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IN THE CHANCERY COURT OF DAVIDSON COUNTY, TENNESSEE
TWENTIETH JUDICIAL DISTRICT PART IV

KEVIN BURNS, et al.,,	)	CAPITAL CASE
	)	EXECUTION DATE:
Plaintiffs,	)	AUGUST 5, 2025
	)	
v.	)	No. 25-0414-IV
	)	
	)	
FRANK STRADA, et al.,	)	
	)	
	)	
Defendants.	)	

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

JOSEPH F. ANTOGNINI, does hereby declare under penalty of perjury that the following is true and correct:

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 States of California and Georgia. 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 Preliminary Injunction *Kevin Burns, et al., v. Frank Strada, et al.,* No. 25-0414-IV, dated June 30, 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 Tennessee's lethal injection protocol, and the effectiveness of the procedures therein, especially related to Mr. Byron Black. 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.

Materials Reviewed

4. I have conferred with attorneys for Defendants. Among the documents I have relied upon in connection with this declaration are: Tennessee's execution protocol; Plaintiff's Motion (dated June 30, 2025); publications and materials listed in the "References Cited" section; medical records of Byron Black; and the declaration of Dr. Gail Van Norman, dated June 27, 2025.

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,* 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-4617-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; 10) I have been deposed and testified in *Hoffman v. Westcott, et al*., cv-25-169, in the United States District Court for the Middle District of Louisiana.

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

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.

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. And, Dr. Van Norman has stated that "Pentobarbital and thiopental have been used in medical care and are essentially equivalent in all pharmacological characteristics relevant to lethal injection executions." (page 18, at bottom, of her report).

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

15. 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.

16. More recent studies in humans using post-mortem computed tomography (PMCT) show that fluid accumulates in the 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 with the passage of time after death in accordance with progression of postmortem changes (pulmonary congestion and edema) in the corpse."

17. 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.

18. 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[3]) 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

[3] See Acta Scandinavica Medica 1964 in References Cited

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.

19. 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.[4] Thus, the finding of froth in inmates who were executed by lethal injection does not indicate that this froth was generated ante-mortem.

20. 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 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

[4] 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

injection does not conclusively indicate that this froth was generated ante-mortem, or by conscious attempts to breathe.

21. 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).[5]

22. In her declaration Dr. Van Norman states that Mr. Black, because of his medical conditions, particularly his depressed cardiac function (decreased ventricular contractility), will be more sensitive to pentobarbital (see her report, at top of page 16). I agree. Mr. Black's blood pressure and blood flow will decrease more quickly as compared to a person with normal cardiac function.

23. Dr. Van Norman's discussion of awareness during anesthesia, which she attempts to support with studies using the isolated forearm technique (see her references 52, 62-66, among other reports), is misplaced in the setting of execution with pentobarbital. First off, the studies that used the isolated forearm techniques administered light levels of anesthesia, for obvious reasons: if the anesthetic depth was too great the patients would not have responded. She has not produced any evidence that awareness and

5 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."

responsiveness occur at the doses of pentobarbital that are contemplated in the Tennessee protocol (nor could she, because that evidence is not there). Secondly, she admits that patients in these IFT studies "do not respond with movement to even strong stimuli, such as a surgical incision" (p. 20 of her report). If the patients do not respond to surgical incision, an obvious noxious and painful stimulus, then why would we expect an inmate to respond to a purported noxious stimulus?[6]

24. A third problem with Dr. Van Norman's opinion regarding awareness is that it flies in the face of well-accepted anesthesia practice. On the one hand, she states that anesthesia awareness is common, and that multitudes of patients are aware of their surgeries (some of which are hours long, with body cavities cut wide open). But if anesthesiologists were really concerned about this issue of patient suffering during general anesthesia then we would stop administering anesthesia to patients. Furthermore, I suspect that Dr. Van Norman has practiced anesthesia at the standard of care, i.e., she administered anesthesia like most anesthesiologists, more-or-less, so she apparently thinks it's acceptable for her patients to suffer, but that Mr. Black should be spared.

6 Just to be clear, my opinion, to a medical and scientific certainty, is that an inmate administered 5 grams of pentobarbital will not be subjected to, nor experience, any significant noxious stimulus.

