Università degli Studi di Brescia
Scuola di Specializzazione in Anestesia, Rianimazione e Terapia Intensiva

Brescia 21 gennaio 2015

NEURO-CRITICAL CARE FOR EVERYDAY CLINICAL PRACTICE

Prof. N. Latronico

Università degli Studi di Brescia

What is Neurocritical Care Medicine

WHAT IS NEUROCRITICAL CARE MEDICINE

Primary neurological diseases

Any life-threatening *diseases* affecting:

Brain
Spinal cord
Peripheral nerve
NM transmission
Muscle

Systemic complications of neurologic diseases

SAH & Tako-tsubo
Coma & Pneumonia
Diabetes insipidus & Hypovolemia
SUDEP & Cardiac arrest
Spinal cord injury & Ventilator dependency
GBS & Neuromuscular respiratory failure

Neurologic complications of systemic diseases

Acute respiratory failure/brain hypoxia
Cardiac arrest/Hypoxic ischemic brain damage
DVT, embolization/brain infarction
Electrolyte imbalance/osmotic-demyelinating brain damage
Hypo- hyperthermia/metabolic brain changes
Immobility, sedation/delirium, disuse muscle atrophy
Shock, systemic hypotension/brain infarction
Systemic infections/meningitis
Sepsis/sepsis-associated CNS (delirium) & PNS (ICUAW) damage

WHAT IS NEUROCRITICAL CARE

PRIMARY NERVOUS SYSTEM DISORDERS

Epilepsy
Status epilepticus and related conditions

Stroke
Ischemic stroke, especially with impaired consciousness
Hemorrhagic Stroke: Intraparenchymal hemorrhages

Subarachnoid hemorrhage
with complications and impaired consciousness, vasospasm or other issues

Intraventricular hemorrhage

Traumatic brain and spinal cord injury

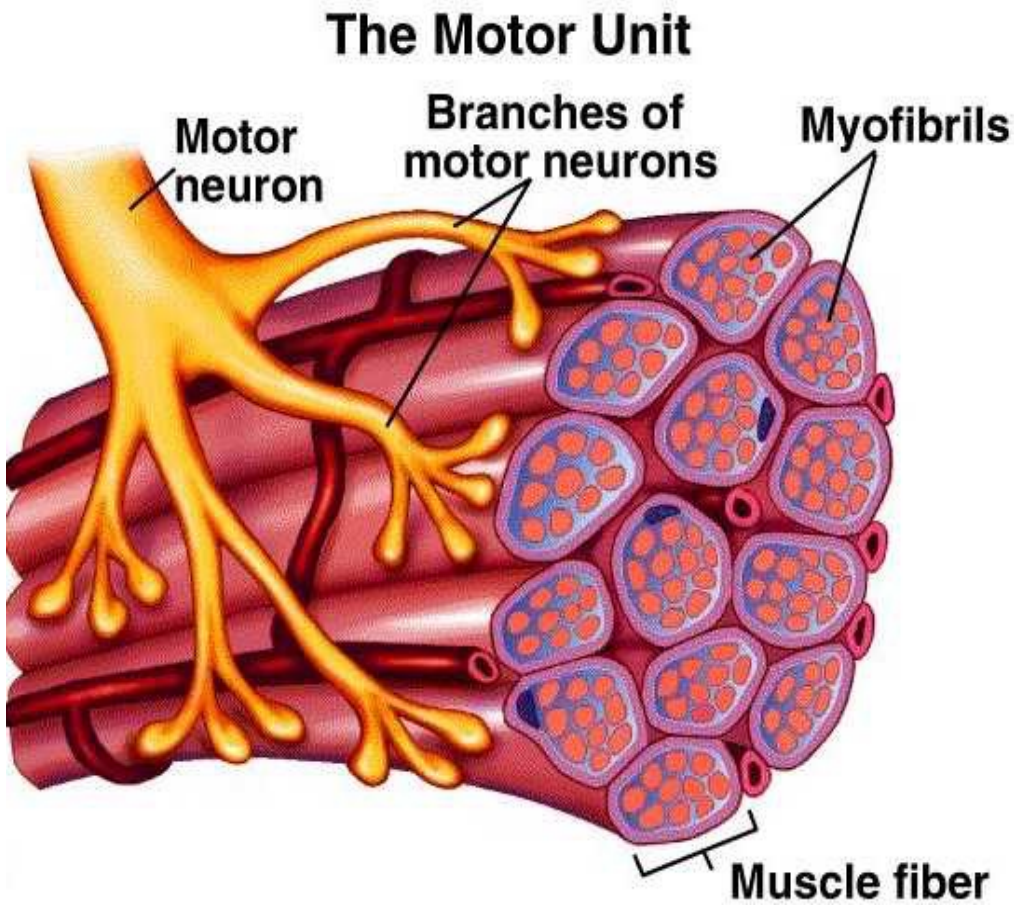
Central Nervous System Infections, Inflammations and Degenerations:
Bacterial, tuberculous, fungal, parasitic meningitis, encephalitides, demyelinating syndromes, auto-immune encephalitis and myelitis, motor neuron diseases

Neurosurgical post-operative brain and spinal cases

Modified from Thenayan EA, Bolton C, Jichici D, Savard M, Teitelbaum J, Young B, Zygun D. Neurocritical Care in Canada: Evolving Streams in a New Discipline. *Can J Neurol Sci* **2008;** 35: 405

WHAT IS NEUROCRITICAL CARE

PRIMARY ***MOTOR UNIT*** DISEASES



Localisation	Pre-existing	Previously Undiagnosed/New Onset	ICU Complication
Brain cortex and brainstem	Encephalitis Epilepsy Multiple sclerosis Vascular causes (brainstem infarction or haemorrhage; cerebral haemorrhage; ischaemic stroke)	Acute disseminated encephalomyelitis Encephalitis (including paralytic form of rabies) Multiple sclerosis Post-cardiac arrest encephalopathy Status epilepticus Tetanus Vascular causes	Post-cardiac arrest encephalopathy Status epilepticus (including nonepileptic) Vascular causes
Spinal cord (including anterior horn cells)	Amyotrophic lateral sclerosis Ischaemia Malformations (Arnold-Chiari) Poliomyelitis Post-polio syndrome Spinal muscular atrophy Trauma	Compression (tumour, infection, haematoma) Herpes zoster Ischaemia Transverse myelitis Surgery Tetanus Trauma West Nile virus poliomyelitis	Hopkins syndrome
Peripheral nerve	Alcohol abuse Chronic inflammatory demyelinating polyneuropathy Drugs* (bortezomib, cisplatin, dichloroacetate, epothilone, isoniazid, ixabepilone, leflunomide, linezolid, nitrofurantoin, oxaliplatin, pyridoxine, reverse transcriptase inhibitors, statins, taxanes, thalidomide, tumour necrosis factor-alpha blockers, vincristine) Guillain-Barré syndrome Hormonal disorders (acromegaly, hypothyroidism) Infections (diphtheria, HIV, Lyme disease) Tumours (carcinoma, lymphoma, multiple myeloma) Metabolic (diabetes, porphyria, tyrosinaemia, uraemia) Nutritional (thiamine deficiency) Sarcoidosis Toxic (acrylamide; heavy metals: arsenic, thallium, lead, gold; organophosphates; hexacarbons) Vasculitis (polyarteritis nodosa, lupus erythematous, rheumatoid arthritis, Churg-Strauss)	Acute intermittent porphyria Entrapment neuropathy Guillain-Barré syndrome HIV Tetanus Tick paralysis Toxic Vasculitis	Entrapment neuropathy Critical illness polyneuropathy
Neuromuscular junction	Botulism. Lambert-Eaton syndrome. Myasthenia gravis Drugs*. Anesthetic agents (desflurane, enflurane, halothane, isoflurane, nitrous oxide, opioids, propofol, sevoflurane). Antibiotics: *amynoglycosides*$^{+}$ (amikacin, clindamycin, gentamycin, kanamycin, lincomycin, neomycin, streptomycin, tobramycin); *fluoroquinolones* (ciprofloxacin, gemifloxacin, levofloxacin, lomefloxacin, moxifloxacin, norfloxacin, ofloxacin, and trovafloxacin); *macrolides* (azithromycin, erythromycin, telithromycin), *other antibiotics* (ampicillin, bacitarcin, polymyxins, tetracyclin, imipenem/cilastatin, penicillin, vancomycin). anti-arrhythmic agents (etafenone, peruvoside, procainamide, propafenone). antiepileptics (carbamazepine, gabapentin, phenytoin, trimethadione). beta-blockers** (atenolol, nadolol, oxprenolol, practolol, propranolol, sotalol, ophthalmic timolol). calcium-channel blockers** (amlodipine, felodipine, nifedipine, verapamil). corticosteroids***. Chemotherapics (doxorubicin, etoposide, cisplatin). H-2 receptor antagonists (cimetidine, ranitidine, roxatidine). quinolone derivatives (chloroquine, quinidine, quinine). Noncompetitive neuromuscular blocking agents^{+}. Psychotropic medications (amitriptyline, chlorpromazine, haloperidol, imipramine, lithium). Other drugs (interferon, penicillamine).	Hypermagnesaemia Myasthenia gravis Snake, scorpion and spider bites fish, shellfish, jellyfish and crab toxins Tetanus	Hypermagnesaemia Prolonged neuromuscular blockade
Muscle	Metabolic/congenital Mitochondrial myopathies Muscular dystrophies Periodic paralyses (muscle channelopathiyes) Polymyositis	Adult-onset acid maltase deficiency Hypo- and hyperkalaemia Hypophosphataemia Muscular dystrophies Polymyositis Pyomyositis Rhabdomyolysis Tetanus Toxic myopathies	Corticosteroid myopathy Critical illness myopathy Hypo- and hyperkalaemia Hypophosphataemia Propofol infusion syndrome Disuse atrophy Rhabdomyolysis

Latronico N, Fagoni N. ***Neuromuscular disorders and ICU acquired neuromuscular weakness***. In: Oxford Textbook of Neurointensive Critical Care. Smith M, Kofke AW, Citerio G (Eds). Oxford University Press, Oxford, England, 2014.

