APPENDIX

INDEX

SOUTH CAROLINA DEPARTMENT OF CORRECTIONS
Post Office Box 21787, Columbia, South Carolina 29221

Pursuant to Code Section 24-3-530, Code of Laws of South Carolina, 1976, the Director of the South Carolina Department of Corrections has designated Willie Davis as his duly authorized agent for the purpose of making service of the Notice of Election the below named individual.

STATE OF SOUTH CAROLINA)
) AFFIDAVIT OF PERSONAL SERVICE
COUNTY OF RICHLAND)

On the 21st day of February 2025, I served the Notice of Election on Brad Keith Sigmon SK 006008, by delivering personally and leaving a copy of the same at Broad River Correctional Institution, 4460 Broad River Road, Columbia, South Carolina. Deponent is not a party to this action.

S/ Willie Davis
Willie Davis

SWORN TO AND SUBSCRIBED before me this February 21, 2025.

Jacqueline Murrell (L.S.)
Notary Public for South Carolina

My Commission Expires: 4.27.26

JACQUELINE MURRELL
Notary Public
South Carolina
My Comm. Expires April 27, 2026

ACCEPTANCE OF SERVICE

Service of a copy of the within Notice of Election is accepted at Broad River Correctional Institution, 4460 Broad River Road, Columbia, South Carolina, this 21st day of February 2025.

S/ Brad Sigmon
Brad Keith Sigmon SK 006008

STATE OF SOUTH CAROLINA)
) NOTICE OF ELECTION
COUNTY OF RICHLAND)

In accordance with Section 24-3-530(A), S.C. Code of Laws, 1976, as amended, "[a] person convicted of a capital crime and having imposed upon him the sentence of death shall suffer the penalty by electrocution or, at the election of the convicted person, by firing squad or lethal injection, if it is available at the time of election, under the direction of the Director of the Department of Corrections. The election for death by electrocution, firing squad, or lethal injection must be made in writing fourteen days before each execution date, or it is waived. If the convicted person receives a stay of execution or the execution date has passed for any reason, then the election expires and must be renewed in writing fourteen days before a new execution date. If the convicted person waives the right of election, then the penalty must be administered by electrocution."

Methods of Execution

I, Brad Keith Sigmon, pursuant to Section 24-3-530, South Carolina Code of Laws, 1976 as amended, hereby elect electrocution as the method for execution. By my signature below I select electrocution as the method for execution.

S/_____ _____
Brad Keith Sigmon Date

I, Brad Keith Sigmon, pursuant to Section 24-3-530, South Carolina Code of Laws, 1976 as amended, hereby elect firing squad as the method for execution. By my signature below I select firing squad as the method for execution.

S/ Brad Sigmon 2/21/25
Brad Keith Sigmon Date

I, Brad Keith Sigmon, pursuant to Section 24-3-530, South Carolina Code of Laws, 1976 as amended, hereby elect lethal injection as the method for execution. By my signature below I select lethal injection as the method for execution.

S/_____ _____
Brad Keith Sigmon Date

WITNESSES:

_____ 2-21-25
Witness Signature Date

_____ 2-21-25
Witness Signature Date

Jacqueline Murrell 2/21/25 a2

JACQUELINE MURRELL
Notary Public
South Carolina
My Comm. Expires April 27, 2026

IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF SOUTH CAROLINA
No. 3:25-cv-00199-JDA

MARION BOWMAN, JR.,	)	
	)	
Plaintiff,	)	
	)	
v.	)	**CAPITAL CASE**
	)	
BRYAN P. STIRLING, in his official capacity as the Director of the South Carolina Department of Corrections, and;	)	**EXECUTION SCHEDULED JANUARY 31, 2025**
	)	
HENRY DARGAN McMASTER, in his official capacity as Governor of the State of South of South Carolina,	)	
	)	
Defendants.	)	

DECLARATION OF JOSEPH F. ANTOGNINI, M.D., M.B.A.

JOSEPH F. ANTOGNINI, does hereby declare and say:

1. My name is Joseph F. Antognini. I am a medical doctor, board-certified in anesthesiology. I received a B.A. degree from the University of California, Berkeley in Economics in 1980. I received my M.D. degree from the University of Southern California in 1984. I also received an M.B.A. from California State University, Sacramento in 2010. I was previously the Director of Peri-operative Services at the University of California, Davis Health System and a Professor of Anesthesiology and Pain Medicine and Professor of Neurobiology, Physiology and Behavior at the University of California, Davis. I am licensed to practice medicine in the State of

California. I have over 30 years of experience practicing anesthesiology since 1984 when I began my residency at the University of California, Davis Health System. I am the author or co-author of over 200 publications (papers, abstracts, book chapters, etc.). My area of research has focused on anesthetic mechanisms, specifically related to where anesthetics produce unconsciousness, amnesia and immobility. I currently perform clinical research, and I am Chief Scientific Officer for a small pharmaceutical company that develops new anesthetics. A true and correct copy of my curriculum vitae is attached hereto as Exhibit A.

2. I have reviewed, and am familiar with, the allegations made in the Motion for Injunctive Relief, *Marion Bowman, Jr. v. Bryan P. Stirling and Henry Dargan McMaster*, No. 3:25-cv-00199-JDA, dated January 13, 2025, and additional information in the documents described below.

Scope of Engagement

3. I have been asked to render expert opinions in the fields of general medicine and anesthesiology, especially regarding the use, actions and efficacy of pentobarbital, in relation to South Carolina's lethal injection protocol, and the effectiveness of the procedures therein. This declaration contains a complete statement of my opinions, and the basis and reasons therefore, including the facts or data I have considered in forming them. I may supplement this declaration as appropriate. The opinions that I do

provide are within my field of anesthesiology and such fields as are necessarily related to anesthesiology, including general medicine, pharmacology and physiology, and fall within the scope of my expertise. All opinions expressed herein are stated to a reasonable degree of medical and scientific certainty unless otherwise noted.

Materials Reviewed

4. I have conferred with attorneys for Defendants. Among the documents I have reviewed in connection with this case are: South Carolina's execution protocol; Plaintiff's Motion for Injunctive Relief (dated January 13, 2025); publications and materials listed in the "References Cited" section; the declaration of Dr. David Waisel (dated January 10, 2025); the affidavit of Bryan P. Stirling, dated January 7, 2025; the affidavit of Dr. Michaela Almgren, dated August 31, 2024; and the autopsy report on Richard Moore.

5. Should additional documents or information be provided to me for review and analysis, I may take those additional materials into account, and modify and/or supplement my opinions accordingly. If I am present at hearings and/or trial in this case, I may take into account any testimony or other evidence to the extent related to my opinions and modify and/or supplement my opinions accordingly. In performing my analysis, I have relied on my professional training, education and experience. The opinions

presented in this declaration are my opinions and mine alone. I have reviewed and considered documents and information and identified those materials above. These documents and other information that I reviewed and considered are of a type reasonably relied upon by experts in the field of anesthesiology, general medicine, physiology and pharmacology in forming opinions or inferences on questions in this area. My fee schedule for this engagement is: $575/hour for phone consultation, research, declaration preparation; $675/hour for deposition; $7000/day for courtroom appearance; $287/hour for travel time plus travel expenses at cost.

6. I have testified and submitted expert reports in the following cases in the past four years: 1) I have submitted reports and given testimony *In the Matter of the Federal Bureau of Prisons' Execution Protocol Cases* (No. 19-mc-00145-TSC); 2) I have submitted reports and have testified in *Glossip et al. v. Chandler et al.*, Case No. CIV-14-665-F, in the United States District Court for the Western District of Oklahoma; 3) I have submitted reports and have testified in *Bigler Stouffer. v. Scott Crow*, Case No. 21-cv-1000-F, in the United States District Court for the Western District of Oklahoma; 4) I have submitted reports and have been deposed in *Terry Lynn King v. Tony Parker*, Case No. 3:18-cv-01234, in the United States District Court for the Middle District of Tennessee; 5) I have submitted reports and testified in *Michael Nance v. Oliver & Caldwell,* Case No. 1:20-CV-107-JPB, in the United States

District Court for the Northern District of Georgia, Atlanta Division; 6) I have submitted reports and testified in *Kenneth Eugene Smith v John Q. Hamm*, 2:22-cv-00497-RAH, in the United States District Court for the Middle District of Alabama; 7) I have submitted reports and been deposed in *Martin v Oliver & Caldwell,* 1:18-cv-4615-MLB in the US District Court, Northern District of Georgia, Atlanta Division; 8) I have submitted a report and been deposed in *Miller v. Marshall et al.* 2:24-cv-197 in the United States District Court for the Middle District of Alabama; 9) I have submitted a report and testified in *Grayson v. Hamm et al.,* 2:24-cv-00376-RAH-KFP in the United States District Court for the Middle District of Alabama.

Discussion

7. The intravenous administration of five (5) grams of pentobarbital causes rapid unconsciousness followed by respiratory arrest, cardiovascular collapse and death. After intravenous injection of 5 grams pentobarbital, concentrations of pentobarbital in the body will far exceed the lethal concentrations—see Table 1, package insert for pentobarbital in References Cited and extrapolating from data of Ehrnebo (1974). Once respiratory depression and respiratory arrest occurs within 1-2 minutes, the unconscious inmate then begins to use up the oxygen stores in his body. Before all the oxygen is used, however, the heart will be affected, will begin to slow and will then have periodic irregular beats. It likely will take several minutes before

the heart stops all together. At that point, death is declared. This process, as described, is irrefutable. It is based on the known actions of pentobarbital and sound pharmacological and physiological principles, and the known effects of these doses of pentobarbital in lethal injection executions.

8. Pentobarbital administered to humans results in unconsciousness in 20-30 sec, on average,[1] and this effect is dose dependent, with greater doses (>5 mg/kg) having onset times in the 20 sec range (Dundee, 1957). In a 100-kg person (about 220 pounds), this dose would be 500 mg, which is only 10% of the dose used in the South Carolina lethal injection protocol. At this point, pulmonary edema, if it occurs at all during the execution (as opposed to post-mortem lung changes), would not set in because it would only result from a much larger dosage (i.e. an overdose).[2] As the additional 4500 mg of pentobarbital is administered, the inmate would have progressive brain depression, with electrical brain silence occurring, followed by cardiovascular collapse, as noted above. Before becoming unconscious, the individual would not feel the sensations of pain, suffocation or air hunger. And the inmate

1 It is important to note that the time to unconsciousness depends on the speed with which the drug is administered and when the "clock starts". For example, my estimate for 20-30 sec is based on when a clinical dose of pentobarbital has actually entered the person, not when the drug begins to enter the IV tubing.
2 Clinical doses of barbiturates, such as thiopental and pentobarbital, cause unconsciousness, but not pulmonary edema. If clinical doses of these drugs caused pulmonary edema, the drugs would have been abandoned soon after their introduction in the 1930s.

would not feel the sensation of pain, suffocation or air hunger after becoming unconscious.

9. These actions of pentobarbital are consistent with data published by Aleman et al., (2015), a study discussed in the recent US Supreme Court case *Bucklew v. Precythe,* No. 17-8151 (decided April 1, 2019). In the Aleman study, horses were administered large, lethal doses of pentobarbital, with a mean time of infusion of 47 seconds, and the horses developed electroencephalographic brain silence (i.e., flat line) at a mean of 53 seconds after the initiation of the infusion, that is, EEG silence occurred on average, 6 seconds after the infusion finished. Because loss of consciousness occurs before EEG silence, these data fit with a time frame of 20-30 seconds for loss of consciousness after the initiation of the pentobarbital infusion.

10. In a similar study (Buhl et al., 2013), the time to collapse (when the horses went from standing to falling to the ground, and which is considered to be the onset of unconsciousness) was about 27 seconds (the average of the means of the four groups studied; see their table 2) after the initiation of the infusions. They also noted that respiratory arrest occurred simultaneously with falling to the ground in most horses (2nd paragraph in discussion).

