h?
- Hipoxemia a las 12h?
- Hipoxemia persiste a las 72 h?
- Hipoxemia a partir 2ª semana?



CONCLUSIÓN

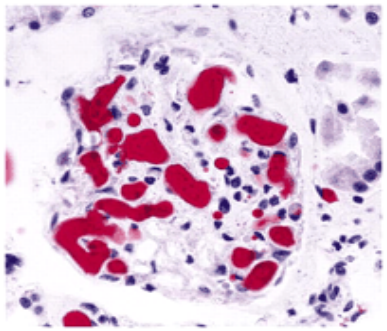


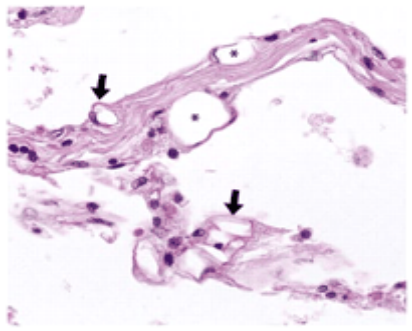
RESPIRATORIA

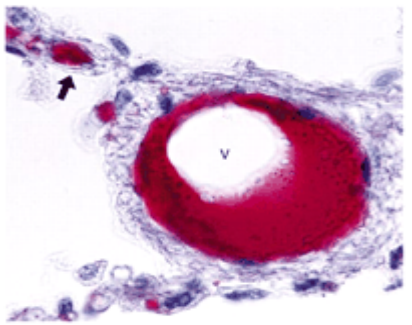


PETEQUIAS

NEUROLÓGICA







GRACIAS POR VUESTRA ATENCIÓN