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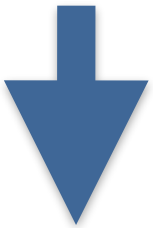

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WHAT IS NEUROCRITICAL CARE

SECONDARY NERVOUS SYSTEM DISORDERS

Central Nervous System Disorders

- Anoxic-Ischemic encephalopathy and cardioembolic strokes
- Drug intoxications and poisonings
- Endocrine disorders
- Fat embolism
- Fluid and electrolyte disturbances
- Hyper and hypoglycemia
- Hypertensive encephalopathies
- Hyperthermia and hypothermia
- Metabolic encephalopathies secondary of organ failure
- Nutritional deficiencies
- Pregnancy complications
- Sepsis and SIRS-associated encephalopathy (ICU delirium)
- Surgical complications
- Vascular complications of hematological and autoimmune disorders
- Withdrawal states

Modified from Thenayan EA, Bolton C, Jichici D, Savard M, Teitelbaum J, Young B, Zygun D. Neurocritical Care in Canada: Evolving Streams in a New Discipline. *Can J Neurol Sci* **2008;** 35: 405

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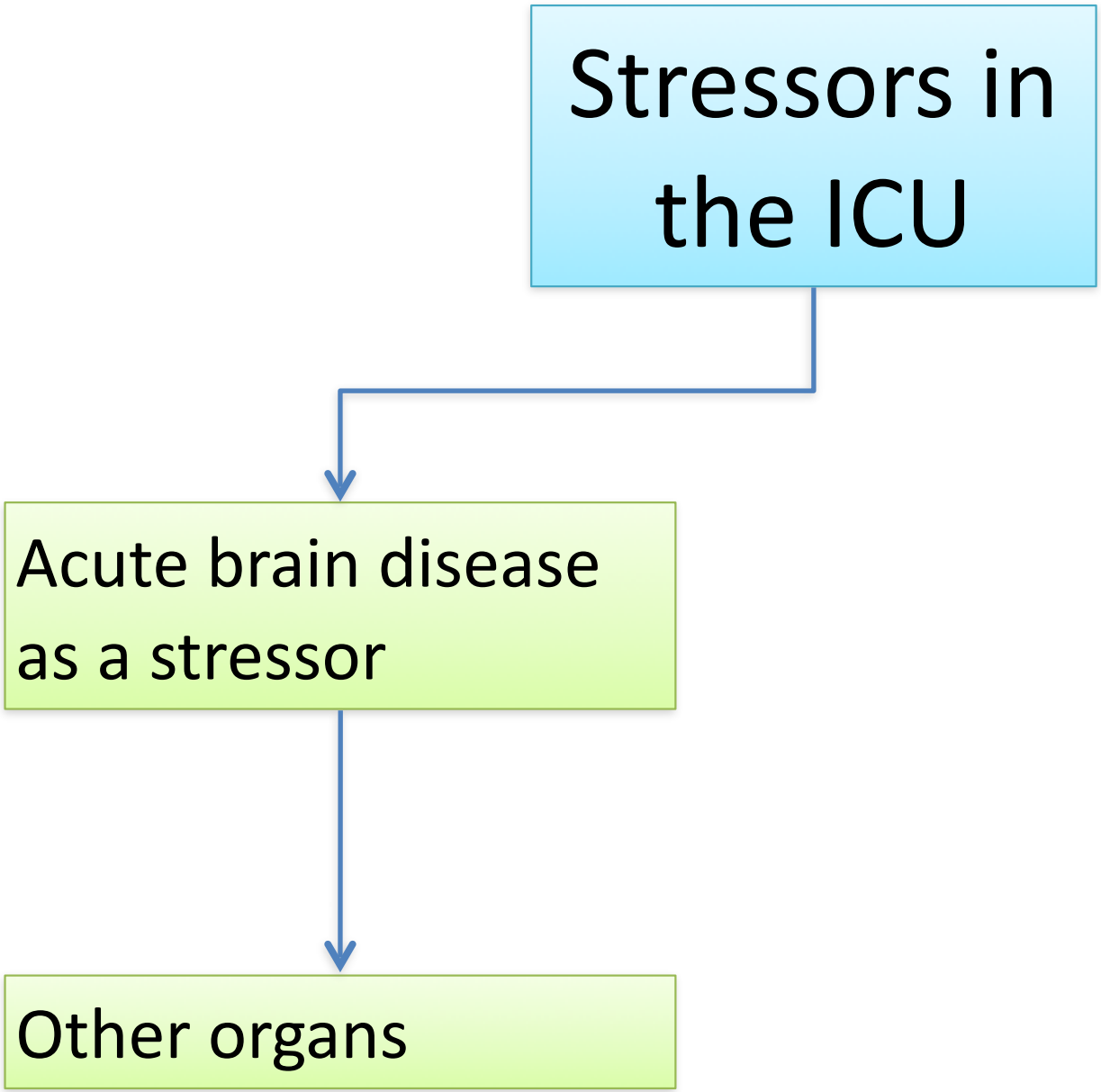

Stressors in the ICU
Acute brain disease as a stressor
Other organs

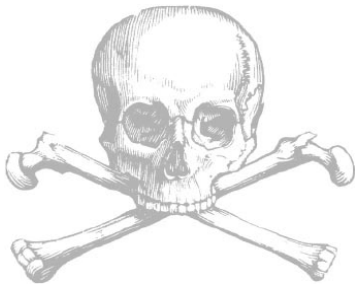
In records of anthropologists and others who have lived with primitive people in widely scattered parts of the world is the testimony that when subjected to spells or sorcery or the use of "black magic" men may be brought to death.

Among the natives of South America and Africa, Australia, New Zealand, and the islands of the Pacific, as well as among the negroes of nearby Haiti, "voodoo" death has been reported by apparently competent observers.

The phenomenon is so extraordinary and so <u>foreign to the experience of civilized people</u> that it seems incredible…

2A

"VOODOO" Death



Walter Bradford Cannon, MA, MD. From: "Voodoo" death. *American Anthropologist.* 1942;44(new series):169–181.

Great fear and great rage ….. Bring into action an elemental division of the nervous system, the so-called sympathetic or sympathico-adrenal division……

The suggestion which I offer, therefore, is that "voodoo death" may be real, and that it may be explained as due to shocking emotional stress—to obvious or repressed terror.

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 FEBRUARY 10, 2005 VOL. 352 NO. 6

Neurohumoral Features of Myocardial Stunning Due to Sudden Emotional Stress

Ilan S. Wittstein, M.D., David R. Thiemann, M.D., Joao A.C. Lima, M.D., Kenneth L. Baughman, M.D., Steven P. Schulman, M.D., Gary Gerstenblith, M.D., Katherine C. Wu, M.D., Jeffrey J. Rade, M.D., Trinity J. Bivalacqua, M.D., Ph.D., and Hunter C. Champion, M.D., Ph.D.

19 patients who presented with left ventricular dysfunction after sudden emotional stress (median age 63 years, 95% females).

Clinical presentations included chest pain, pulmonary edema, and cardiogenic shock.

Diffuse T-wave inversion and a prolonged QT interval occurred in most patients; mildly elevated serum troponin I levels;18 had no angiographic evidence of clinically significant coronary disease.

Severe left ventricular dysfunction was present on admission (median ejection fraction, 0.20; IQR 0.15 to 0.30) and rapidly resolved in all patients (ejection fraction at two to four weeks, 0.60).

Endomyocardial biopsy → mononuclear infiltrates and “contraction-band necrosis”.

Plasma catecholamine levels at presentation were markedly elevated.

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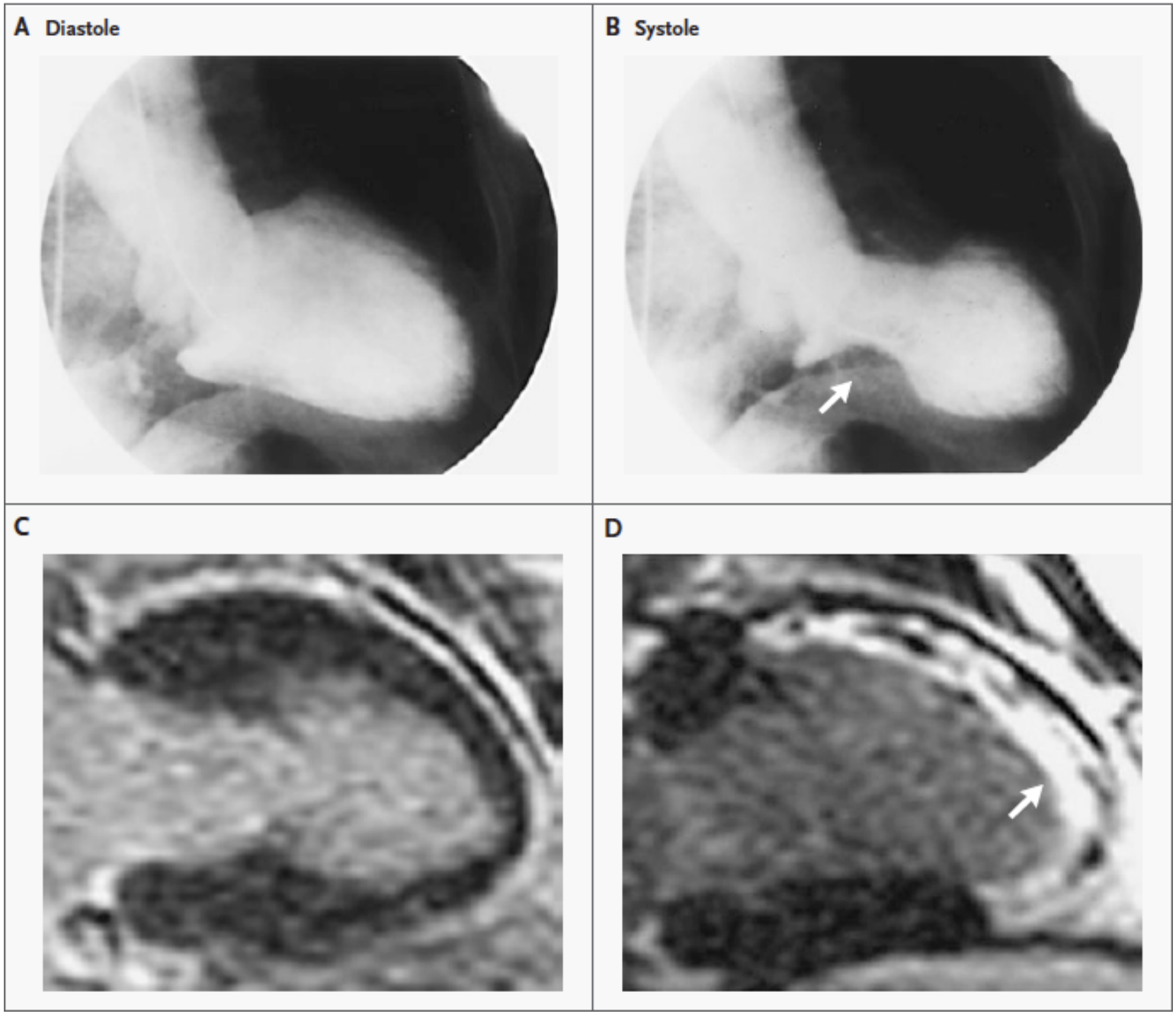
Emotional stress can precipitate severe, reversible left ventricular dysfunction in patients without coronary disease.

Exaggerated sympathetic stimulation is probably central to the cause of this syndrome.