11. These studies cited above collectively lead to the conclusion that intravenous pentobarbital administered at 5 grams would cause rapid onset

of unconsciousness, followed by coma, respiratory arrest, circulatory collapse and death.

12. Thiopental and pentobarbital are equipotent (Barron & Dundee, 1961). For example, 100 mg of thiopental has the same effect as 100 mg of pentobarbital, 500 mg of thiopental has the same effect as 500 mg of pentobarbital, and so forth. Thus, studies reporting on the effects of thiopental can be used to infer the effects of pentobarbital.

13. Both thiopental and pentobarbital cause brain suppression (including suppression of electrical activity in the brain as measured with the electroencephalogram, EEG). The dose at which EEG silence begins to occur is about 17 mg/kg, based on studies utilizing thiopental infused over 10-15 minutes (Buhrer et al., 1992; Hung et al., 1992). But, in the setting of an execution, pentobarbital would be infused more quickly and at a greater dose than that described in Buhrer et al. Five (5) grams (equivalent to 5000 mg) of pentobarbital administered to a 100-kg person (approximately 220-lbs person) is 50 mg/kg, and about 71 mg/kg in a 70 kg person, doses that far exceed 17 mg/kg. Thus, EEG silence would be expected to occur within 60 seconds after initiation of pentobarbital infusion, consistent with the data reported by Aleman et al.

14. The State of Georgia has executed at least 24 inmates in the past decade using pentobarbital, and these times to death were submitted as evidence in *Martin v. Ward & Ford*, No. 1:18-cv-04617-MLB, in which both Dr Waisel and I were expert witnesses.[3] The times between initiation of pentobarbital infusion and time of death reported for 24 executions ranged from 8 to 27 minutes, with an average of about 14 minutes. These times comport with what I would expect with 5 grams of pentobarbital administered according to the South Carolina protocol. The longer times between pentobarbital administration and time of death are most likely related to the process by which death is declared related to cessation of electrical heart activity. The electrocardiogram (ECG) measures electrical heart activity and in the process of dying the heart may have occasional electrical activity for many minutes after complete cardiovascular collapse and respiratory arrest. The amount of time it takes for the heart to stop can be variable, so the ranges reported for these 24 executions are not surprising and do not indicate any problems with the way in which the Georgia protocol is implemented.[4,5]

---

3 Dr. Waisel considered and discussed these times in his May 10, 2024 report submitted to the Court, as did I in my report dated April 10, 2024.

4 Also, during an execution there is an additional variable amount of time between when the heart stops beating and when time of death is determined. The physician must wait a variable amount of time after the last heartbeat to ensure he or she has actually observed the "last" heartbeat. This time might be 1-2 minutes or longer, depending on the physician. Then, the physician enters the chamber, examines the inmate for signs of life, and declares the time of death.

5 The Georgia method of lethal injection execution and the South Carolina method are similar

15. In the Aleman study, horses administered pentobarbital developed asystole (cessation of the heartbeat) in the range of 5.5 to 16.3 minutes (a ratio 16.3/5.5 ≈ 3), and in the Buhl study the range was 3.3 to 20 minutes (based on the data in their Figure 2; ratio 20/3.3 ≈ 6). The ratio of the times to death (longest/shortest) in the 24 executions is 27/8 ≈ 3.4, like those found in the Aleman and Buhl studies. Taken together, these execution times and the animal studies indicate that variability is the norm, not the exception.

16. Intravenous administration of 5 grams of pentobarbital would cause profound brain depression and unconsciousness well before any lung congestion and pulmonary edema forms.

17. Whether pentobarbital causes pulmonary edema directly, or indirectly as a natural consequence of the dying process, is immaterial because the inmate would be profoundly unconscious, to the point of electrical brain silence. Furthermore, it is unclear how much of the pulmonary edema and lung congestion found at autopsy is due to post-mortem changes.

18. More recent studies in humans using post-mortem computed tomography (PMCT) show that fluid accumulates in lung over time in the post-mortem period (Shiotani et al., 2011). Shiotani et al. write in their concluding paragraph: "PMCT findings of the lung are not fixed and change

---

regarding procedures and administration of pentobarbital.

with the passage of time after death in accordance with progression of postmortem changes (pulmonary congestion and edema) in the corpse."

19. Likewise, fluid accumulation in the airways increases during the post-mortem period (Ishida et al. 2014); these authors showed that fluid accumulated in the airways (main bronchi) as the interval between death and PMCT increased. This fluid accumulation is akin to the fluid that has been found at autopsy in inmates executed by lethal injection.

20. Published data on how post-mortem pulmonary edema and lung congestion occur and progress is based in large part on animal studies. Durlacher et al. (1950) examined post-mortem changes in rabbit lungs after various causes of death, including pentobarbital overdose. They found that lung weight increased as the time between pentobarbital-induced death and autopsy increased, as shown in their table 2:

TABLE 2

EFFECT OF INTERVAL AFTER SACRIFICE BY NEMBUTAL (100 MG./KG.) ON LUNG WEIGHT

Interval after sacrifice	*Treatment*	*Number of animals*	*Lung weight per kilo ± S.E. mean*
			Grams
Immediate		5	3.83 ± .27
1 hours	Cannula in trachea	5	5.42 ± .58
2 hours	Cannula in trachea	5	7.09 ± 1.39
3 hours	Cannula in trachea	19	9.46 ± .62
4 hours	Cannula in trachea	5	10.88 ± 1.53
6 hours	Cannula in trachea	5	10.95 ± .74

Note that lung weight increased when comparing lung weight at immediate autopsy to lung weight at 1, 2, 3, 4 and 6 hours after death, indicating that

lungs can develop edema *after* death. These researchers (and others[6]) also found that, for a variety of causes of death, lung weight increased as the interval between death and autopsy increased (see table 1 in Durlacher et al., 1950). These data indicate that post-mortem edema formation is a generalized phenomenon and is not specific to drug overdose Thus, the animal data indicate that all of the pulmonary edema and lung congestion found at autopsy in inmates executed by lethal injection could be generated post-mortem.

21. Frothy fluid and foam are sometimes found in humans and animals after death, and there is evidence that this froth can occur immediately prior to death (in the period from apnea to cardiac death; see Swann 1964) and after death.[7] Thus, the finding of froth in inmates who were executed by lethal injection does not indicate that this froth was generated ante-mortem.

22. Post-mortem froth and foam could be generated by the release of gasses from the lung tissues and interacting with the lung surfactant, a substance that, during life, keeps alveoli (small lung units, or air sacs) open. Related to this issue, Pattle (1955) wrote that "....oedema foam is thus not produced by

---

[6] See Acta Scandinavica Medica 1964 in References Cited

[7] Animals (rabbits) made uremic (kidney failure) and who subsequently developed pulmonary edema were found to not only have increasing lung weights as the period between death and post-mortem exam increased, but the presence of froth was found in animals that had later post-mortem exams, while none was found upon immediate post-mortem examination. See Acta Scandinavica Medica 1964 in References Cited

agitation of the oedema fluid with air during respiration; it can only have been formed by air originally in the fine air spaces of the lung being broken up into bubbles and afterwards expelled into the bronchi and trachea." Thus, the post-mortem finding of froth in inmates who were executed by lethal injection does not conclusively indicate that this froth was generated ante-mortem, or by conscious attempts to breathe.

23. The presence of pulmonary edema at autopsy is a common and non-specific finding and is associated with variety causes of death (Saukko & Knight, 2004; Sogawa et al., 2014).[8]

24. The witnesses to the executions of Freddie Owens and Richard Moore describe what would be expected from lethal injection of pentobarbital (see links to the press conferences cited in the References section). In the Owens execution, Owens appeared to be conscious for about 1-1.5 minutes after initiation of the pentobarbital, followed by deep breathing akin to snoring, then shallow breathing. No movement occurred after about 6 minutes following the initiation of the pentobarbital. In the Richard Moore execution, several deep breaths started about 1 minute following the initiation of the pentobarbital, followed by shallow breathing, with no movement observed

8 Saukko & Knight: Knight's Forensic Pathology, 3rd Edition, page 356: "Pulmonary oedema is such a common and non-specific phenomenon in a whole range of fatal conditions that it has little diagnostic significance."

after about 3 minutes. These observations comport with what I would expect to occur following a lethal dose of pentobarbital.

25. In his declaration Dr. Waisel lists several opinions that are not well-founded and are based on faulty reasoning and erroneous interpretation of the data and events.[9] In section V.7 of his declaration, he opines that a properly administered dose of pentobarbital should eliminate breathing in less than one minute, but he ignores important factors. For example, the speed with which the drug is administered impacts responses to the drug, with slower administration causing longer times for drug effects to occur. Also, the presence of agonal breaths, which are the last "gasps" that a person or animal takes immediately prior to death, prolongs the time to apnea (lack of breathing). In my opinion, breathing efforts can occur for a few minutes after the pentobarbital has been administered, however, these breathing efforts will become shallow in the few minutes after drug administration.

26. In section V.8 Dr. Waisel states that it is "physiologically and pharmacologically impossible for Mr. Moore to remain alive for ten minutes after a dose of five grams of fully-potent pentobarbital, unless that dose was not delivered completely". Dr. Waisel completely ignores the expected effect of

9 As an aside, Dr. Waisel discusses in section V.2 of his declaration the hypothetical administration of a barbiturate at a dose of 350 mg/kg, which is clearly a typographical error. But because the reader is left wondering what dose he meant to write, Dr. Waisel's reasoning is further muddled.

the pentobarbital and the practical aspects of the execution process. As outlined above, the heart can have occasional beats for many minutes after pentobarbital is administered (as long as 20 minutes between drug administration and loss of cardiac electrical activity—see Buhl et al., 2013). While the inmate is deeply unconscious, the person who declares death will not do so until a waiting period after the electrocardiogram is "flatline", e.g., there is no electrical activity of the heart.

27. In section V.9, Dr. Waisel opines that Mr. Moore "consciously experienced feelings of drowning and suffocation during the 23 minutes that it took to bring about his death". Dr. Waisel completely ignores the effects of the lethal doses of pentobarbital used. Dr. Waisel expects the reader to believe that the massive dose of pentobarbital will not cause unconsciousness but will result in "sudden" death at minute 23. If Mr. Moore was conscious and drowning in his own fluids then why didn't he move prior to minute 23? Why didn't he breathe fast, as would be expected if he was awake and had pulmonary edema? The answer to both questions is that Mr. Moore was profoundly unconscious from the pentobarbital.

28. Dr. Waisel also questions the need for an additional 5-grams of pentobarbital in the Moore execution. As noted above, it would not be unexpected that some electrical activity of the heart persisted after 10

minutes, so a second 5-gram injection was probably used for that reason. The witnesses to the Moore execution reported that Mr. Moore did not move after about 3 minutes, so Mr. Moore likely was profoundly unconscious at that point. Also, the pentobarbital concentration found in Mr. Moore at autopsy (85 mcg/ml) greatly exceeded the lethal level.[10] In the 24 Georgia executions using 5-grams pentobarbital, the mean pentobarbital concentration at autopsy was 38 mcg/ml, so the 85 mcg/ml level found in Mr. Moore is consistent with the administration of a 10-gram dose of pentobarbital, which refutes Dr. Waisel's claim that an insufficient amount of pentobarbital was administered.[11]

29. In section V.11 Dr. Waisel states that intravenous access in obese persons might be difficult, but this is true for any patient, and thousands of obese patients have surgery every day after the successful placement of an intravenous catheter.