2A

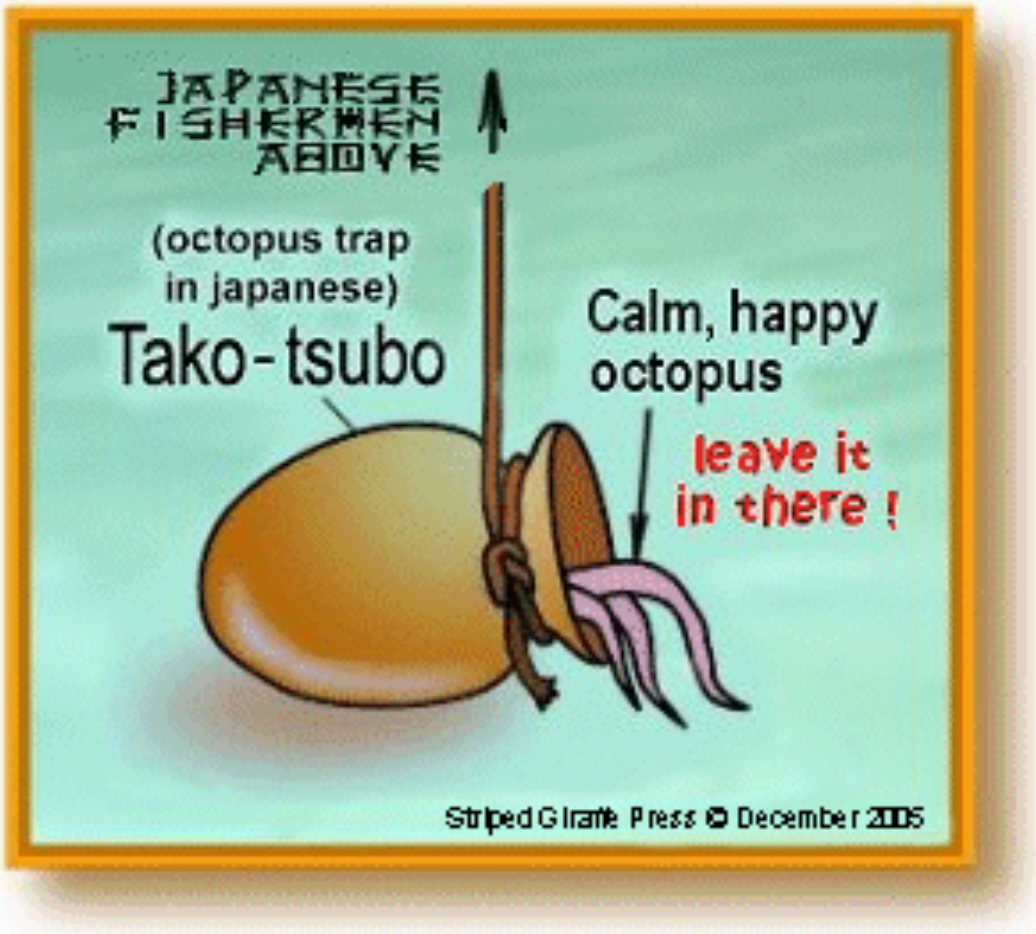


Wittstein IS, et al. N Engl J Med 2005; 352:539-42–1397

Tako-tsubo

Lee VH, et al. *J Neurosurg* **2006**; 105:264-270

Takotsubo cardiomyopathy



http://www.takotsubo.com/

Donna muore di crepacuore due giorni dopo il marito Lui si era spento in auto sabato per un malore Oggi i funerali fianco a fianco

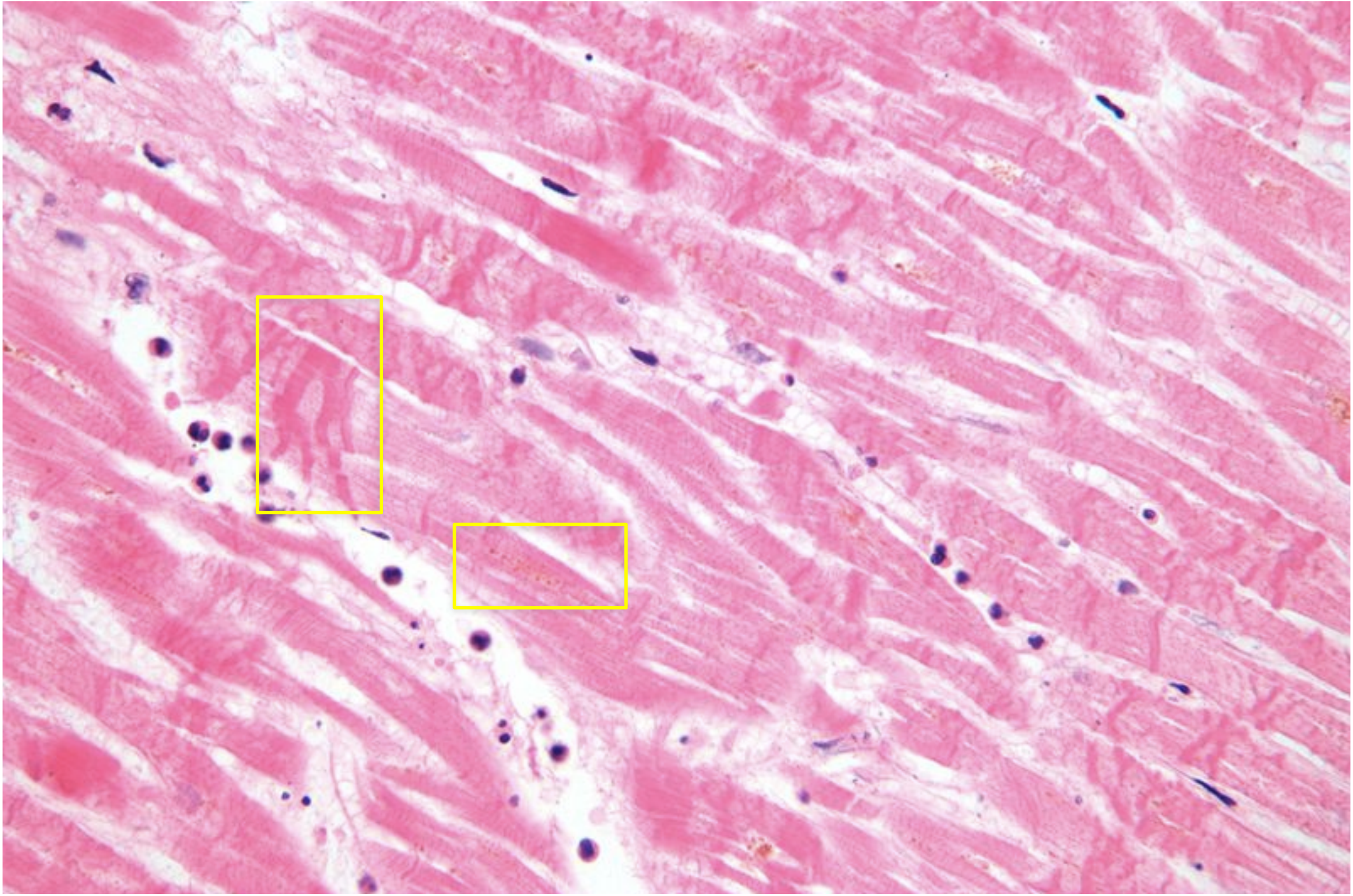
Era stato trovato morto in auto a Ceciliano: un malore, anche in base all'autopsia. La moglie non ha retto il colpo. E oggi saranno insieme per l'ultimo addio

Morire insieme a pochi giorni di distanza

http://www.lanazione.it/arezzo/cronaca/2013/09/19/952342-donna_muore_crepacuore.shtml

You really can die of a broken heart, new research suggests. Simon Monjack, 39, died five months after his wife, Brittany Murphy, died of pneumonia

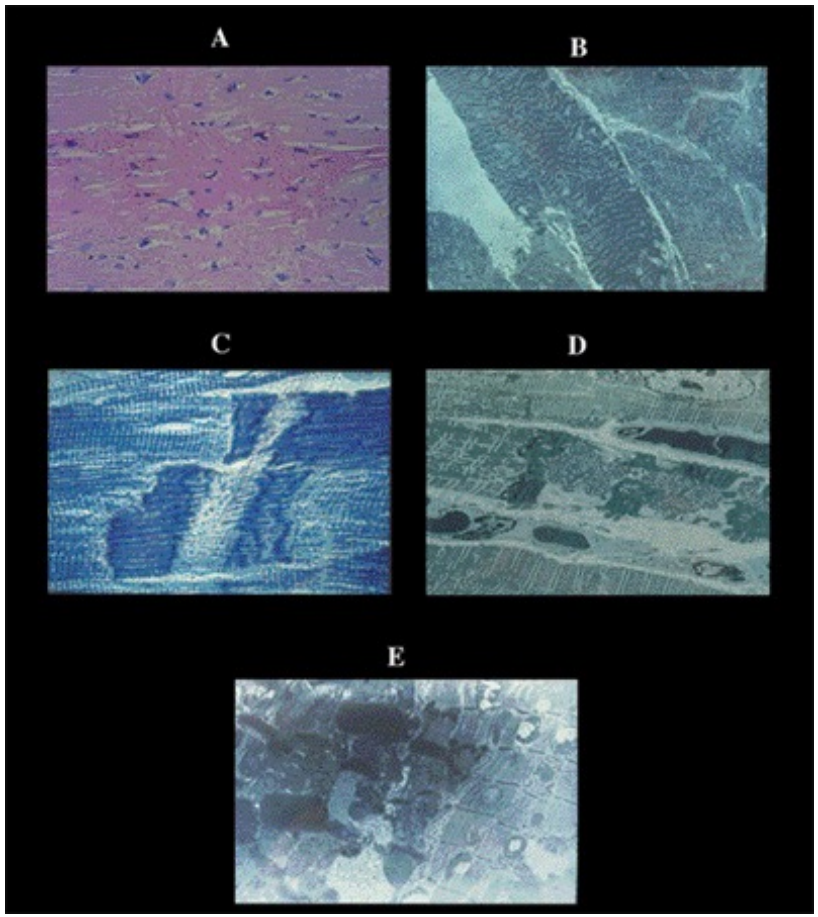
http://www.dailymail.co.uk/health/article-2566854/Grief-broken-heart-doubles-risk-heart-attack-stroke.html



CONTRACTION BAND NECROSIS

http://commons.wikimedia.org/wiki/File:MI_with_contraction_bands_very_high_mag.jpg *accessed 21-09-14*

CONTRACTION BAND NECROSIS



<u>Coagulative myocytolysis or contraction band necrosis or catecholamine necrosis (CN).</u>

Pancellular lesion involving the whole myocardial cells (*catecholamine infusion, pheochromocytoma*).

A) histological view of a CN focus.