Conclusion

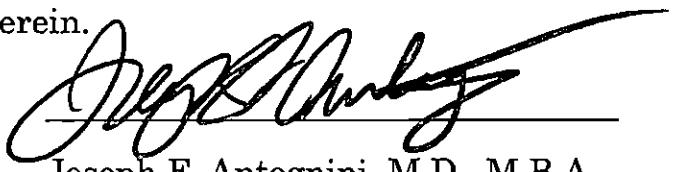
30. It is my opinion, to a reasonable degree of medical and scientific certainty, that 1) the inmate would become unconscious within 20-30 sec after pentobarbital first enters the inmate, which would be followed by respiratory arrest, cardiovascular collapse and death; 2) injection of massive

10 The package insert states that 10-15 mcg/ml causes coma, while 15-40 mcg/ml is lethal.
11 In his report of May 10, 2024, Dr. Waisel states he reviewed the toxicology data for these executions.

doses (5 grams) of pentobarbital would not inflict mild, moderate or severe pain; 3) pulmonary edema, if it occurs ante-mortem, would not be perceived by the inmate because of the profound brain suppression caused by pentobarbital.

31. Should additional information become available I reserve the opportunity to amend my statements herein.

Date: January 21, 2025



Joseph F. Antognini, M.D., M.B.A.

References Cited

Acta Medica Scandinavica. Control Material. Acta Medica Scandinavica 1964; 176 (s418):29-40.

Aleman M, Williams DC, Guedes A, Madigan JE. Cerebral and brainstem electrophysiologic activity during euthanasia with pentobarbital sodium in horses. J Vet Int Med 2015; 29:663-72

Barron DW, Dundee JW. The recently introduced rapidly-acting barbiturates; a review and critical appraisal in relation to thiopentone. Brit J Anaesthesia 1961; 33:81-91

Buhl R, Andersen LOF, Karlshoj M, Kanters JK. Evaluation of clinical and electrocardiographic changes during the euthanasia of horses. The Veterinary Journal 2013; 196:483-91

Buhrer M, Maitre PO, Hung OR, et al. Thiopental Pharmacodynamics. I. Defining the pseudo-steady-state serum concentration-EEG effect relationship. Anesthesiology 1992; 77:226-236

Dundee JW. Abnormal responses to barbiturates. Brit J Anaesthesia 1957; 29:440-46

Durlacher et al., Post-mortem pulmonary edema. Yale Journal of Medicine 1950; 565-72

Ehrnebo M. Pharmacokinetics and distribution properties of pentobarbital in humans following oral and intravenous administration. J Pharmaceutical Sciences 1975; 63:1114-18

Hung OR, Varvel JR, Shafer SL, Stanski DR. Thiopental pharmacodynamics. II. Quantitation of clinical and electroencephalographic depth of anesthesia. Anesthesiology 1992; 77:237-244

Ishida M, Gonoi W, Hagigawa K, et al. Fluid in the airway of nontraumatic death on postmortem computed tomography. Am J Forensic Med Path 2014; 35:113-17

Lafferty KA. Barbiturate Toxicity.

http://emedicine.medscape.com/article/813155-overview#a5

(accessed 1-21-2025)

Pattle RE. Properties, function and origin of the alveolar lining layer. Nature 1955; 175: 1125-26

Saukko P, Knight B. Knight's Forensic Pathology, 3rd. Ed. Hodder-Arnold, 2004

Shiotani S, Kobayashi T, Hayakawa H, Kikuchi K, Kohno M. Postmortem pulmonary edema: A comparison between immediate and delayed postmortem computed tomography. Legal Medicine 2011; 13:151-55

Sogawa et al., Postmortem virtual volumetry of the heart and lung in situ using CT data for investigating terminal cardiopulmonary pathophysiology in forensic autopsy. Legal Medicine 2014;16:187-92

Swann HE. The development of pulmonary edema during the agonal period of sudden asphyxia deaths. J Forensic Sciences 1964; 9:360-73

Press conference following execution of Richard Moore on November 1, 2024 (accessed January 18, 2025):

Witnesses speak after execution of South Carolina inmate Richard Moore

Press conference following execution of Freddie Owens on September 20, 2024 (accessed January 18, 2025):

FULL PRESS CONFERENCE Freddie Owens Execution: 9.20.2024

Pentobarbital package insert (accessed 1-21-2025):

https://dailymed.nlm.nih.gov/dailymed/fda/fdaDrugXsl.cfm?setid=e9f4b344-

b092-4eec-b49d-d8cfe8ebc05d&type=display

EXHIBIT A

CURRICULUM VITAE
Joseph F. Antognini, M.D., M.B.A.

CONTACT:

jfantognini@icloud.com

jfantognini@ucdavis.edu

EDUCATION:

1980	University of California, Berkeley (B.A., Economics)
1984	University of Southern California (M.D., Medicine)
2010	California State University, Sacramento (M.B.A., Business)

INTERNSHIP/RESIDENCY:

1984-1987	Anesthesiology, UC Davis Medical Center
1986-1987	Chief Resident

PROFESSIONAL POSITIONS:

6/24-present	Chief Scientific Officer/Interim Chief Medical Officer Expanesthetics, Inc Davis, CA
1/22-present	Principal Investigator Next Level Clinical Trials, LLC West Covina, CA
1/22-present	Sub-Investigator SmartCures Clinical Research, LLC Anaheim, CA
7/22-present	Sub-Investigator Long Beach Clinical Trials, LLC Long Beach, CA

7/17-present	Director Emeritus University of California, Davis
2015-present	Clinical Advisory Board Expanesthetics, Davis, CA
9/21-7/23	Surgical Wound Specialist Advantage Surgical and Wound Care El Segundo, CA
1/20-12/22	Adjunct Faculty Los Medanos College Pittsburg, CA
1/20-5/20	Adjunct Faculty Holy Names University Oakland, CA
9/16-11/19	Physician Surveyor The Joint Commission Oakbrook Terrace, IL
2011-2020	Clinical Professor of Anesthesiology and Pain Medicine (Volunteer Clinical Faculty appointment) University of California, Davis—School of Medicine
11/10-6/16	Director of Peri-operative Services UC Davis Health System
7/00-7/11	Professor of Anesthesiology and Pain Medicine12 (with tenure) Department of Anesthesiology and Pain Medicine University of California, Davis—School of Medicine
12/02-7/11	Professor of Neurobiology, Physiology and Behavior (with tenure; WOS appointment) College of Biological Sciences

12 My research publications place me in the top 1.5% of scientists worldwide based on number of citations of my papers (October 2023 data-update for "Updated science-wide author databases of standardized citation indicators" - Elsevier BV (digitalcommonsdata.com) accessed 5-17-2024). Also, I am in the category of "outstanding scientist" based on the h-index (h-index = 42 as of 1-9-25, with >5600 citations according to Google Scholar). The h-index is a measure of how often a person's work is cited. See: Hirsch JE. An index to quantify an individual's scientific output. PNAS 2005; 103:16569-572

	University of California, Davis
11/98-7/10	Vice Chairman, Director of Research
11/98-3/02	Director of Malignant Hyperthermia Diagnostic Laboratory Department of Anesthesiology
7/96-7/00	Associate Professor (with tenure) Department of Anesthesiology University of California, Davis—School of Medicine
10/91-6/96	Assistant Professor Department of Anesthesiology University of California, Davis—School of Medicine
7/87-9/91	Staff Anesthesiologist (Private Practice) American River Hospital Department of Anesthesiology Carmichael, CA
7/87-9/91	Assistant Clinical Professor (volunteer) Department of Anesthesiology University of California, Davis—School of Medicine

LICENSURE & CERTIFICATIONS:
State of California #G55662 (expires 7-31-2025)
State of Georgia #100252 (expires 7-31-2025)
DEA certificate BA0948870 (expires 6-30-2027)
Diplomate, National Board of Medical Examiners (1985)
Diplomate, American Board of Anesthesiology (1989; Life-time, not time limited)
Certificate of Recertification, American Board of Anesthesiology (1999, 2009)
Certified Yellow Belt, 2017

PROFESSIONAL SOCIETIES AND RECOGNITION:
American Society of Anesthesiologists 1987--present
California Society of Anesthesiologists 1987—present
Fellow of the American Society of Anesthesiologists 2018—present

ADVOCACY
ASA Grassroots Network (ASA Team 535) 2018
ASAPAC Donor—2018
FAER Donor—1999-2022

RESEARCH INTERESTS:

Mechanisms of anesthesia; factors influencing anesthetic requirements; OR efficiency

AWARDS AND HONORS

Dean's Mentoring Award, UC Davis School of Medicine, 2006
Associated Students of UC Davis "Excellence in Education Award" College of Biological Sciences, 2007
Associated Students of UC Davis "Excellence in Education Award" Outstanding Educator, 2007
Foundation for Anesthesia Education and Research, Mentor Academy, 2008
Phi Kappa Phi Honor Society, 2010

GRANTS

1. UC Davis Faculty Research Grant 1991-92—The effect of intrathecal aspirin on anesthetic requirements in rabbits, $2500
2. UC Davis Faculty Research Grant 1993-94—Validation of a preferentially anesthetized goat brain model, $1500
3. Foundation for Anesthesia Education and Research 1994—Determination of gross anatomic sites of anesthetic action, $25,000 ($25,000 matching departmental funds)
4. UC Davis Faculty Research Grant 1994-95—The effects of general anesthesia on cerebral blood flow patterns as assessed by functional magnetic resonance imaging, $1500
5. UC Davis Faculty Research Grant 1996-97—The effect of differential isoflurane delivery to brain and spinal cord on inhibitory and excitatory output from the brain, $10,000
6. Foundation for Anesthesia Education and Research 1997-99—The effect of differential isoflurane delivery to brain and spinal cord on inhibitory and excitatory output from the brain, $70,000 ($70,000 matching departmental funds)
7. NIH R01 GM57970 Brain and Spinal Cord Contributions to Anesthetic Action 8/98-4/02 (Priority Score 120, Percentile 1.0). Total costs $713,026
8. NIH R01 GM61283 Anesthetic Effects on Sensorimotor Integration 2/01-2/06 (Priority Score 194, Percentile 16.9). Total costs $672,791
9. U.C. Davis Faculty Research Grant. Indirect effect of isoflurane and lidocaine on EEG activation. 7/1/01-6/30/02, $4,000
10. NIH R01 GM57970-4A1 Brain and Spinal Cord Contributions to Anesthetic Action 4/02-12/05 (Priority Score 197, Percentile 20). Total costs $1,284,689
11. NIH 3R01GM057970-05S1 Brain and Spinal Cord Contributions to Anesthetic Action. Minority Supplement grant. 7/03-7/04. Total costs $55,932
12. NIH P01 GM47818 Anesthetic Effects on Spinal Nociceptive Processing 8/04-7/09 (Priority Score 185). Total costs $804,325
13. NIH R01 GM61283A1 Anesthetic Effects on Sensorimotor Integration 12/05-12/9 (Priority Score 158, Percentile 9). Total costs $748,432

TEACHING

Post-Graduate:

1. Resident lectures on neuroanesthesia, anesthetic mechanisms, malignant hyperthermia, neuromuscular blocking drugs, volatile anesthetics, anesthesia research. 1991-2019
2. Anesthesiology Department Journal Club 2013-2016
3. UCSF Changing Practice of Anesthesia—Faculty. September 2014: Peri-operative Medicine and Healthcare Reform: Challenges and Opportunities for Anesthesiology

Graduate:

Guest lecturer for NPB 219 (E. Carstens, Instructor). 1998-2003

Guest lecturer for NPB 112 (E. Carstens, Instructor). 2001-2008

Guest lecturer for first year medical students—pain physiology 2002-2003

Facilitator, Application of Medical Principles 2002-2008

Guest Lecturer, 210B (Systemic Physiology) January 2006

Instructor of Record, Applied Physiology and Pharmacology 2007, 2008

Undergraduate:

NPB 10—Elementary Human Physiology (4 units). 2001-2009

Freshman Seminar: The Supreme Court and You. (2 units) 1998-2010

Human Physiology (Los Medanos College) 2020

Biology of Health (Los Medanos College) 2020-22

Epidemiology (Holy Names University) 2020

MENTORED STUDENTS, RESIDENTS AND POST-DOCTORAL SCHOLARS

1.	Kevin Schwartz, M.D.	Resident	1993
2.	Michael Borges, M.D	Resident	1994
3.	Agi Melton, M.D.	Resident	1994
4.	Etsuo Tabo, M.D.	Post-Doctoral Scholar	1997
5.	Steven Jinks	Graduate Student	1998-2001
6.	Chris Simons	Graduate Student	1998
7.	Xiao Wei Wang, M.D.	Post-Doctoral Scholar	1999
8.	Xiaoguang Chen, M.D.	Post-Doctoral Scholar	2000
9.	Makoto Sudo, M.D.	Post-Doctoral Scholar	2000
10.	Satoko Sudo, M.D.	Post-Doctoral Scholar	2000
11.	Alison Fitzgerald	Undergraduate Student	2000-2001
12.	Andrew Hall	Undergraduate Student	2001

13. John Martin, M.D.	Resident	2001
14. Steve Jinks, PhD.	Post-Doctoral Scholar	2001-2004
15. Jason Cuellar, BS	Graduate Student	2003-2004
16. Linda Barter, MsVM	Graduate Student	2004-2007
17. Mashawn Orth	Graduate Student	2004-2005
18. Carmen Dominguez, MD	Assistant Professor	2003-2005
19. Lauire Mark	Undergraduate Student	2005-2006
20. Matthew LeDuc	Medical Student	2005
21. Toshi Mitsuyo, M.D.	Post-Doctoral Scholar	2004-2005
22. Kevin Ng, M.D.	Resident	2005-2006
23. JongBun Kim, M.D.	Post-Doctoral Scholar	2006
24. Sean Shargh	Undergraduate Student	2006-2007
25. Aubrey Yao, M.D.	Resident	2006-2007
26. Alana Sulger	Undergraduate Student	2006-2007
27. Gudrun Kungys, M.D.	Resident	2007-2008
28. Jason Talavera	Medical student	2007
29. Onkar Judge	Medical student	2008
30. Andrew Cunningham	Undergraduate Student	2008
31. Lauren Boudewyn	Undergraduate Student	2008
32. Austin Kim	Undergraduate Student	2008
33. Jason Andrada	Graduate Student	2009-2010
34. Jun Ye	Graduate Student	2014-2015
35. Reihaneh Forghany	Resident/Faculty	2018-2021

SPECIAL ACTIVITIES:

Staff Anesthesiologist, American River Hospital, 1987-1992

Medical Advisor, CMT International (Charcot-Marie-Tooth), 1991-2000

Director, Case Conferences, Department of Anesthesiology, April-June, 1992

Proctor, Medical Board of California, 1992

Staff Membership, Sutter Davis Hospital, Davis, CA, 1992-1995

Consultant, Malignant Hyperthermia Hotline, Malignant Hyperthermia Association of the United States (MHAUS), 1992-2002

Associate, UC Davis Diagnostic Malignant Hyperthermia Laboratory, 1992-2010

Member, Subcommittee on Experimental Neuroscience and Biochemistry, American Society of Anesthesiologists, 1996

Finance and Executive Committees, UC Davis Department of Anesthesiology, 1996-2002

Quality Assurance Committee, U.C. Davis Department of Anesthesiology, 1998-2004

Course Director, Annual U.C. Davis Anesthesiology Update (CME meeting), 1996-2003
California Society of Anesthesiologists: Educational Programs Committee, 1998-2000

Coordinator, Grand Rounds, Department of Anesthesiology, 1996

Professional Billing Workgroup, U.C. Davis, 1996-98

Question Writer, American Board of Anesthesiology, 1998-2001

Member, UC Davis Animal Care Committee, 2000-2003

Member, UC Davis School of Medicine Personnel Committee, 2003—2007; Chair 2007

Member, UCD Committee on Academic Personnel (Appellate Sub-committee) 2009-11

Management Advisory Committee, Department of Anesthesiology, 2007

Ad Hoc Reviewer for *Anesthesiology, Hospital Topics, Journal of Clinical Anesthesia, Journal of Comparative Neurology, Regional Anesthesia and Pain Medicine, Pain, Brain Research, Journal of Neuroscience, Anesthesia and Analgesia, British Journal of Anaesthesia, Neuroscience, Cephalgia, Neuroscience Letters, Journal of Chromatography, Basic & Clinical Pharmacology & Toxicology, Therapeutics and Clinical Risk Management.*

Member, VA Merit Review Subcommittee, Alcohol and Drug Dependence, 2002-2005

Editor, American Board of Anesthesiology/ American Society of Anesthesiologists In-Training Examination 2003-2008

Associate Editor, *Anesthesiology* 2005—2011

Faculty Executive Committee, School of Medicine 2009-2010

Chair, Faculty Executive Committee, School of Medicine 2010-2011

Member of various hospital committees 2011-2016: Medical Staff Executive Committee, Quality Safety Committee, OR Committee, Surgical Services Steering Committee, Hospital Billing Group

BIBLIOGRAPHY

EDITED BOOKS

1. Antognini JF, Carstens EE, Raines DE. Neural Mechanisms of Anesthesia,

Humana Press, Totowa, NJ, 2002.

PUBLICATIONS

1. Antognini JF. Anaesthesia for Charcot-Marie-Tooth disease: a review of 86 cases. Canadian Journal of Anaesthesia 1992; 39(4):398-400.

2. Antognini JF and ND Kien. Cardiopulmonary bypass does not alter canine enflurane requirements. Anesthesiology 1992; 76:953-957.

3. Antognini JF. Intrathecal acetylsalicylic acid and indomethacin are not analgesic for a supramaximal stimulus. Anesthesia and Analgesia 1993; 76:1079-1082.

4. Antognini JF. Hypothermia eliminates isoflurane requirements at 20°C. Anesthesiology 1993; 78:1152-1156.

5. Antognini JF and GA Gronert. Succinylcholine causes profound hyperkalemia in hemorrhagic, acidotic rabbits. Anesthesia and Analgesia 1993; 77:585-588.

6. Melton AT, JF Antognini and GA Gronert. Prolonged duration of succinylcholine in patients receiving anticonvulsants: evidence for mild up-regulation of acetylcholine receptors? Canadian Journal of Anaesthesia 1993; 40(10):939-942.

7. Antognini JF and K Schwartz. Exaggerated anesthetic requirements in the preferentially anesthetized brain. Anesthesiology 1993; 79:1244-1249.

8. Antognini JF and PH Eisele. Anesthetic potency and cardiopulmonary effects of enflurane, halothane, and isoflurane in goats. Laboratory Animal Science 1993; 43(6):607-610.

9. Antognini JF. Splanchnic release of potassium after hemorrhage and succinylcholine in rabbits. Anesthesia and Analgesia 1994; 78:687-690.

10. Antognini JF, M Anderson, M Cronan, JP McGahan and GA Gronert. Ultrasonography: not useful in detecting susceptibility to malignant hyperthermia. Journal of Ultrasound in Medicine 1994; 13:371-374.

11. Antognini JF and ND Kien. A method for preferential delivery of volatile anesthetics to the *in situ* goat brain. Anesthesiology 1994; 80:1148-1154.

12. Antognini JF, BK Lewis and JA Reitan. Hypothermia minimally decreases nitrous oxide anesthetic requirements. Anesthesia and Analgesia 1994; 79:980-982.

13. Borges M and JF Antognini. Does the brain influence somatic responses to noxious stimuli during isoflurane anesthesia? Anesthesiology 1994; 81:1511-1515.

14. Antognini JF and ND Kien. Potency (minimum alveolar anesthetic concentration) of isoflurane is independent of peripheral anesthetic effects. Anesthesia and Analgesia 1995; 81:69-72.

15. Antognini JF and K Berg. Cardiovascular responses to noxious stimuli during isoflurane anesthesia are minimally affected by anesthetic action in the brain. Anesthesia and Analgesia 1995; 81:843-848.

16. Antognini JF. Creatine kinase alterations after acute malignant hyperthermia episodes and common surgical procedures. Anesthesia and Analgesia 1995; 81:1039-1042.

17. Gronert GA, NW Fleming and JF Antognini. Aberrant responses to muscle relaxants produced by diseases or drugs. Seminars in Anesthesia 1995; 14(4):283-290.

18. Hwang F, K Chun, JF Antognini and GA Gronert. Caffeine-halothane accuracy in MH testing. Acta Anaesthesiologica Scandinavica 1995; 39:1036-1040.

19. Antognini JF and K Mark. Hyperkalaemia associated with haemorrhagic shock in rabbits: modification by succinylcholine, vecuronium and blood transfusion. Acta Anaesthesiologica Scandinavica 1995; 39:1125-1127.

20. Antognini JF, R Wood and GA Gronert. Metocurine pharmacokinetics and pharmacodynamics in goats. Journal of Veterinary Pharmacology and Therapeutics 1995; 18:464-467.

21. Antognini JF. Movement associated with high cerebral concentrations of isoflurane: no evidence of seizure activity. Canadian Journal of Anaesthesia 1996; 43(3):310-314.

22. Antognini JF and GA Gronert. Extra-junctional receptors and neuromuscular blocking drugs. Current Opinion in Anaesthesiology 1996; 9:344-347.

23. Kien ND, JF Antognini, DA Reilly and PG Moore. Small-volume resuscitation using hypertonic saline improves organ perfusion in burned rats. Anesthesia and Analgesia 1996; 83:782-788.

24. Fleming NW, S Macres, JF Antognini and J Vengco. Neuromuscular blocking action of suxamethonium after antagonism of vecuronium by edrophonium, pyridostigmine or neostigmine. British Journal of Anaesthesia 1996; 77:492-495.

25. Antognini JF, PH Eisele and GA Gronert. Evaluation for malignant hyperthermia susceptibility in black-tailed deer. Journal of Wildlife Diseases 1996; 32(4): 678-681.

26. Antognini JF. The relationship among brain, spinal cord and anesthetic requirements. Medical Hypotheses 1997; 48:83-87.

27. Antognini JF and GA Gronert. Continued puzzles in malignant hyperthermia. Journal of Clinical Anesthesia 1997; 9:1-3.

28. Antognini JF and GA Gronert. Effect of temperature variation (22°C-44°C) on halothane and caffeine contracture testing in normal humans. Acta Anaesthesiologica Scandinavica 1997; 41: 639-642.

29. Antognini JF, MH Buonocore, EA Disbrow and E Carstens. Isoflurane anesthesia blunts cerebral responses to noxious and innocuous stimuli: a fMRI study. Life Sciences 1997; 61:PL349-354.

30. Antognini JF. Isoflurane potentiates metocurine via peripheral not central nervous system action. Journal of Veterinary Anaesthesia 1997; 24:6-9.

31. Disbrow E, M Buonocore, J Antognini, E Carstens and HA Rowley. The somatosensory cortex: a comparison of the response to noxious thermal, mechanical and electrical stimuli using functional magnetic resonance imaging. Human Brain Mapping 1998; 6:150-59.

32. Antognini JF, E Carstens, E Tabo and V Buzin. Effect of differential

delivery of isoflurane to head and torso on lumbar dorsal horn activity. Anesthesiology 1998; 88:1055-61

33. Antognini JF, E. Carstens. A simple, quantifiable, and accurate method for applying a noxious mechanical stimulus. Anesthesia and Analgesia 1998; 87:1446-9.

34. Antognini JF, S. Jinks, V. Buzin, E. Carstens. A method for differential delivery of intravenous drugs to the head and torso of the goat. Anesthesia and Analgesia 1998; 87:1450-2.

35. Antognini JF, E. Carstens. Macroscopic sites of anesthetic action: brain versus spinal cord. Toxicology Letters 1998; 100-101:51-58.

36 Antognini JF, E Carstens. Increasing isoflurane from 0.9 to 1.1 minimum alveolar concentration minimally affects dorsal horn cell responses to noxious stimulation. Anesthesiology 1999; 90:208-14.

37. Antognini JF, E Carstens, V Buzin. Isoflurane depresses motoneuron excitability by a direct spinal action: an F-wave study. Anesthesia and Analgesia 1999; 88:681-5.