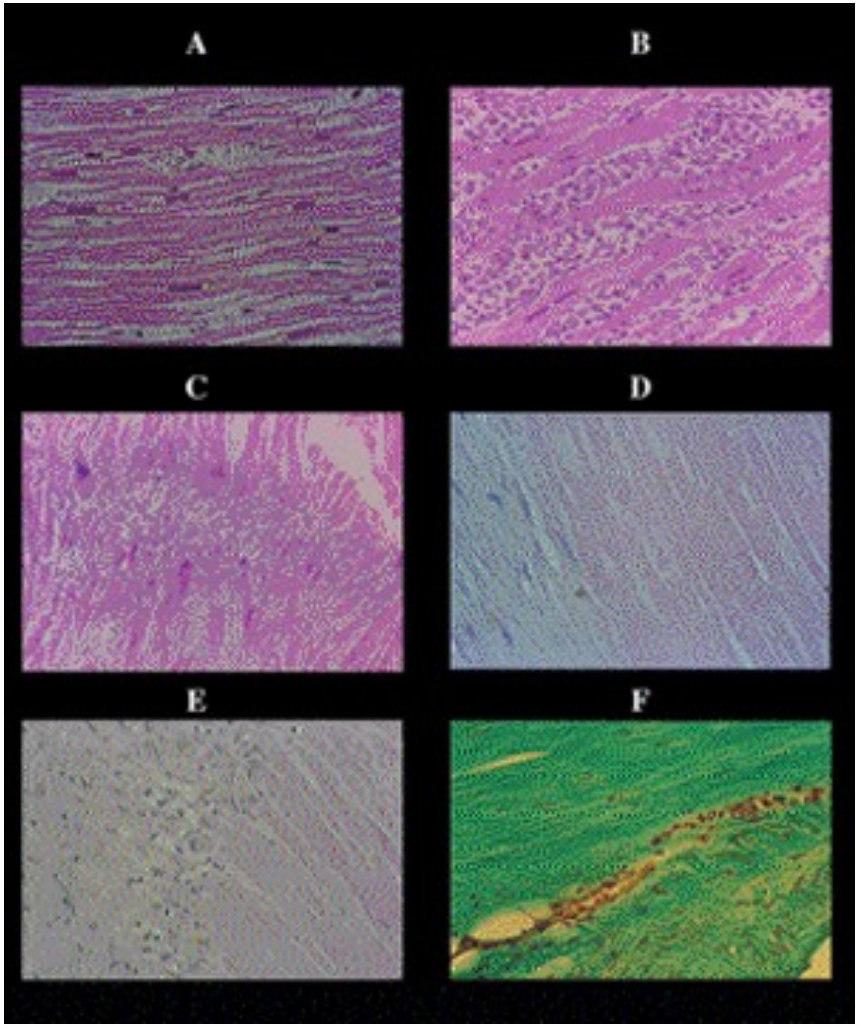
B) ultrastructural hypercontraction with extremely short sarcomeres and highly thickened Z lines and focal myofibrillar rhexis.

C) rupture of a hypercontracted myocell.

EM view of pathological bands **(D)** formed by segments of hypercontracted and coagulated sarcomeres *(intravenous infusion of catecholamines in dogs)*.

Baroldi G, Bigi R, Cortigiani L. Ultrasound imaging versus morphopathology in cardiovascular diseases. Myocardial cell damage. *Heart Views* **2005**; 3:32

INFARCT NECROSIS



Infarct necrosis. The first change is loss of contraction with stretching of the myocardium in flaccid paralysis, resulting in a very early elongation of sarcomeres and nuclei **(A)** already visible within 30 minutes in experimental infarction.

(B), polymorphonuclear leukocyte infiltration from the periphery of the infarct after 6–8 hours.

In the largest infarcts this infiltration arrests, along a line (maximal myocardial stretching in central part of infarct?) with occasional abscess-like formation **(C)**.

This infiltration disappears by lysis of the leukocytes, without evidence of myocellular colliquation or destruction **(D)**.

The myocardial cells maintain their sarcomeric registered order even in terminal healing phase.

The repair process is carried out by macrophagic digestion **(E)** – and not by granulation tissue – ending in a compact and dense scar **(F)**.

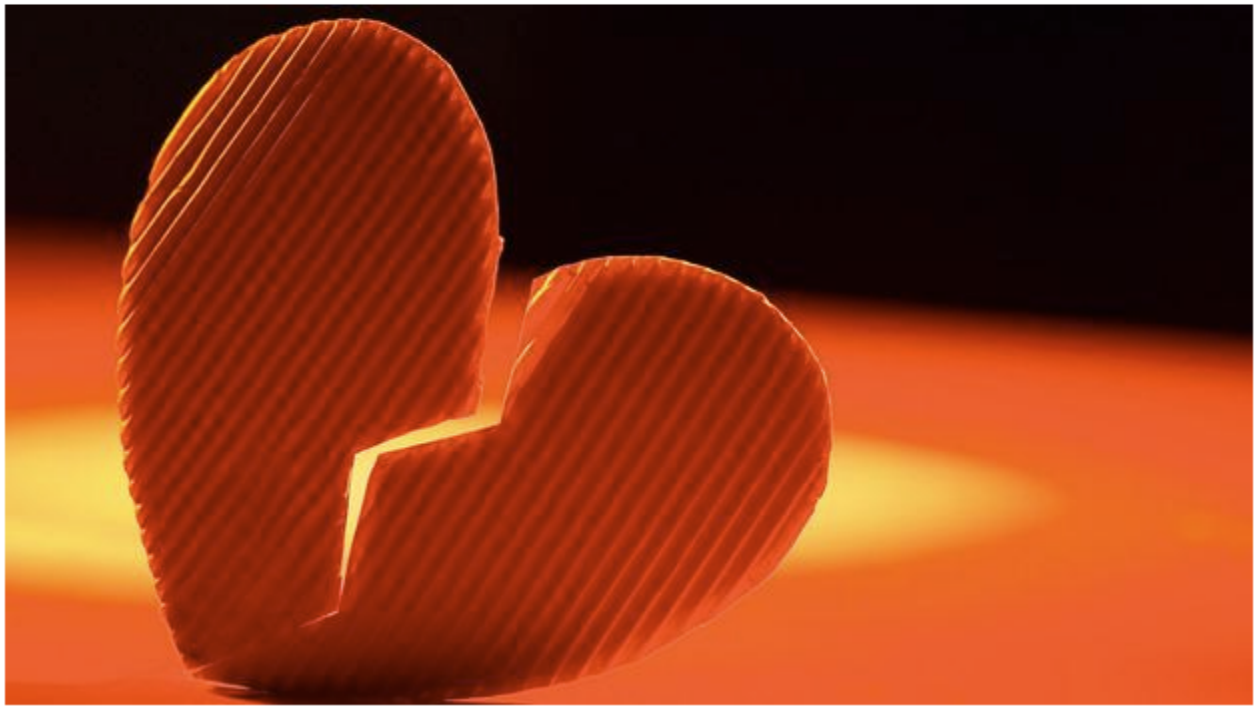
Baroldi G, Bigi R, Cortigiani L. Ultrasound imaging versus morphopathology in cardiovascular diseases. Myocardial cell damage. *Heart Views* **2005**; 6: 32

UNIQUELY HUMAN | 31 March 2014

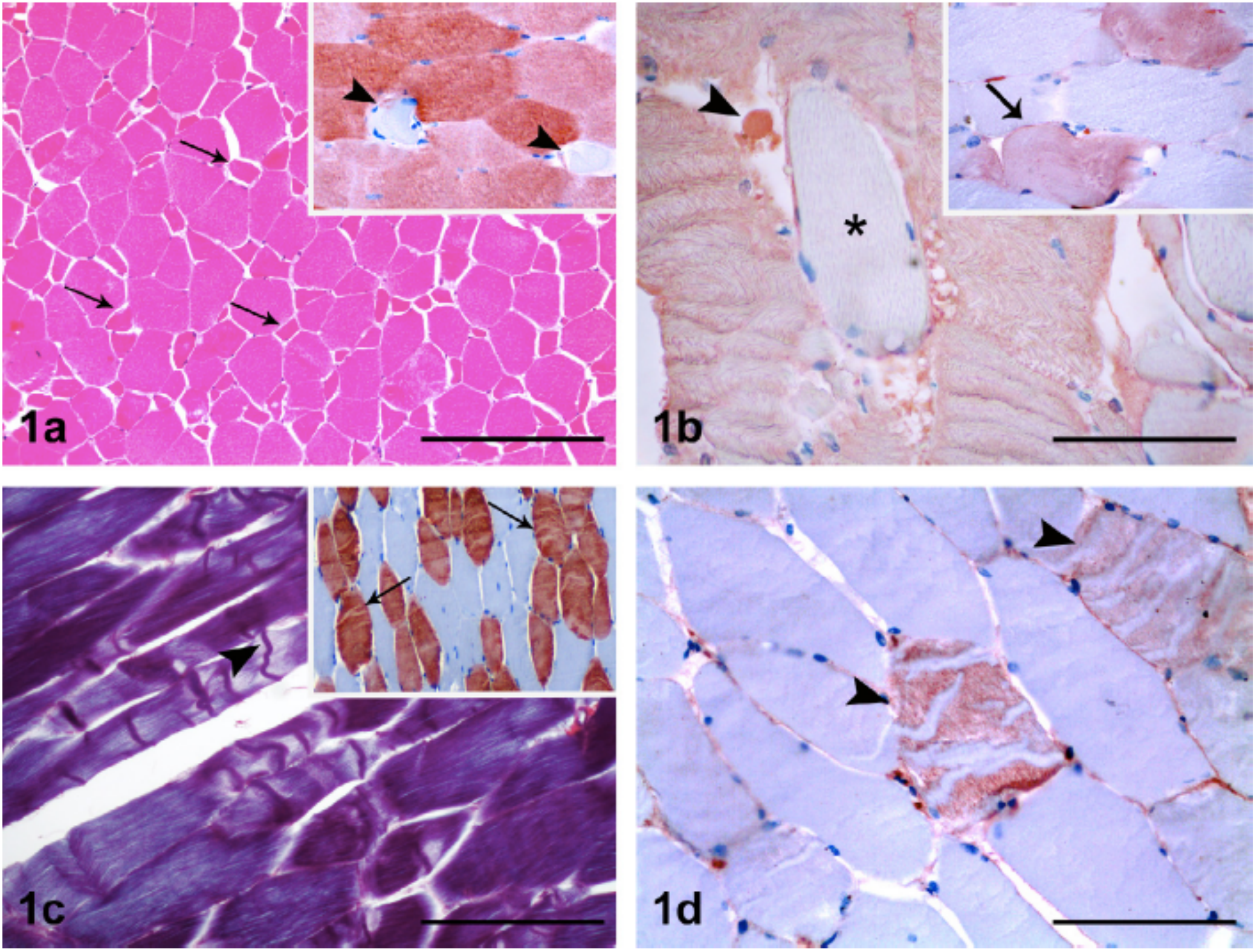
Can you die of a broken heart?