38. Jinks S, JF Antognini, E Carstens V Buzin, C Simons. Isoflurane can indirectly depress lumbar dorsal horn activity via action within the brain. British Journal of Anaesthesia 1999; 82:244-49

39. Antognini JF, XW Wang. Isoflurane can indirectly depress auditory evoked potentials by action in the spinal cord. Canadian Journal of Anaesthesia 1999; 46:692-95

40. Melton AT, JF Antognini, GA Gronert. Caffeine- or halothane-induced contractures of masseter muscle are similar to those of vastus muscle in normal humans. Acta Anaesthesiologica Scandinavica 1999; 43:764-69

41. Antognini JF, XW Wang, E Carstens. Quantitative and qualitative effects of isoflurane on movement occurring after noxious stimulation. Anesthesiology 1999; 91:1064-71

42. Antognini JF, E Carstens. Isoflurane blunts electroencephalographic and thalamic/reticular formation responses to noxious stimulation in goats. Anesthesiology 1999; 91:1770-9

43. Antognini JF, XW Wang, E Carstens. Isoflurane action in the spinal cord blunts electroencephalographic and thalamic-reticular formation responses to noxious stimulation in goats. Anesthesiology 2000; 92:559-66

44. Antognini JF, XW Wang, M Piercy, E Carstens. Propofol directly depresses lumbar dorsal horn neuronal responses to noxious stimulation. Canadian Journal of Anesthesia 2000; 47:273-79

45. Antognini JF, Saadi J, Wang XW, Carstens E, Piercy M. Propofol action in both spinal cord and brain blunts electroencephalographic responses to noxious stimulation in goats. Sleep 2000; 24:26-31

46. Antognini JF, XW Wang, E Carstens. Isoflurane anaesthetic depth in goats monitored using the bispectral index of the electroencephalogram. Veterinary Research Communications 2000; 24:361-370

47. Antognini JF, Sudo M, Sudo S, Carstens E. Isoflurane depresses electroencephalographic and medial thalamic responses to noxious stimulation via an indirect spinal action. Anesthesia and Analgesia 2000; 91:1282-8

48. Sudo M, Sudo S, Chen XG, Piercy M, Carstens E, Antognini JF. Thiopental directly depresses lumbar dorsal horn neuronal responses to noxious mechanical stimulation. Acta Anaesthesiologica Scandinavica 2001; 45:823-829

49. Antognini JF, Chen XG, Sudo M, Sudo S, Carstens E. Variable effects of nitrous oxide at multiple levels of the central nervous system in goats. Veterinary Research Communications 2001; 25:523-538

50. Rosenberg H, Antognini JF, Muldoon S. Testing for malignant hyperthermia. Anesthesiology 2002; 96:232-37

51. Antognini JF, Carstens E, Atherley R. Does the immobilizing effect of thiopental in brain exceed that of halothane? Anesthesiology 2002; 96:980-6

52. Jinks SL, Antognini JF, Martin JT, Jung S, Carstens E, Atherley R. Isoflurane, but not halothane, depresses c-fos expression in rat spinal cord at concentrations that suppress reflex movement after supramaximal noxious stimulation. Anesth Analg 2002; 95:1622-8

53. Martin JT, Tautz TJ, Antognini JF. Safety of regional anesthesia in Eisenmenger's syndrome.Reg Anesth Pain Med. 2002;27:509-13.

54. Antognini JF, Carstens E. In vivo characterization of clinical anaesthesia and its components.Br J Anaesth. 2002;89:156-66.

55. Jinks SL, Simons CT, Dessirier JM, Carstens MI, Antognini JF, Carstens E. C-fos induction in rat superficial dorsal horn following cutaneous application of noxious chemical or mechanical stimuli. Exp Brain Res. 2002;145:261-9.

56. Jinks SL, Martin JT, Carstens E, Jung SW, Antognini JF. Peri-mac depression of a nociceptive withdrawal reflex is accompanied by reduced dorsal horn activity with halothane but not isoflurane. Anesthesiology 2003; 98:1128-38

57. Antognini JF, Atherley RJ, Carstens E. Isoflurane action in spinal cord indirectly depresses cortical activity associated with electrical stimulation of the reticular formation. Anesthesia Analgesia 2003; 96:999-1003

58. Jinks SL, Antognini JF, Carstens E. Isoflurane depresses diffuse noxious inhibitory controls in rats between 0.8-1.2 MAC. Anesthesia Analgesia 2003; 97:111-116

59. Eger EI 2nd, Xing Y, Laster M, Sonner J, Antognini JF, Carstens E. Halothane and isoflurane have additive minimum alveolar concentration (MAC) effects in rats.Anesth Analg. 2003;96:1350-3

60. Antognini JF, Jinks SL, Atherley R, Clayton C, Carstens E. Spinal anaesthesia indirectly depresses cortical activity associated with electrical stimulation of the reticular formation.Br J Anaesth. 2003;91:233-8

61. Sonner JM, Antognini JF, Dutton RC, Flood P, Gray AT, Harris RA, Homanics GE, Kendig J, Orser B, Raines DE, Trudell J, Vissel B, Eger EI 2nd. Inhaled anesthetics and immobility: mechanisms, mysteries, and minimum alveolar anesthetic concentration. Anesth Analg. 2003;97:718-40.

62. Jinks SL, Antognini JF, Carstens E. Spectral analysis of movement patterns during anesthesia.Anesth Analg. 2004; 98:698-702.

63. Jinks SJ, Antognini JF, Dutton RC, Carstens E, Eger EI. Isoflurane depresses windup of c-fiber evoked limb withdrawal with variable effects on nociceptive lumbar spinal neurons in rats. Anesth Analg 2004; 99:1413-9

64. Atherley RJ, Antognini JF. A rapid and simple method for determination of halothane, isoflurane and sevoflurane in blood using gas chromatography. Biomedical Chromatography 2004; 18:714-8

65. Jinks SJ, Antognini JF, Carstens E. Isoflurane differentially modulates medullary on and off neurons while suppressing hind-limb motor withdrawals. Anesthesiology 2004; 100:1224-34

66. Antognini JF, Jinks SJ, Carstens E, Atherley RJ. Preserved reticular neuronal activity during selective delivery of supra-clinical isoflurane concentrations to brain in goats and its association with spontaneous movement. Neuroscience Letters 2004; 361:94-7

67. Cuellar JC, Antognini JF, Carstens E. An in vivo method for recording single unit activity in lumbar spinal cord in mice anesthetized with a volatile anesthetic. Brain Res Prot 2004; 13:126-34

68. Cuellar JC, Antognini JF, Eger EI, Carstens E. Halothane depresses C-fiber-evoked windup of deep dorsal horn neurons in mice. Neurosci Letters 2004; 363:207-11

69. Atherley RJ, Weatherford V, Antognini JF, Jinks SL, Carstens E. A model for differential volatile anesthetic delivery to the upper and lower torso of the rabbit. J Pharmacol Tox Methods 2004; 50:145-52

70. Dominguez CL, Carstens E, Antognini JF. Carbon dioxide depresses the f-wave by a central, not peripheral, mechanism during isoflurane anesthesia.Anesth Analg 2005; 100:398-403

71. Jinks SL, Dominguez CL, Antognini JF. Drastic decreases in isoflurane MAC and limb movement force following acute reversible spinal cold-block and chronic spinalization in rats. Anesthesiology 2005; 102:624-32

72. Cuellar JM, Dutton RC, Antognini JF, Carstens E. Differential effects of halothane and isoflurane on lumbar dorsal horn neuronal windup and excitability. Brit J Anaesth 2005; 94:617-25

73. Antognini JF, Carstens E. Anesthesia, Amnesia and the Amygdala: reducing the fear of intraoperative awareness. (Editorial) Anesthesiology 2005; 102:711-2

74. Cuellar JM, Montesano PX, Antognini JF, Carstens E. Application of nucleus pulposus to L5 dorsal root ganglion in rats enhances nociceptive dorsal horn neuronal windup. J Neurophysiol 2005 Mar 2.

75. Barter L, Dominguez CL, Carstens E, Antognini JF. The effect of isoflurane and halothane on electroencephalographic activation elicited by repetitive noxious c-fiber stimulation. Neurosci Lett 2005 382:242-7.

76. Dominguez CL, Barter LS, Antognini JF. Intrathecal picrotoxin minimally alters electroencephalographic responses to noxious stimulation during halothane and isoflurane anesthesia. Acta Anaesth Scan 2005; 49:763-70

77. Orth M, Barter L, Dominguez C, Atherley R, Carstens E, Antognini JF. Halothane and propofol differentially affect electroencephalographic responses to noxious stimulation. Brit J Anaesth 2005; 95:477-84

78. Jinks SL, Atherley RJ, Dominguez CL, Sigvardt KA, Antognini JF. Isoflurane disrupts central pattern generator activity and coordination in the lamprey isolated spinal cord. Anesthesiology 2005; 103:567-75.

79. Antognini JF, Jinks SL, Carstens EE. The spinal cord, anesthesia and immobility: a re-examination. International Congress Series 2005

80. Carstens E, Antognini JF. Anesthetic effects on the thalamus, reticular formation and related systems. Thalamus and Related Systems. 2005

81. Antognini JF, Barter L, Carstens E. Overview movement as an index of anesthetic depth in humans and experimental animals. Comp Med, 2005; 55(5): 413-8.

82. Antognini JF, Carstens E. Measuring minimum alveolar concentration: more than meets the tail. Anesthesiology, 2005; 103(4): 679-80.

83. LeDuc ML, Atherley RJ, Jinks SL, Antognini JF. Nitrous oxide depresses electroencephalographic responses to repetitive noxious stimulation in the rat. Brit J Anaesth 2006; 96:216-21.

84. Barter LS, Hawkins MG, Brosnan RJ, Antognini JF, Pypendop BH.

Median effective dose of isoflurane, sevoflurane, and desflurane in green iguanas. Am J Vet Res. 2006; 67:392-7.

85. Mitsuyo T, Antognini JF, Carstens E. Etomidate depresses lumbar dorsal horn neuronal responses to noxious thermal stimulation in rats. Anesth Analg. 2006; 102:1169-73.

86. Orth M, Bravo E, Barter L, Carstens E, Antognini JF. The differential effects of halothane and isoflurane on electroencephalographic responses to electrical microstimulation of the reticular formation. Anesth Analg. 2006; 102:1709-14.

87. Hemmings HC, Jr, , Antognini JF. Do general anesthetics add up? Anesthesiology. 2006; 104:1120-2.

88. Merrill AW, Barter LS, Rudolph U, Eger EI 2nd, Antognini JF Carstens MI, Carstens E,. Propofol's effects on nociceptive behavior and spinal c-fos expression after intraplantar formalin injection in mice with a mutation in the gamma-aminobutyric acid-type(A) receptor beta3 subunit. Anesth Analg. 2006; 103:478-83

89. Antognini JF, Atherley RJ, Laster MJ, Carstens E, Dutton RC, Eger EI. A method for recording single unit activity in lumbar spinal cord in rats anesthetized with nitrous oxide in a hyperbaric chamber. J Neurosci Methods, 2006; 160(2): 215-22.

90. Ng KP, Antognini JF. Isoflurane and propofol have similar effects on spinal neuronal windup at concentrations that block movement. Anesth Analg, 2006, 103(6): 1453-8.

91. Antognini JF, Bravo E, Atherley R, Carstens E. Propofol, more than halothane, depresses electroencephalographic activation resulting from electrical stimulation in reticular formation. Acta Anaesthesiol Scand, 2006, 50(8): 993-8.

92. Mitsuyo T, Dutton RC, Antognini JF, Carstens E. The differential effects of halothane and isoflurane on windup of dorsal horn neurons selected in unanesthetized decerebrated rats. Anesth Analg, 2006, 103(3): 753-60.

93. Dutton RC, Carstens MI, Antognini JF, Carstens E. Long ascending propriospinal projections from lumbosacral to upper cervical spinal cord in the rat. Brain Res, 2006; 1119(1): 76-85.