Jason G Goldman

Health | Science & Environment | Animal | Biology | Death | Hospital



http://www.bbc.com/future/story/20140331-can-you-die-of-a-broken-heart


1a
1b
*
1c
1d

Capture myopathy in live-stranded cetaceans

P. Herráez, et al.
Vet J **2013**; 196:181-188

A group of 51 cetaceans that had been stranded alive on the coasts of the Canary Islands, experienced human capture/rescue interactions and then died, were necropsied over a 12-year period.

CAPTURE MYOPATHY

In their book ***Zoobiquity***, Kathryn Bowers and Barbara Natterson-Horowitz described this attitude: "Among many physicians, the idea that emotions could cause actual physical events within the architecture of the heart was viewed with nearly the same sideways glance as an interest in healing crystals or homeopathy. Real cardiologists concentrated on real problems you could see: ***arterial plaque, embolising blood clots, and rupturing aortas***. Sensitivity was for psychiatrists."

Despite this, the evidence that extreme emotions can impact the heart goes back decades – not only among humans. It was wildlife biologists and veterinarians who first noticed that extreme emotions can wreak havoc on body physiology. By the mid-20th Century, they noticed that a curious thing happens when an animal experiences a sudden jolt of life-or-death fear. When it's caught by an advancing predator, adrenaline fills the bloodstream to such an extent that the blood almost becomes like a poison, damaging the animal's muscles, including the heart. It's called "***capture myopathy***".

http://www.bbc.com/future/story/20140331-can-you-die-of-a-broken-heart

nature International weekly journal of science

Letters to Nature

Nature **247**, 577 (22 February 1974) | doi:10.1038/247577a0

Possible Therapy for Capture Myopathy in Captured Wild Animals

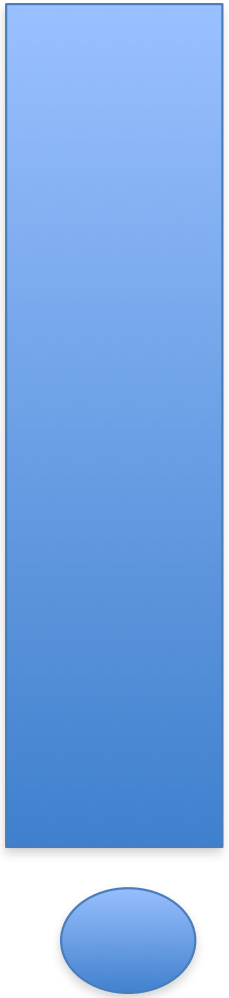
A. M. HARTHOORN[*], K. VAN DER WALT[†] & E. YOUNG[‡]

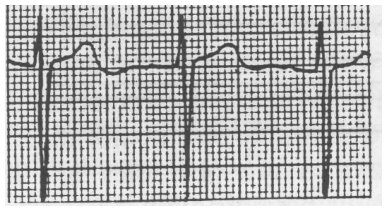
1. [*]Nature Conservation Division, P. Bag X*209*, Pretoria
2. [†]Faculty of Veterinary Science, Onderstepoort
3. [‡]Division Veterinary Services, Kruger National Park, P.O. Skukuza, South Africa

CAPTURE myopathy (so-called overstraining disease) in wild animals has gained increasing prominence over the past decade as attempts to capture remaining nuclei of rare species for relocation and restocking, are rendered abortive by high death rates. The proportion of deaths is usually highest in the calves and in gravid females. High death rates in diminishing animal species such as tsessebe (*Damaliscus lunatus*) have occurred in spite of all precautions, such as the use of a helicopter to reduce the time between alerting the subject animal and the placement of a syringe containing suitable immobilising compounds.

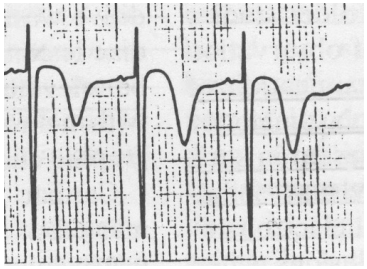
▲ Top

NEUROCRITICAL CARE?

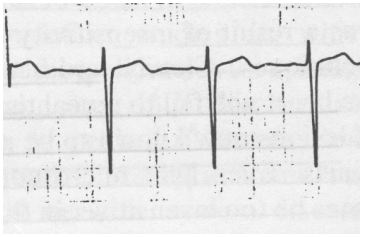




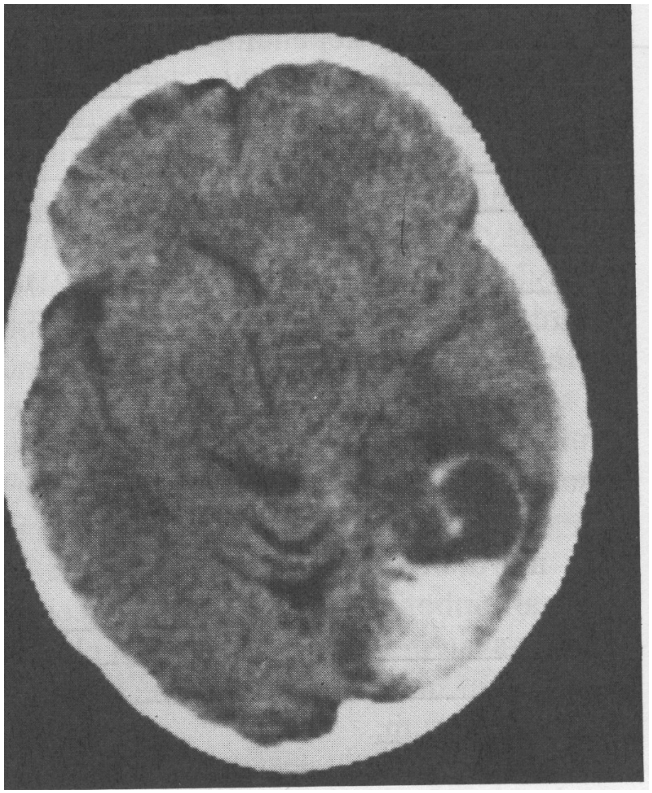
A. In the ER



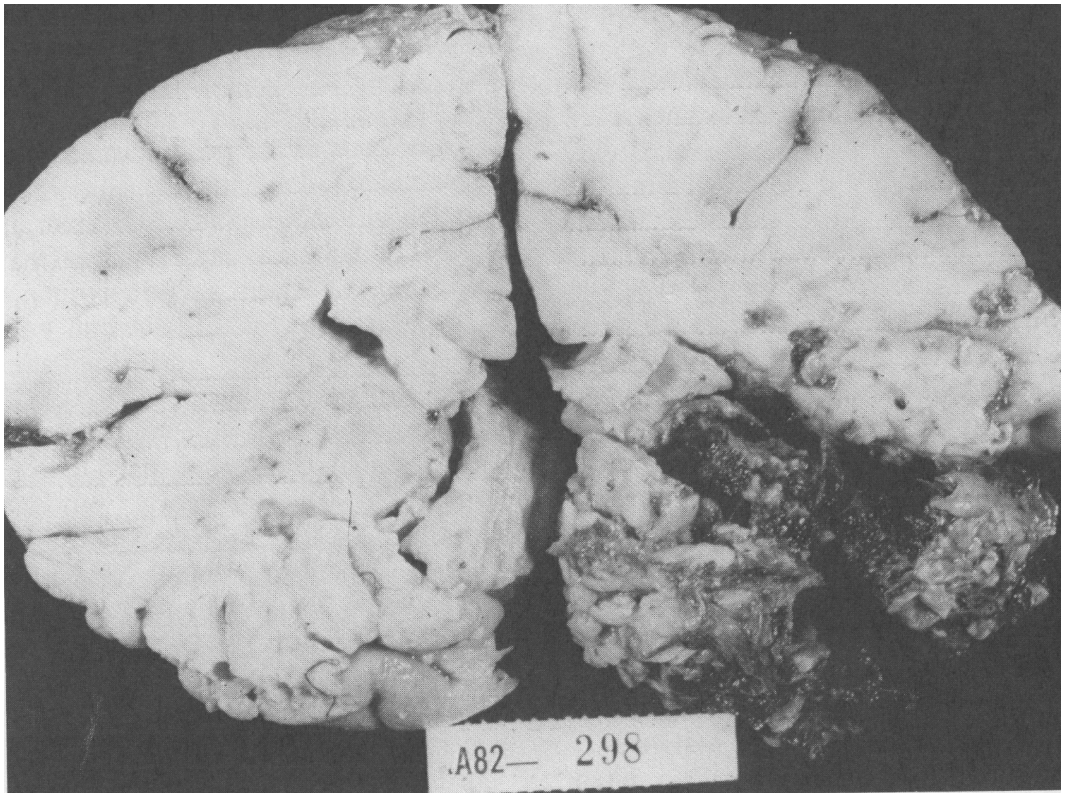
B. 7 days later, mild hemiparesis



C. Brain death

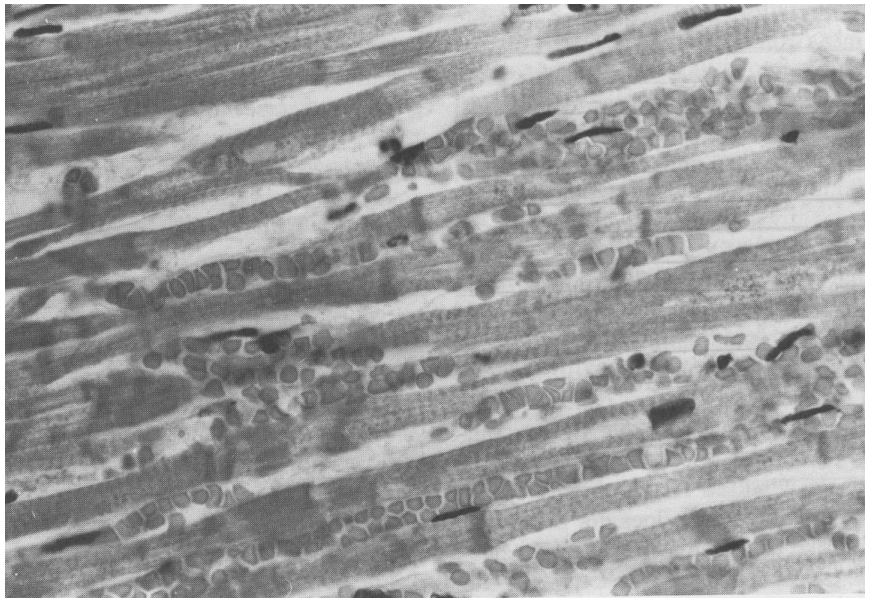


D. Cerebral hemorrhage in the left hemisphere



E. Gross brain pathology showing hemorrhage into a metastatic tumor

Ropper AH. *Neurological and neurosurgical Intensive Care*. 3° Ed. Raven Press, New York **1993**



D. Contraction bands with hemorrage

E. Contraction bands with intensive calcification of the lesion

Ropper AH. *Neurological and neurosurgical Intensive Care*. 3° Ed. Raven Press, New York **1993**

Non-neurologic organ dysfunction in severe traumatic brain injury*

David A. Zygun, MD, MSc, FRCPC; John B. Kortbeek, MD, FRCSC; Gordon H. Fick, PhD; Kevin B. Laupland, MD, MSc, FRCPC; Christopher J. Doig, MD, MSc, FRCPC

Crit Care Med 2005;33: 654-660

Non-neurologic organ dysfunction is common in patients with severe traumatic brain injury and is independently associated with worse outcome.