94. Barter LS, Mark LO, Smith AC, Antognini JF. Isoflurane potency in the Northern Leopard Frog Rana pipiens is similar to that in mammalian species and is unaffected by decerebration. Vet Res Commun, 2007; 31(6): 757-63.

95. Antognini JF, Atherley RJ, Dutton RC, Laster MJ, Eger EI, Carstens E. The excitatory and inhibitory effects of nitrous oxide on spinal neuronal responses to noxious stimulation. Anesth Analg, 2007; 104(4): 829-35.

96. Antognini JF, Raines DE, Solt K, Barter LS, Atherley RJ, Bravo E, Laster MJ, Jankowska K, Eger EI. Hexafluorobenzene acts in the spinal cord, whereas o-difluorobenzene acts in both brain and spinal cord, to produce immobility. Anesth Analg, 2007; 104(4): 822-8.

97. Kim J, Atherley R, Werner DF, Homanics GE, Carstens E, Antognini JF. Isoflurane depression of spinal nociceptive processing and minimum alveolar anesthetic concentration are not attenuated in mice expressing isoflurane

resistant gamma-aminobutyric acid type-A receptors. Neurosci Lett, 2007; 420(3): 209-12.

98. Jinks SL, Carstens EE, Antognini JF. Glutamate receptor blockade in the rostral ventromedial medulla reduces the force of multisegmental motor responses to supramaximal noxious stimuli. Neurosci Lett, 2007; 426(3): 175-80.

99. Dutton RC, Cuellar JM, Eger EI, Antognini JF, Carstens E. Temporal and spatial determinants of sacral dorsal horn neuronal windup in relation to isoflurane-induced immobility. Anesth Analg, 2007; 105(6): 1665-74.

100. Kim J, Yao A, Atherley R, Carstens E, Jinks SL, Antognini JF. Neurons in the ventral spinal cord are more depressed by isoflurane, halothane, and propofol than are neurons in the dorsal spinal cord. Anesth Analg, 2007; 105(4): 1020-6, table of contents.

101. Barter LS, Mark LO, Jinks SL, Carstens EE, Antognini JF. Immobilizing doses of halothane, isoflurane or propofol, do not preferentially depress noxious heat-evoked responses of rat lumbar dorsal horn neurons with ascending projections. Anesth Analg, 2008; 106(3): 985-90, table of contents.

102. Barter LS, Antognini JF. Kinetics and potency of halothane, isoflurane, and desflurane in the Northern Leopard frog Rana pipiens. Vet Res Commun, 2008; 32(5): 357-65.

103. Yao A, Kim J, Atherley R, Jinks SL, Carstens E, Shargh S, Sulger A, Antognini JF. The effects of aromatic anesthetics on dorsal horn neuronal responses to noxious stimulation. Anesth Analg, 2008; 106(6): 1759-64.

104. Shnayderman D, Laster MJ, Eger EI 2nd, Oh I, Jinks SL, Antognini JF, Raines DE. Increases in spinal cerebrospinal fluid potassium concentration do not increase isoflurane minimum alveolar concentration in rats. Anesth Analg, 2008; 107(3): 879-84.

105. Talavera JA, Esser SK, Amzica F, Hill S, Antognini JF. Modeling the GABAergic action of etomidate on the thalamocortical system. Anesth Analg, 2009; 108: 160-67.

106. Barter LS, Mark LO, Antognini JF. Proprioceptive function is more sensitive than motor function to desflurane anesthesia. Anesth Analg, 2009; 108: 867-72.

107. Kungys G, Kim J, Jinks SL, Atherley RJ, Antognini JF. Propofol produces immobility via action in the ventral horn of the spinal cord by a GABAergic mechanism. Anesth Analg, 2009; 108: 1531-37.

108. Rivera R, Antognini JF. Perioperative drug therapy in elderly patients. Anesthesiology, 2009; 110: 1176-81.

109. Barter LS, Carstens EE, Jinks SL, Antognini JF. Rat dorsal horn nociceptive-specific neurons are more sensitive than wide dynamic range neurons to depression by immobilizing doses of volatile anesthetics: an effect patially reversed by the opioid receptor antagonist naloxone. Anesth Analg 2009; 109: 641-47.

110. Jinks SL, Carstens E, Antognini JF. Nitrous oxide-induced analgesia does not influence its immobilizing requirements. Anesth Analg 2009; 109:1111-6.

111. Judge O, Hill S, Antognini JF. Modeling the effects of midazolam on cortical and thalamic neurons. Neuroscience Letters 2009; 464:135-9.

112. Tautz TJ, Urwyler A, Antognini JF. Case scenario: Increased end-tidal carbon dioxide: a diagnostic dilemma. Anesthesiology 2010; 112:440-6.

113. Antognini JF. Anesthetic action: the importance of the spinal cord to immobility. Vet J. 2011; 187:151:2

114. Singh A, Antognini JF. Perioperative pharmacology in elderly patients. Curr Opin Anaesthesiology 2010; 23:449-54.

115. Singh A, Antognini JF. Perioperative hypotension and myocardial ischemia: diagnostic and therapeutic approaches. Ann Card Anaesth 2011; 14:127-32.

116. Andrada J, Livingston P, Lee BJ, Antognini J. Propofol and etomidate depress cortical, thalamic and reticular formation neurons during anesthetic-induced unconsciousness. Anesth Analg 2012; 114:661-9.

117. Antognini JF. Adventures in anesthetic mechanisms. Anesthesiology 2012; 116:701-4.

118. Cuellar J, Alataris K, Walker A, Yeomans DC, Antognini JF. Effect of high-frequency alternating current on spinal afferent nociceptive transmission. Neuromodulation 2013; 16:318-27.

119. Sohrakoff K, Westlake C, Key E, Barth E, Antognini JF Johnson V. Optimizing the OR: a bottom-up approach. Hosp Top 2014; 92:21-7.

120. O'Brien-Antognini JM, Antognini JF, Khatri V. How many operating rooms are needed to manage non-elective surgical cases? A Monte Carlo simulation study. BMC Health Services Res 2015; 15:487.

121. Antognini JF. Hospital surveys by the Centers for Medicare and Medicaid Services: An analysis of more than 34,000 deficiencies. J Patient Safety. 2019 Mar 20.

CASE REPORTS

1. Antognini JF and LH Hanowell. Intraoperative hypoxemia complicating sequential resection of bilateral pulmonary metastases. Anesthesiology 1991; 74:1137-1139.

2. Antognini JF and S Andrews. Anaesthesia for caesarean section in a patient with acute fatty liver of pregnancy. Canadian Journal of Anaesthesia 1991; 38(7):904-907.

3. Antognini JF. Chronic pain after methysergide: a new cause of ischemic monomelic neuropathy. Regional Anesthesia 1991; 16:337-338.

4. Lee G, JF Antognini and GA Gronert. Complete recovery after prolonged resuscitation and cardiopulmonary bypass for hyperkalemic cardiac arrest. Anesthesia and Analgesia 1994; 79:172-174.

5. Ogletree JW, JF Antognini and GA Gronert. Postexercise muscle cramping associated with positive malignant hyperthermia contracture testing. American Journal of Sports Medicine 1996; 24(1):49-51.

BOOK CHAPTERS

1. Gronert GA and JF Antognini. Malignant hyperthermia. In: Anesthesia, 1994; 4th Edition, Chapter 31, Volume 1, RD Miller (Ed.), Churchill Livingstone, New York,; pp. 1075-1093.

2. Jaffe RS, GA Gronert, NW Fleming and JF Antognini. Neuromuscular disorders and muscle relaxants. In: Clinical Neuroanesthesia, 1998; RF Cucchiara and JD Michenfelder (Eds.), Churchill Livingstone, pp. 449-474.

3. Gronert GA and JF Antognini. Clinical management of malignant hyperthermia. In: Hyperthermic and Hypermetabolic Disorders, 1996; Chapter 9, PM Hopkins and FR Ellis (Eds.), Cambridge University Press, England, pp. 119-131.

4. Antognini JF, T Tautz. Human Stress Syndrome. In: Malignant Hyperthermia. Eds: Schulte am Esch J, Scholz J, Wappler F., 2000; pp 346-353.

5. Gronert GA, Antognini JF. How to perform animal experiments. In: Conducting research in anaesthesia and intensive care. Eds: Zbinden AM, Thomson R. Butterworth-Heinemann, Oxford, 2000; pp. 468-498

6. Gronert GA, JF Antognini, I Pessah. Malignant Hyperthermia. In: Anesthesia, 2000; 5th Edition, RD Miller (Ed.), Churchill Livingstone, New York.

7. Antognini JF. Research of anesthetic mechanisms. In: Neural Mechanisms of Anesthesia. Eds: Antognini JF, Raines DE, Carstens E. Humana Press, 2002; Totowa, NJ

8. Caton D, Antognini JF. The development of concepts of mechanisms of anesthesia. In: Neural Mechanisms of Anesthesia. Eds: Antognini JF, Raines DE, Carstens E. Humana Press, 2002; Totowa, NJ

9. Antognini JF, Carstens E. Anesthesia, the spinal cord and motor responses to noxious stimulation. In: Neural Mechanisms of Anesthesia. Eds: Antognini JF, Raines DE, Carstens E. Humana Press, 2002; Totowa, NJ

10. Antognini JF, Raines DE, Carstens E. The future of anesthetic mechanisms research. In: Neural Mechanisms of Anesthesia. Eds: Antognini JF, Raines DE, Carstens E. Humana Press, 2002; Totowa, NJ

11. Perounasky M, Antognini JF. Glutamate receptors: physiology and anesthetic pharmacology. In: Neural Mechanisms of Anesthesia. Eds: Antognini JF, Raines DE, Carstens E. Humana Press, 2002; Totowa, NJ

12. Antognini JF, Carstens E. Spinal cord actions of halothane, thiopental and isoflurane. In: Molecular and basic mechanisms of anesthesia. Eds: Urban BW, Barann M. Pabst, 2002, Berlin, pp 474-79.

13. Antognini JF, Carstens E, Sudo M, Sudo S. Thiopental directly depresses lumbar dorsal horn neurons in goats. In: Molecular and basic mechanisms of anesthesia. Eds: Urban BW, Barann M. Pabst, 2002, Berlin, pp 480-83.

14. Jinks SL, Antognini JF. Anesthetic-induced immobility. In: Neuroscientific Foundations of Anesthesiology. Eds: Mashour GA, Lydic R. Oxford University Press, 2011, Oxford, pp 107-119.

LETTERS TO THE EDITOR

1. Antognini JF. Response to Angell editorial regarding prior release of studies. New England Journal of Medicine 1992; 326(14):958.

2. Antognini JF. Anesthetic management in Charcot-Marie-Tooth disease. Anesthesia and Analgesia 1992; 75:313.

3. Borges M and JF Antognini. Anaesthesia for Mauriac's syndrome. Anaesthesia and Intensive Care 1993; 21(1): 123-124.

4. Antognini JF. Suppression of information by medical journals. New England Journal of Medicine 1993; 328(7):511.

5. Antognini JF. Response to Drs. Hall and Sullivan Letter to the Editor. Anesthesiology 1993; 79:1443-1444.

6. Antognini JF. Response to Dr. Adachi *et al* Letter to the Editor regarding exaggerated anesthetic requirements. Anesthesiology 1994; 81(2):522-523.

7. Antognini JF. Neurologic dysfunction after isoflurane sedation. Critical Care Medicine 1995; 23:789.

8. Antognini JF and GA Gronert. Succinylcholine sensitivity in cerebral palsy. Anesthesia and Analgesia 1995; 80:1250.

9. Fleming NW, S Macres, JF Antognini and J Vengco. Response to comment from Dr. Graham regarding anticholinesterases and subsequent duration of block of suxamethonium. British Journal of Anaesthesia 1997; 78(4):480-481.