Continuing Medical Education Article

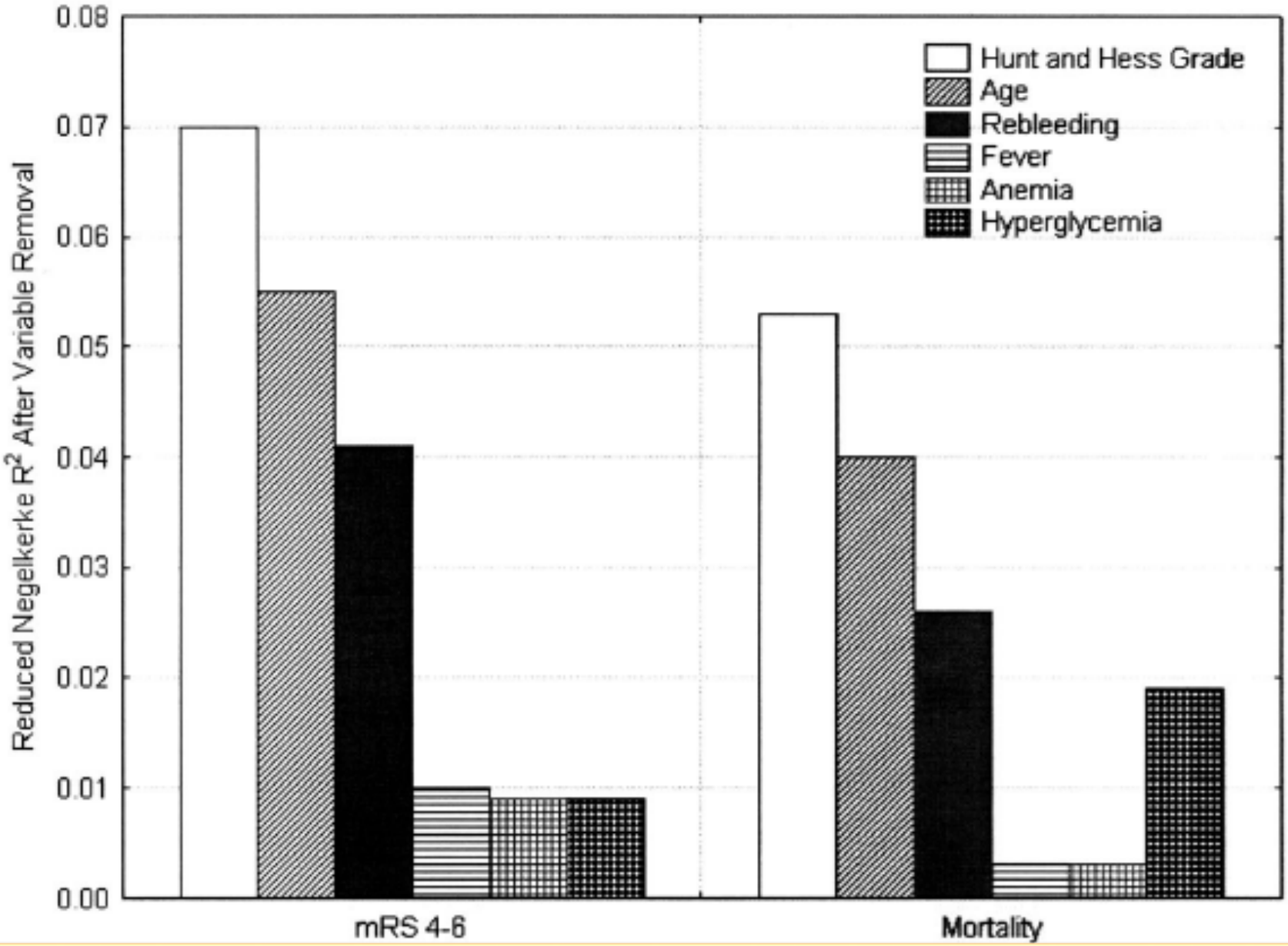
Impact of medical complications on outcome after subarachnoid hemorrhage*

Katja E. Wartenberg, MD; J. Michael Schmidt, PhD; Jan Claassen, MD; Richard E. Temes, MD; Jennifer A. Frontera, MD; Noeleen Ostapkovich, MS; Augusto Parra, MD, MPH; E. Sander Connolly, MD; Stephan A. Mayer, MD

Crit Care Med 2006; 34: 617-623

We identified three common medical complications—fever, hyperglycemia, and anemia—that significantly predict poor outcome after subarachnoid hemorrhage.

Fever, anemia, and hyperglycemia affect 30% to 54% of patients with SAH and are significantly associated with mortality and poor functional outcome



Wartenberg KE, et al. Impact of medical complications on outcome after subarachnoid hemorrhage. *Crit Care Med* **2006**; 34:617–623

Case report

A 69-year-old woman admitted to the ED because of chest pain and diaphoresis. She had a 1-year history of untreated hypertension.

On admission: awake and collaborative, with no evidence of focal neurological signs, complaining general malaise and dyspnoea.

Blood pressure was 170/80 mmHg, heart rate was 100 beats /min; temperature was 37 °C, skin was cold and wet. On chest auscultation there were widespread crackles in both lungs.

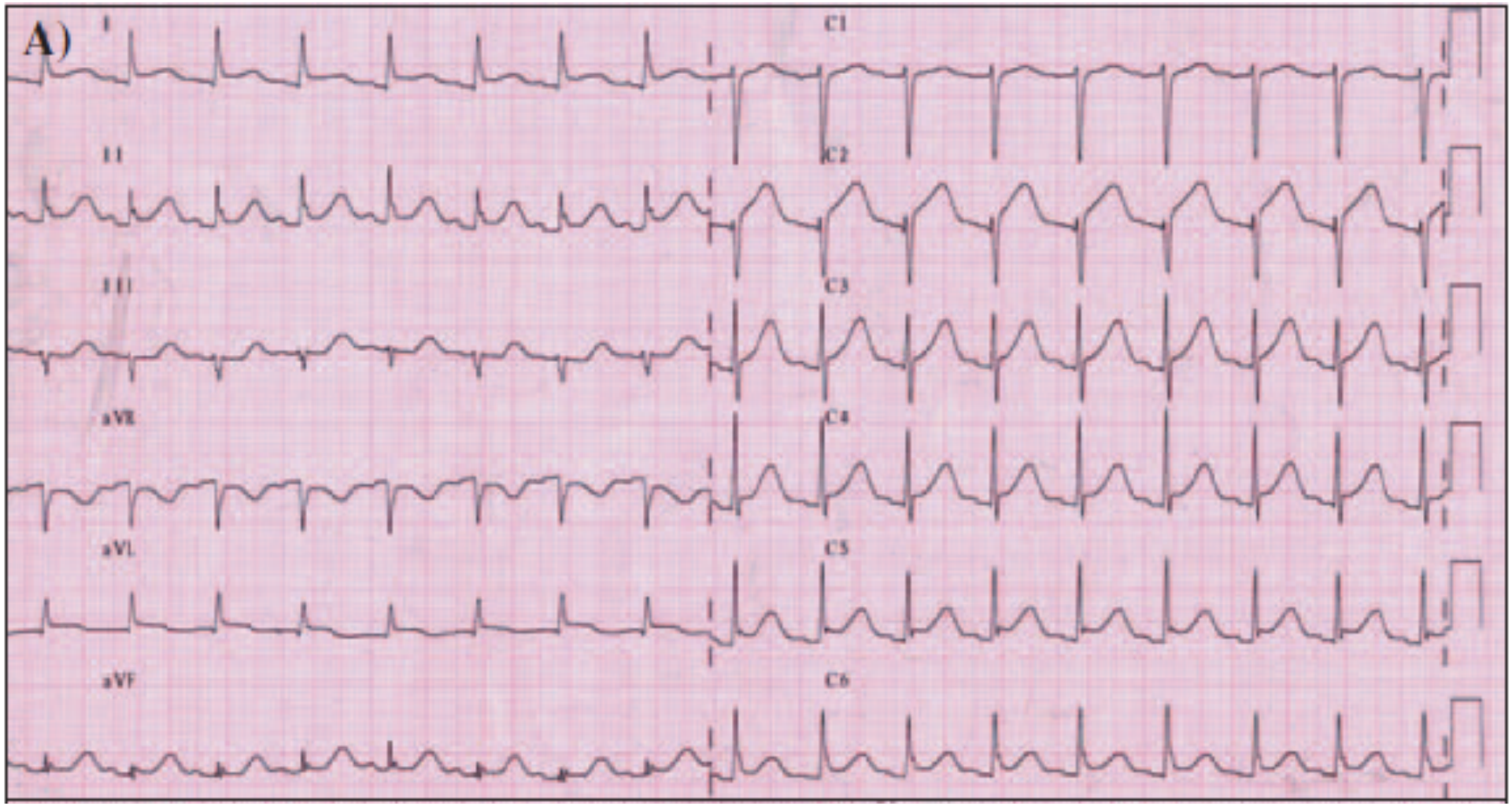
Arterial blood gas analysis showed hypoxemia (pO2: 64 mmHg), hypocapnia (pCO2: 35 mmHg), pH: 7.4; HCO_3^- 22 mmol/l.

The patient rapidly developed acute pulmonary edema requiring oro-tracheal intubation.

Cardiac enzymes 1 hour after the onset of symptoms were normal.