10. Melton A, Gronert GA, Antognini JF. Chemical skinning artifact appears to increase sensitivity of masseter muscle to halothane and succinylcholine. Anesthesiology 2000; 92:628-629.

ABSTRACTS

1. Melton AT, JF Antognini and GA Gronert. Absence of abnormal potassium efflux after succinylcholine in patients on anticonvulsants: evidence for mild up-regulation of acetylcholine receptors. Western Anesthesia Residents Conference. 1993

2. Schwartz K and JF Antognini. Is the brain the major site of anesthetic action? Western Anesthesia Residents Conference. 1993

3. Macres SM, NW Fleming and JF Antognini. Neuromuscular blocking effects of succinylcholine before and after administration of cholinesterase inhibitors. Western Anesthesia Residents Conference. 1994

4. Borges MF and JF Antognini. Does the brain influence somatic responses to noxious stimuli? Western Anesthesia Residents Conference. 1994

5. Kien ND, JF Antognini, DA Reilly and PG Moore. Small-volume resuscitation using hypertonic saline improves organ perfusion in burn rats. European Journal of Emergencies 1994; 7:34.

6. Reilly DA, JF Antognini, PG Moore and ND Kien. Small volume resuscitation using hypertonic saline improves organ perfusion in burn rats. Proceedings of the American Burn Association 1994; 26:142.

7. Borges MF and JF Antognini. Does the brain influence somatic responses to noxious stimuli during isoflurane anesthesia? Third Annual Biomedical Research Colloquium, 1994; page 6.

8. Kien ND, JF Antognini, DA Reilly and PG Moore. A comparison of hypertonic to isotonic solution on organ blood flow in burned rats. Anesthesiology1994; 81(3A):A310.

9. Antognini JF, BK Lewis and JA Reitan. Hypothermia minimally decreases nitrous oxide anesthetic requirements. Anesthesiology 1994; 81(3A): A891.

10. Antognini JF and M Borges.Does the brain influence somatic responses to noxious stimuli during isoflurane anesthesia? Anesthesiology 1994; 81(3A): A1483.

11. Buonocore MH, RJ Maddock and J Antognini. Noise cancellation techniques for functional MRI. Cognitive Neuroscience Society Second Annual Meeting, 1995; page 54.

12. Disbrow E, M Buonocore, J Antognini, E Carstens and R Shumway. Time series analysis: an alternative method for processing FMRI data. Cognitive Neuroscience Society Second Annual Meeting, 1995; page 61.

13. Antognini JF, MH Buonocore, E Disbrow and E Carstens. The effect of isoflurane on cerebral responses to noxious stimuli as assessed by functional magnetic resonance imaging. Anesthesiology 1995; 83(3A):A861.

14. Antognini JF. Creatine kinase after acute malignant hyperthermia (MH) episodes compared to CK changes after common surgical procedures. Anesthesiology 1995; 83(3A):A1003.

15. Antognini JF and GA Gronert. Effect of temperature on halothane caffeine contracture testing in humans. VIIIth International Workshop on Malignant Hyperthermia, 1996; page 74.

16. Melton AT, JF Antognini and GA Gronert. In vitro contracture tests on normal human masseter muscle. Anesthesia and Analgesia 1997; 84:S368.

17. Antognini J, E Carstens, E Tabo and V Buzin. The effect of selective delivery of isoflurane to the brain on nociceptive responses of spinal dorsal horn neurons. Association of University Anesthesiologists, 1997; pp. 26-27.

18. Antognini J, E Carstens, E Tabo and V Buzin. Effects of selective delivery of isoflurane to the brain on nociceptive responses of lumbar dorsal horn neurons in the goat. American Pain Society Annual Meeting, 1997; May.

19. Antognini J, E Carstens, E Tabo and V Buzin. The effect of selective delivery of isoflurane to the brain on spinal dorsal horn neurons. Fifth International Conference on Molecular and Cellular Mechanisms of Anaesthesia, 1997; page 31.

20. Antognini JF, E Carstens, E Tabo and V Buzin.The effect of selective delivery of isoflurane to the brain on spinal dorsal horn neurons. American Society of Anesthesiologists Annual Meeting; Anesthesiology 1997; 87:A292

21. Buzin V, JF Antognini, S. Jinks, E. Carstens. Does isoflurane action in the brain influence lumbar dorsal horn activity? Association of University Anesthesiologists Annual meeting, San Francisco, 1998; CA pp 85-86.

22. Antognini JF, XW Wang, E Carstens. Quantitative and qualitative effects of isoflurane on movement occurring after noxious stimulation. Association of University Anesthesiologists Annual meeting, Pittsburgh, 1999; PA pp 185-186

23. Antognini JF, E Carstens. Isoflurane blunts EEG responses to noxious stimulation. Association of University Anesthesiologists Annual meeting, Pittsburgh, 1999; PA pp 187-188

24. Antognini JF, Wang XW, E Carstens. Isoflurane action in the spinal cord blunts EEG and thalamic/reticular formation responses to noxious stimulation in goats. American Society of Anesthesiologists Annual Meeting; Anesthesiology 1999; 91:A318

25. Antognini JF, Wang XW, E Carstens. Quantitative and qualitative effects of isoflurane on movement occurring after noxious stimulation. American Society of Anesthesiologists Annual Meeting; Anesthesiology 1999; 91:A324

26. Antognini JF, Sudo M, Sudo S, Carstens E. Isoflurane depresses electroencephalographic and medial thalamic responses to noxious stimulation via an indirect spinal action. Association of University Anesthesiologists Annual meeting, Salt Lake City, UT. 2000; May 2000

27. Antognini JF, Sudo M, Sudo S, Carstens E. Isoflurane depresses electroencephalographic and medial thalamic responses to noxious stimulation via an indirect spinal action. American Society of Anesthesiologists Annual Meeting; 2000; October 2000, A-746

28. Antognini JF, Carstens E, Atherley R, Hall A, Fitzgerald A. Halothane and thiopental ablate movement primarily via a spinal cord action. Soc Neurosci Annual Meeting Abstracts 2001; Nov 2001

29. Antognini JF, Carstens E, Atherley R, Hall A, Fitzgerald A. Halothane and thiopental ablate movement primarily via a spinal cord action. 6th International Meeting Molecular and Cellular Mechanisms of Anesthesia, June 2001, Bonn, Germany, 2001; 5B01, pg 45.

30. Sudo M, Sudo S, Antognini JF, Carstens E, Atherley R. Thiopental directly depresses lumbar dorsal horn neuronal responses to noxious mechanical stimulation in goats. 6th International Meeting Molecular and Cellular Mechanisms of Anesthesia, June 2001, Bonn, Germany, 2001; 5B11, pg 45.

31. Jinks SL, Antognini JF. Peri-mac isoflurane blocks the effect of noxious mechanical counterstimuli on heat-evoked responses of spinal dorsal horn neurons. Program No. 259.14. 2002 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2002. Online.

32. Antognini JF, Jinks SL, Martin JT, Carstens EE. Effects of volatile anesthetics on nociceptive sensorimotor integration. Program No. 667.7. 2002 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2002. Online.

33. Jinks SL, Antognini JF. Differential modulation of on- and off-neurons in the rostral ventromedial medulla by isoflurane is consistent with its depressant action on noxious stimulus-evoked movement. Program No. 481.12. 2003 Abstract Viewer/Itinerary Planner. Washington, DC: Society for Neuroscience, 2003. Online.

34. S.L. Jinks, E. Carstens, J.F. Antognini. Medullary on-cells facilitate multilimb movements elicited by intense noxious stimulation Program No. 296.7. *2004 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2004. Online.

35. C.L. Dominguez, E. Carstens, J.F. Antognini. Carbon dioxide depresses the f-wave by a central, not peripheral, mechanism during isoflurane anesthesia Program No.

374.3. *2004 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2004. Online.

36. J.M. Cuellar, P.X. Montesano, J.F. Antognini, E. Carstens. Application of nucleus pulposus to l5 dorsal root ganglion in rats enhances nociceptive dorsal horn neuronal windup Program No. 407.4. *2004 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2004. Online.

37. J.M. Cuellar, R.C. Dutton, J.F. Antognini, S.L. Jinks, T. Mitsuyo, E. Carstens. Differential effects of halothane (hal) and isoflurane (iso) on dorsal horn neuronal windup Program No. 644.1. *2004 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2004. Online.

38. J.F. Antognini, S.L. Jinks, J.M. Cuellar, R.C. Dutton, E.I. Eger, E.E. Carstens. Isoflurane depresses windup of c-fiber evoked limb withdrawal with variable effects on nociceptive lumbar spinal neurons in rats Program No. 644.2. *2004 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2004. Online.

39. C.T. Simons, S.L. Jinks, C.L. Dominguez, R.J. Atherley, E.E. Carstens, K.A. Sigvardt, J.F. Antognini. Isoflurane disrupts inter-segmental coordination of central pattern generators in lamprey Program No. 644.3. *2004 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2004. Online.

40. J.F. Antognini T.Mitsuyo, R.C. Dutton, E. Carstens. Differential effects of halothane and isoflurane on windup of nociceptive dorsal horn neurons. Prog. No. 863.13, *2005 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2005. Online.

41. L.S. Barter, M.M. Orth, E.E. Carstens, J.F. Antognini. Isoflurane, more than halothane, depresses eeg responses to electrical stimulation in reticular formation Program No. 983.19. *2005 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2005. Online.

42. J.F. Antognini, L.S. Barter, K. Solt, D.E. Raines, E. Eger, M. Laster. Hexafluorobenzene acts in spinal cord, while o-difluorobenzene can act in either

brain or spinal cord to produce immobility. Program No. 54.17. *2006 Abstract Viewer/Itinerary Planner.* Washington, DC: Society for Neuroscience, 2006. Online.

43. Carstens EE, Iodi Carstens M, Antognini JF, Dutton RC. Long ascending propriospinal projections from lumbosacral to upper cervical spinal cord in the rat. Program No. 983.19. *2005 Abstract Viewer/Itinerary Planner*. Washington, DC: Society for Neuroscience, 2005. Online.

44. Ferron J, Antognini JF, Amzica F. Impact of anesthesia induciton on the intrinsic properties of cortical neurons: An in vivo study. 2006 Abstract viewer/Itinerary Planner. Washington DC: Society for Neuroscience, Program No. 237.20 (Online).

45. Barter LS, Jinks SL, Carstens EE, Antognini JF. Anesthetic effects on spinal projection neurons. 2007 Abstract viewer/Itinerary Planner. Washington DC: Society for Neuroscience, Program No. 822.4 (Online).

46. Carstens EE, Dutton RC, Antognini JF, Cuellar JM, Eger EL. Temporal and spatial determinants of sacral dorsal horn neuronal windup in relation to isoflurane-induced immobility. 2007 Abstract viewer/Itinerary Planner. Washington DC: Society for Neuroscience, Program No. 822.8 (Online).

47. Antognini JF, Yao A, Kim J. Effects of aromatic anesthetics on dorsal horn neuronal responses to noxious stimulation. 2007 Abstract viewer/Itinerary Planner. Washington DC: Society for Neuroscience, Program No. 823.6 (Online).

48. Kim JB, Yao A, Carstens E, Jinks SL, Antognini JF. Ventral spinal cord neurons are more depressed by anesthesia than are dorsal spinal cord neurons. A-136, Annual meeting of the American Society of Anesthesiologists; October 17th-21st, 2007, San Francisco, CA.

49. Yao A, Kim JB, Atherley RJ, Antognini JF. Effects of aromatic anesthetics on dorsal horn neuronal responses to noxious stimulation. A-1927, Annual meeting of the American Society of Anesthesiologists; October 17th-21st, 2007, San Francisco, CA.

50. Barter LS, Carstens E, Jinks SL, Antognini JF. Halothane and isoflurane depress dorsal horn nociceptive specific but not wide dynamic range neurons. A-1915, Annual meeting of the American Society of Anesthesiologists; October 17th-21st, 2007, San Francisco, CA.