D'Aloia A, et al. Monaldi Arch Chest Dis 2007; 68:44-47

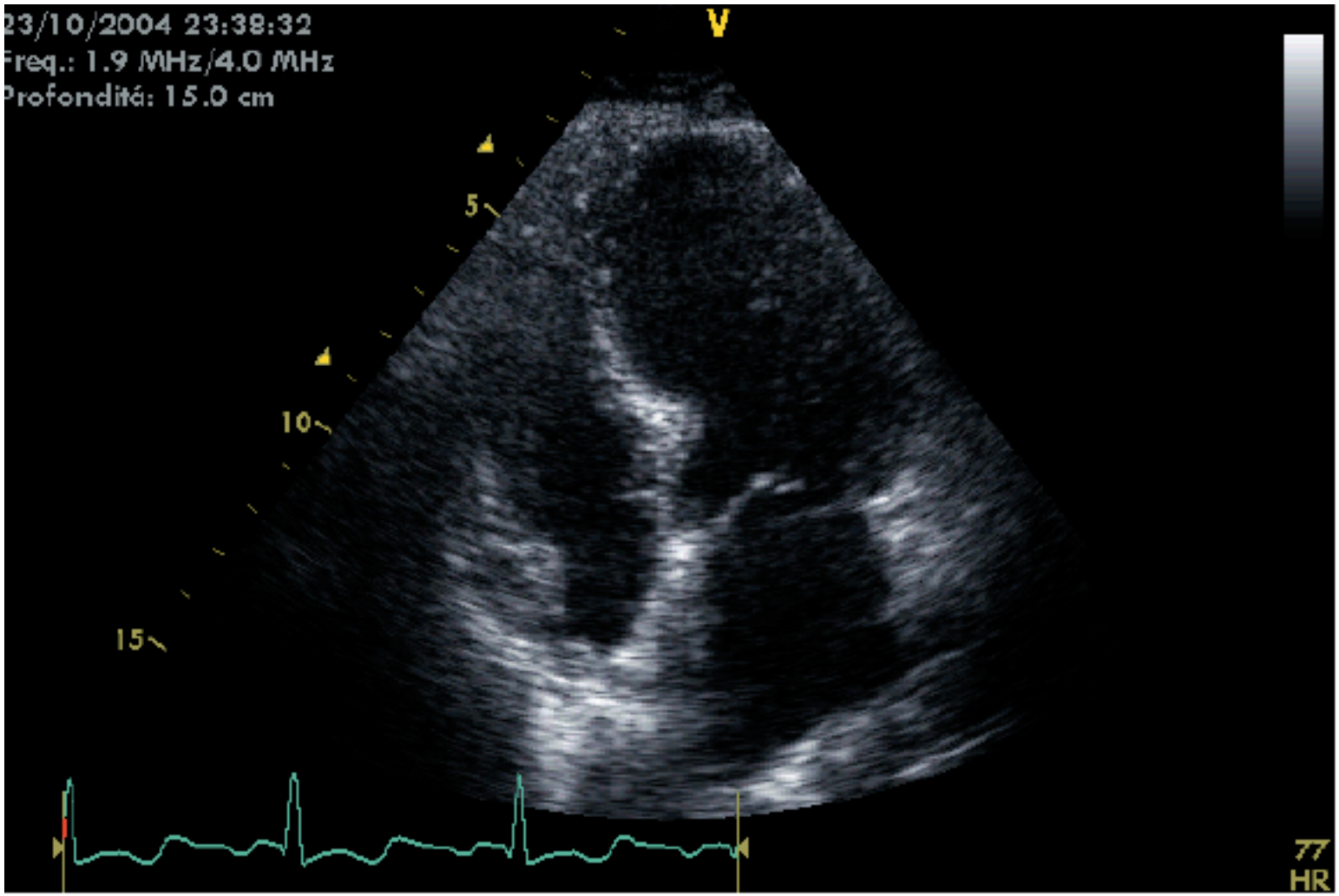
Ecg revealed normal sinus rhythm with ST-segment elevation in leads V2 through V6, D1, aVL.



D'Aloia A, et al. Monaldi Arch Chest Dis 2007; 68:44-47

Transthoracic echocardiography showed a mildventricular hypertrophy with a moderate to severe left ventricular systolic dysfunction (LVEF 35%) due to a marked diskinesia of mid-distal septal antero-lateral and infero-posterior wall segments.

Left ventricular filling pattern on transmitral and pulmonary venous flow was restrictive, indicating high left atrial pressure.



D'Aloia A, et al. Monaldi Arch Chest Dis 2007; 68:44-47

Based on the clinical presentation and features of acute coronary syndrome (with ST segment elevation) the patient received oxygen, nitrates, aspirin, heparin and abciximab bolus.

Urgent coronary angiography showed no evidence of coronary artery stenoses, thrombi, or spasm.

Due to neurological deterioration, urgent brain CT was performed showing SAH. Cerebral angiography showed a right vertebral artery aneurysm, which was treated with coiling.

The patient recovered uneventfully.

D'Aloia A, et al. Monaldi Arch Chest Dis 2007; 68:44-47

Case report

neurocritical care society Neurocrit Care (2009) 10:213–217
DOI 10.1007/s12028-008-9164-x

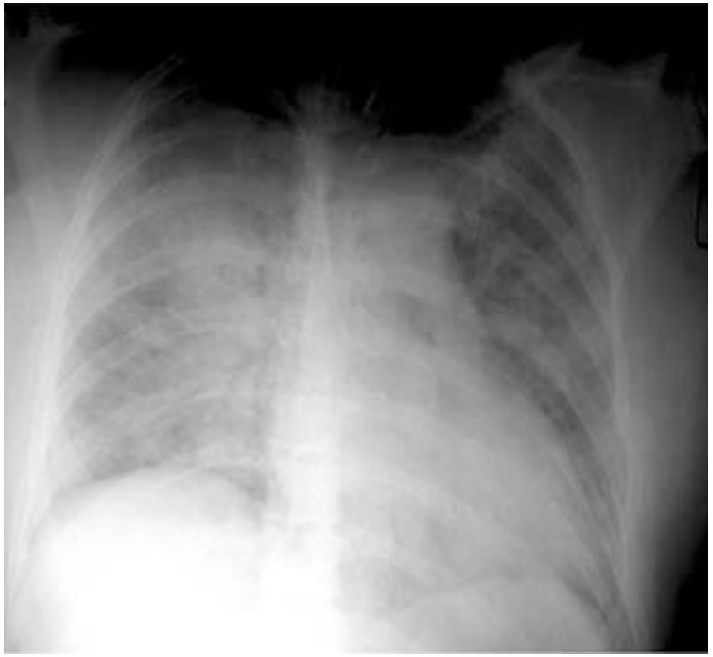
PRACTICAL PEARL

A Change of Position for Neurogenic Pulmonary Edema

Scott A. Marshall · Paul Nyquist

- A previously healthy 41-year-old Caucasian female with a 1-day history of severe occipital headache, intermittent dysarthria, double vision, and progressive weakness in all four limbs; 1 mm pupils not responsive to light, ocular skew deviation, respiratory failure.
- Complete occlusion of the basilar artery.
- Intraarterial thrombolysis with 100,000 units of urokinase resulted in full recanalization.

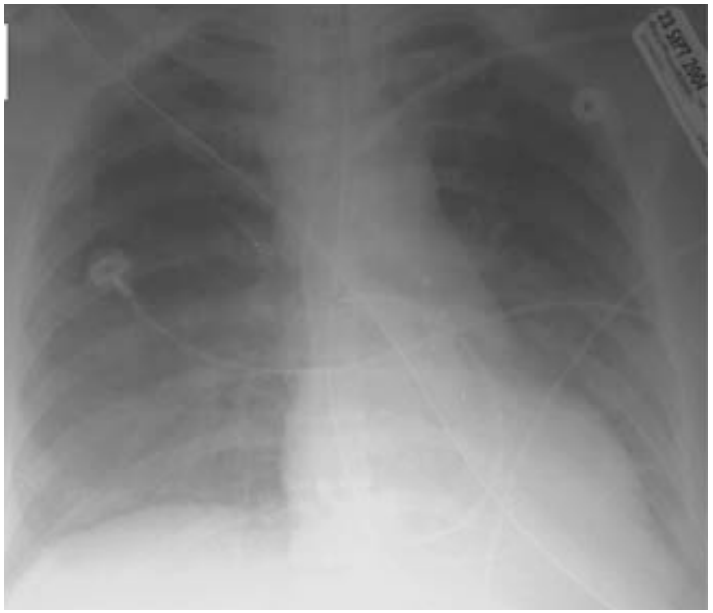
She rapidly deteriorated, developing fulminant pulmonary edema, with over 2 l of pulmonary edema fluid suctioned from her lungs.



100% oxygen, PEEP 20 cmH2O, I/E 2:1, pH 7.21, pCO2 56mmHg, PaO2 40mmHg, SaO2 58%, PaO2/FiO2 <200, CI 1.8.

Due to her critical clinical status, echocardiography could not be performed acutely. A Swan-Ganz catheter was placed, which showed a cardiac index of 1.8.

Despite aggressive diuretic therapy with separate doses of 40, 80, and 120 mg IV furosemide given 2h apart, multiple ventilation strategies, intravenous nitrates, and norepinephrine, the patient continued to deteriorate.



It was decided to ventilate her in the prone position. Within 30 min of initiating prone ventilation, her oxygenation status dramatically improved…..

Within the next 24h the management she was placed into a supine position with a reduction in PEEP to 10 and a FiO2 of 40%. Diuretics were reduced to a single dose of 40 mg.

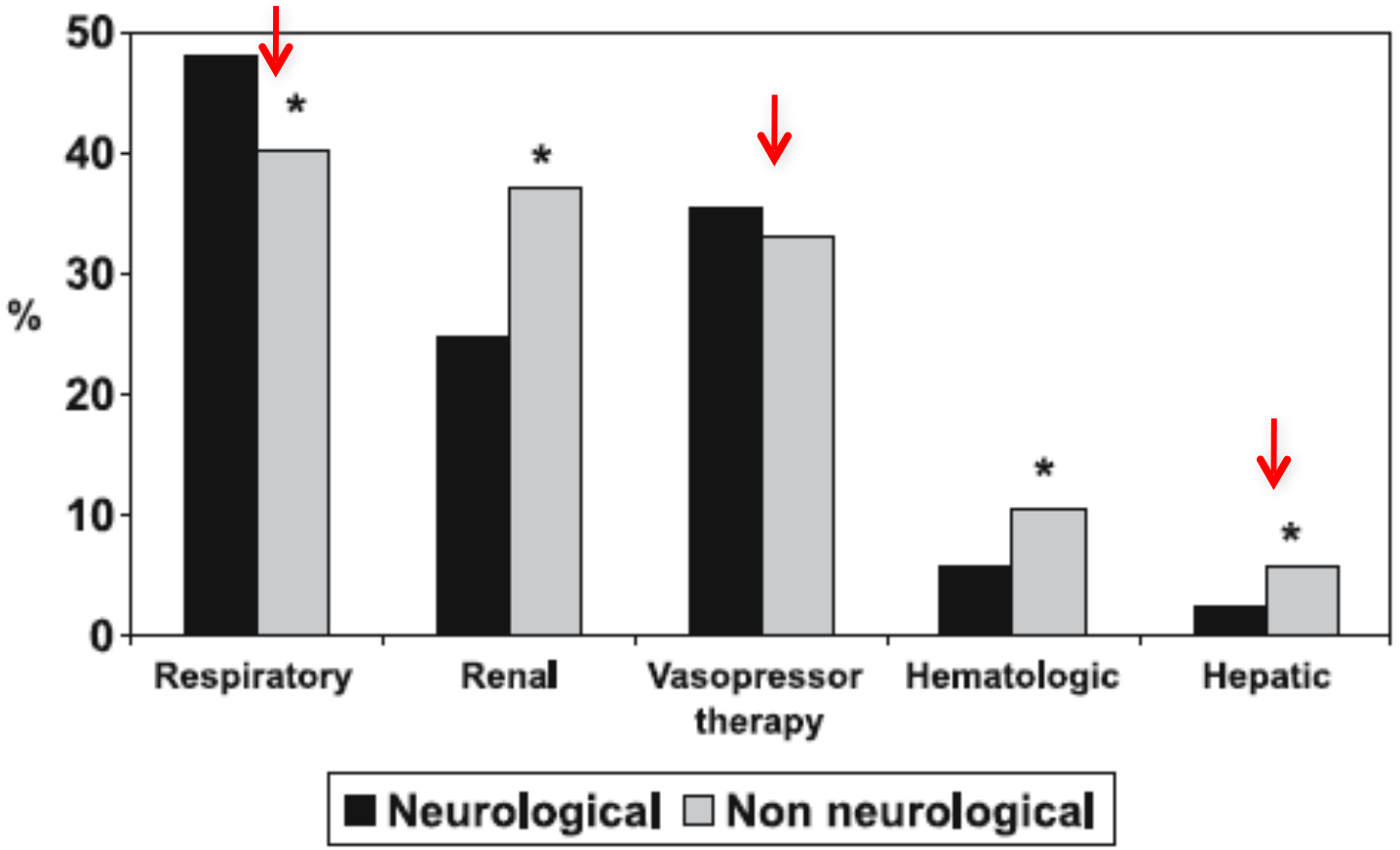
At 72 h chest X-ray was normalized and the patient was successfully extubated.

Intensive Care Med (2008) 34:720–727
DOI 10.1007/s00134-007-0974-7

ORIGINAL

Luciana Mascia
Yasser Sakr
Daniela Pasero
Didier Payen
Konrad Reinhart
Jean-Louis Vincent
Sepsis Occurrence in Acutely Ill Patients (SOAP) Investigators

Extracranial complications in patients with acute brain injury: a post-hoc analysis of the SOAP study



- **CARDIOVASCULAR DYSFUNCTION**
- **RESPIRATORY SYSTEM DYSFUNCTION**
- **COAGULATION DISORDERS**
- **IMMUNODEPRESSION WITH INCREASED INFECTION SUSCEPTIBILITY**
- **ABNORMALITIES OF PLASMA VOLUME AND COMPOSITION**

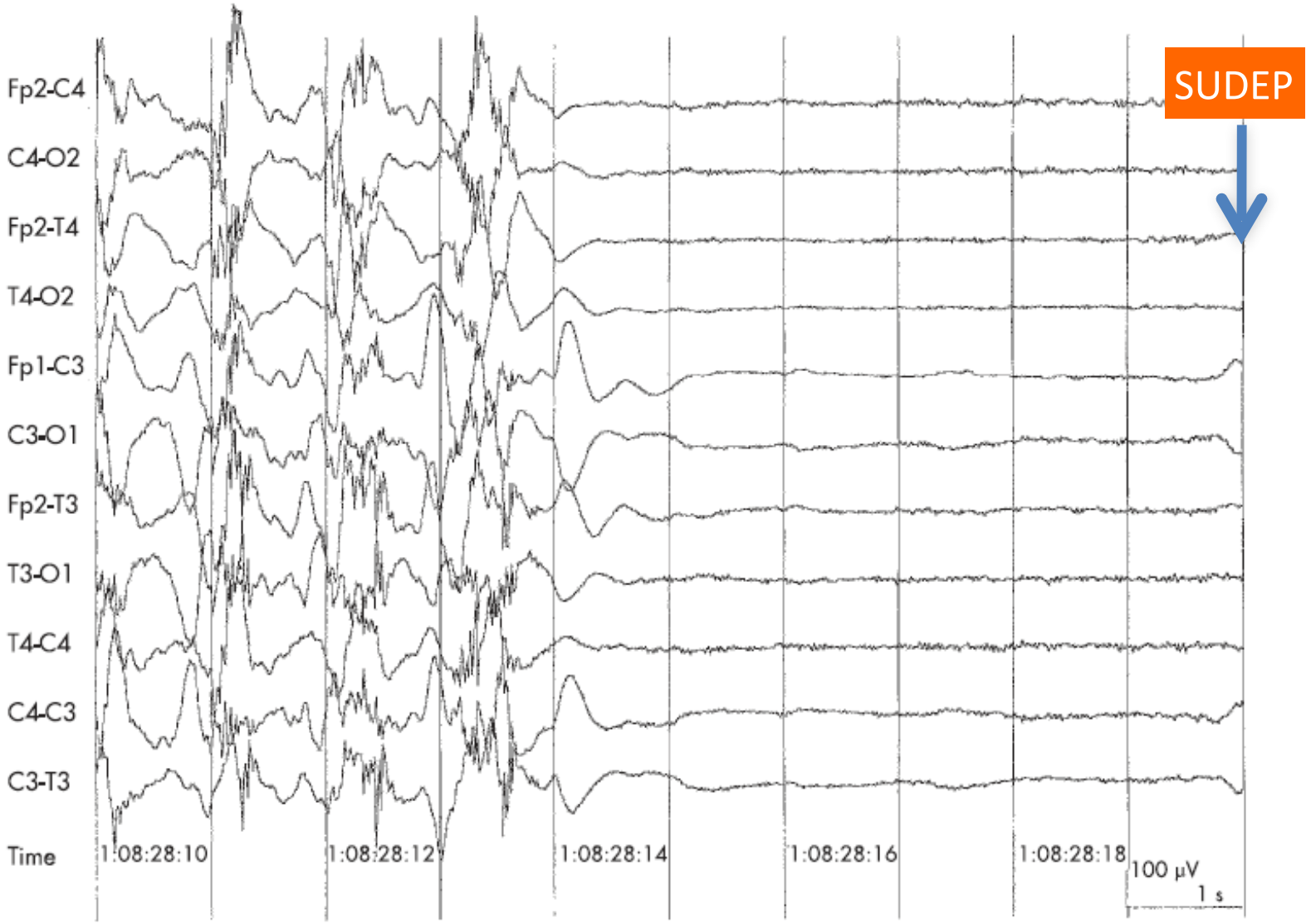
Case report

….. The patient fell asleep around midnight ….. Paroxysmal activity became more frequent and prolonged around 08:00, eventually becoming continuous with spike wave discharges developing to a seizure at 08:27:18.

Seizure activity then became polyspike (up to six spikes) and continued for 52s. The seizure activity abruptly terminated at 08:28:14 and the EEG became a ‘‘flatline’’.

McLean BN, Wimalaratna S. J Neurol Neurosurg Psychiatry 2007; 78:1395–1397

SUDEP **SU**dden **D**eath associated with **EP**ilepsy



McLean BN, Wimalaratna S. *J Neurol Neurosurg Psychiatry* **2007**; 78:1395–1397

Case report

The patient, a 29-year-old man affected by polycystic kidneys, received a kidney transplant in 2005. Since then, his glomerular filtration rate was stable around 30 ml/min.

He was treated with tacrolimus (Prograf), steroid and azathioprine and was taking atenolol, amlodipine, telmisartan, esomeprazole, folic acid, ω3-supplements and darbepoietin.

In August 2010, he anticipated his scheduled control because of somnolence, headache and fever. Laboratory investigations revealed hyponatraemia (117 mEq/l) and leukocytosis (14,000 cells/mm^3) . Since he was a known long-term user of recreational drugs, we looked for urinary drug metabolites. The test was negative for metabolites of ecstasy, but strongly positive for cannabis.

Haemoculture was positive for *Serratia Marcescens* and the patient was successfully treated with antibiotics and NaCl infusions.

Analysis of patient's records revealed that he had been suffering from three other episodes of severe hyponatraemia (113, 122 and 123 mEq/l) between 2006 and 2010. All three episodes were associated with sepsis (two bacterial, one viral) and required hospitalisation.

Catalano C, et al. B*MJ Case Reports* **2011**; doi:10.1136/bcr.01.2011.3755

Cannabinoid receptors are present in the hypothalamic area and it is well known that exogenous cannabinoids have an inhibitory effect on neuroendocrine function, leading to increase in diuresis and suppression of milk-ejection reflex through inhibition of ADH and oxytocin release, respectively.

Exception to this inhibitory activity is the stimulatory effect that cannabinoids have on hypothalamic-pituitary axis during stress conditions like sepsis.

FINAL DIAGNOSIS

Sepsis, cannabinoid-induced SIADH

LEARNING POINT

Cannabinoids may enhance the sepsis-related ADH release which may in turn lead to the development of life-threatening hyponatraemia.

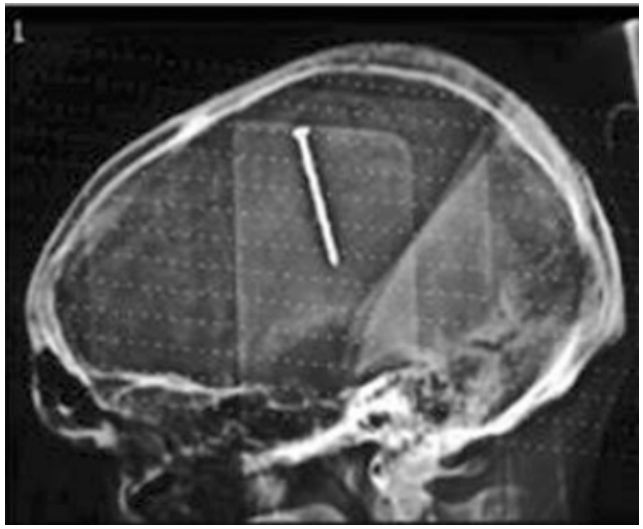
Catalano C, et al. B*MJ Case Reports* **2011**; doi:10.1136/bcr.01.2011.3755

Case report

The patient suffered a penetrating a penetrating skull injury after falling from a ladder. On admission he was hyponatraemic at 125 mmol/L, hypotensive (80/30 mmHg; 70/40 mmHg) & hypoglycaemic.

The patient was euvolaemic with inappropriately concentrated urine and natriuresis, and required vasopressor support and continuous intravenous dextrose infusion.

The combination of hyponatraemia, hypoglycaemia and hypotension suggested the diagnosis of ……



Brain CT showing a nail embedded through the skull into the brain. Note the translucency surrounding the nail, which represents abscess formation.

Agha A, Sherlock M, C.J. Thompson CJ. QJM 2005; 98: 463-464