51. Judge O, Antognini JF. Modeling the effects of midazolam on cortical and thalamic neurons. Annual meeting of the International Society for Anaesthetic Pharmacology; October 17th, 2008, Orlando, FL.

52. Antognini JF, Judge O. Modeling the effects of midazolam on cortical and thalamic neurons. S-280, Annual meeting of the International Anesthesia Research Society; March 16th, 2009, San Diego, CA.

53. Forghany R, Antognini JF. An analysis of the role of anesthesiology providers in hospital deficiencies published by CMS. WARC May 4-6, 2018, San Diego, CA.

LIMITED DISTRIBUTION

1. Antognini, JF. The HOTLINE. The Communicator 12(2):2-3, 1994; March-April.

2. Antognini JF. Neuroanesthesia, Parts I and II. U.C. Davis Anesthesiology Update: 1994; pp. 113-116.

3. Antognini JF. Anesthesia and the CMT patient. CMT Newsletter 12(3):10, 1995; June.

4. Antognini JF. Current research in anesthesia. U.C. Davis Anesthesiology Update: 1995; pp. 66-71.

5. Antognini JF. Anesthesia outcomes—what's important: what we do, or how we do it? U.C. Davis Anesthesiology Update: 1996; pp. 54-61.

6. Antognini JF. Basics of trauma anesthesia. U.C. Davis Anesthesiology Update: 1996; pp. 129-134.

7. Antognini JF. Current issues in trauma anesthesia. U.C. Davis Anesthesiology Update: 1998; pp. 118-122.

8. Antognini JF. Anesthesia outcomes—what's important: what we do, or how we do it? U.C. Davis Anesthesiology Update: 1999; pp. 3-9.

9. Antognini JF. Medical pain relief in childbirth. In: The Baby Guide. Ed: Smith TM. Hazen Publishing, Inc. Auburn, Calif. 1999; pp. 45-47.

separately inflected?

A They were individually inflected.

Q All right. Now, let's look at the item number one, and tell us what that is?

A Item number one is here to the left forehead. That's a laceration. The tear in the skin is this inverted y-shaped tear, and the area of darkening around it is the abrasion of the skin, or the scratching of the skin caused by the striking object. This is a laceration that's approximately one inch in length. And it extends through the full thickness of the skin of the scalp here and extends down to underlying bone.

Q And was there any fracture underlying that?

A No, sir, there was not.

Q All right. And then number two is -- well, let me go back just a second. Did you -- well, go ahead. Number two, talk about number two, the fracture there.

A Laceration number two is right here at the outer -- called the lateral campus (phonetically), or the outside of the left eye here. It's a non-full thickness, or it doesn't go all the way through the skin, but it's a partial tear in the skin, and extends down into the soft tissues here. It was approximately three quarters of an inch in length. There was no palpable fracture, or I could not feel any break in the bones beneath that

laceration.

Q Now, the shaded areas over the eyes, what does that indicate?

A The shaded areas of the eyes are -- are peri-orbital, or around the eye contusions. They're bruises. And they're the result of blood seeping into the soft tissues around the eyes as a result of basilar skull factures, and these are skull factures that -- of -- of the bones of the face behind the eyes and behind the nose.

Q And when you say the bones behind the face fractured, what does fractured mean?

A The bones are broken, and so the bones -- the bones behind these structures here, behind the eyes and behind the nose, penetrated to the cranial vault where the brain is. And when these are fractured, then blood is allowed to seep into the soft tissues of the front part of the face, so you get these -- these peri-orbital contusions here.

Q It looks like bruising?

A Yes, it does. It looks like a black eye.

Q And what was, based on your examination, the cause of the fracture of the bones behind the eyes and the face?

A The blunt force trauma of the head, the multiple

blows to the head resulting in multiple skull factures of the calvarium, or the top of the head as well as the bones behind the face.

Q All right. Let's go to number ---

A There's one other injury on this page if you want to do it.

Q Oh, I'm sorry. Right.

A There is an area in the left forehead extending down into the left side of the face, of a little bit more faint bruising, or an -- it's a bruise of the skin. More than likely this did not result as a direct blow to the skin. At the time of the scene investigation, Mr. Larke's head was laying on the left, so that this part of his face would be in contact with the floor. He has other lacerations and blunt injury to this right side of his face and right side of his head, and very likely that when this side of his head was being impacted, this side of his head was against the floor causing this bruising to happen.

Q Okay. Thank you. Looking at what's been marked as State's Exhibit 22B, and that -- orient us, if you would, what we're looking at there?

A This is the top of the head. It's a diagram of the top of the head of Mr. Larke. It shows what's called the right parietal region of the skull -- or of the

scalp, three separate lacerations. Number five, six and seven. Number five is -- two of them are obliguely oriented lacerations. Number five is 1.7 inches in length. Number six is 1.2 inches in length. And 1.7 is about three-quarters of an inch in length. All of them are full thickness in that they penetrate all the way through the skin down to the underlying bone. And at the time of autopsy, I was able to feel boney fractures, or fractures of the skull, breaks of the bone beneath these lacerations. And each is surrounded by an area of contusion here, or bruising.

Q All right. When you say you were able to determine -- feel bone fractures, what does that mean, when you feel the bone fractures?

A During the autopsy we go through layers. We look at the outer portion of the body, and then we start to look at the inside of the body. As we're doing the outer portion of the autopsy we try to anticipate what we may find. So as I'm feeling with my fingers the depths of these wounds to see if they go all the way through, to see if there is any underlying injury, I'm able to feel the bone. And as I move my finger across that bone I can feel that there has been a break, or a disruption of that bone, and I can tell that it's -- there's going to be a skull fracture. That certainly

indicates a greater amount of force required to break the bone than it would be just to tear the skin.

Q This one had torn skin, fractured bone underneath and the skull, and then the shaded area is bruising or blood collecting?

A That's correct.

Q Let me show you State's Exhibit Number 22D.

A State's Exhibit D is a diagram of the right side of the head of Mr. Larke showing -- starting on the right side of the forehead and extending back into the temporal region, a large laceration of skin which is three inches in length with its surrounding abrasion. There was a palpable -- there was -- I was able to feel beneath this laceration a skull fracture as well.

Q So the skull underneath that wound was also broken?

A That's correct.

Q State's Exhibit Number 22E?

A 22E is a diagram of the left side of the head of Mr. Larke, showing a laceration in what would be called the left parietal region, which is one and a half inches in length. I was not able to feel a fracture associated with this one, excuse me, however, it did extend all the way through the skull down to the underlying bone, but there was no palpable fracture that I could feel.

Q State's Exhibit 22C?

A State's Exhibit 22C is a diagram of the back portion of the head of Mr. Larke, showing two separate lacerations. One that's been numbered as number eight, which is about one and three-quarter inches in length, and number nine which is two inches in length. Both of these are again full thickness. They go all the way through the skin to the underlying bone, and each of them is associated with a skull fracture that I could feel.

Q A skull fracture related to these also?

A That's correct.

Q Okay. Now, so in summary as to these, Mr. Larke had wounds to the top of his head, the left side of his head, the right side of his head, the back of his head, and front of his head?

A That's correct.

Q Nine total?

A Nine total.

Q In addition to those nine lacerations and related skull fractures, did you find any other evidence of injury to his head and neck?

A Besides the lacerations that we described, there were bruises to the tip of each ear, there were -- there was a large bruise involving the left -- yes, there is a large bruise involving the musculature beneath the left

shoulder in this area here. And then there was a bruise on the back of the right hand. As we took the injuries -- as we continued the autopsy, went from the outside inward, we saw lacerations to the head. We saw the skull fractures that we've described, and these were primarily centered here on the right side, the back. And these from the right side extended towards the front to involve the bones of the base of the skull, basilar skull fractures. They went all the way across so at the time of autopsy you were able to move the head in such a way that these created a hinge-type fracture, in that the skull was basically almost broken in two. There were hemorrhage around the brain called subarachnoid hemorrhage, which is blood that has collected on the outer surface of the brain, and there were also bruises of the brain itself beneath these areas of impact, as well associated with the skull fractures to the base of the brain -- base of the skull.

Q Did I ask you also as part of this to prepare what is now marked as State's 22F, a drawing showing a sideview, or cross-section of the skull?

A Yes, you did.

Q Did you review this? Is this accurate as far as relates to this case?

A It is.

said contusions to the brain, she had hemorrhages overlying the brain?

A That's correct.

Q I have asked you to look at what's been marked as State's Exhibit Number 7, the bat. And I will ask you in regard to Mrs. Larke, if the bat, the wounds are consistent with the ones that you found on her body?

A Yes, they are. Yes, they are.

Q Consistent in that they could have been inflicted by that object?

A Correct.

Q I will ask you once again, also in regard to the degree of force that would have been necessary to inflict these wounds on Mrs. Larke's head?

A Much as to Mr. Larke's head, these are not trivial swings with this bat. These are full force, great deal of energy transmitted, homerun type swings.

Q All right. In your exam of -- autopsy, or on the scene, did you also note any other blunt force injuries to Mrs. Larke's body? I'm going to show you what's been marked as State's Exhibit Number 21I. This likewise is a standard form drawing that you use?

A That's correct. This is a diagram they shows both the front and back portion of the body of -- in this case representing Mrs. Larke. The shaded areas are

bruises of the skin. There is one to the back of the left elbow. There are two to the back and inner aspect of the left wrist, a bruise to the inner aspect -- the back and inner aspect of the right forearm, and within the right wrist there is a grouping of bruises and superficial abrasions, or scratches of the skin.

Q Are those consistent with being called defensive wounds?

A Yes, they would be.

Q And if you would, based on your examination, training, and experience, demonstrate to the jury how you believe these most likely occurred in Ms. Larke's case?

A The most consistent way to get these type of injuries, given the trauma or the injuries to Mrs. Larke's head, would be if she put her arms up across her head like this, so as she's getting blows to the top of her head, they're also impacting the elbow and the inner aspects of the forearms and wrists.

Q And could these blows indicated by the marks on her arms, could they have also been separate blows from those shown on the head?

A I believe them to be, yes.

Q All separate? So that the bruising indicates additional blows, in addition to the ones you found on

the head, or have you formed an opinion on that?

A I think that each of the bruises to the forearms -- let's see if I can explain this. I think each one of these is a separate impact to the forearm. I don't believe I can say whether each one of these would be blows 10, 11, 12, 13 and 14, or if they make have occurred at the same time as some of the blows to the top of the head.

Q All right. And based on the information that you have obtained, and your examination, you say you did determine that Mrs. Larke was alive for some period of time. Do you have an estimate of how long she may have been -- survived?

A My estimate would be a few minutes, and when pressed that would be in the range of three to five minutes.

Q How about Mr. Larke?

A Mr. Larke was face down when he was discovered at the time of the scene investigation, and the blood present from his basilar skull fractures is going to probably gravity, and so it's leaking out over his nose as opposed to staying in his sinuses. He did not have blood present within his airways. Although there was a fair amount of blood present at the scene, similar injuries to Mr. Larke as to Mrs. Larke, somewhat similar

circumstances, my estimate would be about the same time, a few minutes, three to five.

Q Okay. Now, Dr. Ward, when you visited the residence of Mr. and Mrs. Larke, it was located in Greenville County, correct?

A That's correct.

MR. ARIAIL: Thank you. Answer any questions that either defense counsel may have.

THE COURT: Mr. Abdalla.

MR. ABDALLA: May I have one, moment, Your Honor, please?

CROSS-EXAMINATION

BY MR. ABDALLA:

Q Good morning, Dr. Ward, it's still morning. As a matter of fact, we've met previously?

A That's correct.

Q I'm intimated, you have too much education. Now, you testified about the blunt force trauma. You say there were approximately nine strikes to each of the victims in this case, isn't that true?

A That's correct.

Q And with regard to Mr. Larke, there was some bruising, let me see if I can find the correct exhibit, I apologize, State's Exhibit Number 22A, if I could show this to the witness, for instance near the eye?