25. As stated above, pentobarbital would produce profound unconsciousness. In addition, pentobarbital would depress cardiac function so much that the brain would not receive adequate blood flow to maintain proper function. And, as Dr. Van Norman has stated, Mr. Black will have oxygen levels "too low to support cardiac contraction" (her report page 16, D.2), and the low oxygen levels would cause unconsciousness.

26. Thus, Dr. Van Norman expects us to believe that despite 1) the presence of a huge dose of pentobarbital which causes profound brain depression; 2) minimal-to-no blood flow to the brain (or any other part of the body); and 3) very low oxygen levels, Mr. Black will have awareness and will be able to perceive 1) the shocks delivered by the CIED (cardiac implantable electronic device); and 2) pulmonary edema, should any occur ante-mortem (before death). When viewed from that perspective, her opinions can and should be soundly rejected.

27. Patients with abnormal cardiac rhythms (such as atrial fibrillation or atrial flutter) often require cardioversion (delivery of electrical shocks). Anesthesia is used for these procedures, and often several shocks are required to convert the heart rhythm (Gallagher et al., 2001; Karthikeyan et al., 2002). Patients tolerate cardioversion without difficulty. If patients were suffering from this procedure, then it would have been abandoned, or at the

very least, restricted in its use. The amount of anesthesia administered for cardioversion is much less than the doses of pentobarbital used in executions. And thiopental (which, as both Dr. Van Norman and I agree, is equivalent to pentobarbital) has been used successfully for anesthesia for cardioversion (Gupta et al., 1990). So, it makes no sense that an inmate who receives five grams of pentobarbital would experience the shock from a CIED when many patients undergo cardioversion daily without difficulty.[7]

28. Regarding claims that the presence of the CIED would prolong the dying process, while the CIED might deliver shocks in a futile attempt to restore cardiac function, for all intents and purposes, the inmate would be clinically dead. Usually, and according to normal medical practice, a person is not declared dead until the physician has examined the patient for signs of life (heart sounds, pulses, breathing, etc.). Thus, even if a person has lost all cardiac function for five minutes, ten minutes or even much longer prior to the determination of death, the time of death will be when the physician determines from the physical examination that no signs of life are present. While it might appear that death has been prolonged, this does not mean that the person (inmate) was clinically alive immediately before the physical examination and declaration of the time of death. It is important to

7 I have personally been cardioverted twice because of atrial fibrillation and I did not experience any pain or suffering. Obviously, I received non-lethal doses of anesthesia for these procedures.

remember that during this time when there is no cardiac output and blood flow, the brain is not getting blood and oxygen (which of course by itself would cause unconsciousness) and the massive amount of pentobarbital that has entered the brain remains there because there is no blood flow to remove it.

29. I agree that electrical shocks delivered by the CIED could result in some muscle movement, but this type of movement is reflexive in nature. And while these shocks can be alarming and painful in the conscious patient, as mentioned above, the combination of lethal doses of pentobarbital, no cardiac function (with no blood flow to the brain) and low oxygen levels guarantee that the inmate will not perceive these shocks. Furthermore, I would expect pentobarbital to lessen the amount of movement that occurs with these shocks, because the spinal reflexes that subserve the movements would be profoundly depressed by pentobarbital. After all, pentobarbital is an anesthetic capable of depressing (profoundly so) movement responses to stimuli, including noxious stimuli.

30. Nothing in this report should be construed as endorsement or approval, or disapproval, of the Tennessee protocol or providing instructions, advice or assistance to the State of Tennessee or any other entity to carry out a legal judicial execution. Furthermore, I do not express any opinions on whether the

protocol would result in a humane or inhumane death, as that is for others to decide.

Conclusion

31. 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 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; 4) the presence of a CIED will not increase the risk of pain and suffering of the inmate.

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

Date: July 14, 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

Gallagher MM, et al. Initial energy settin, outcome and efficiency in direct current cardioversion of atrial fibrillation and flutter. J Am Coll Cardiol 2001; 38:1498-504

Gupta A, et al. Anaesthesia for cardioversion a comparison between propofol, thiopentone and midazolam. Anaesthesia 1990; 45:872-75

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

Karthikeyan S, et al. Anaesthesia for cardioversion: a comparison of sevlflurane and propofol*. Anaesthesia 2002; 57:1114-19

Lafferty KA. Barbiturate Toxicity.

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

(accessed 7-4-2025)

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

Pentobarbital package insert (accessed 7-4-2025):

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
7/25-present	Principal Investigator, Sub-Investigator Kinetic Clinical Research, LLC[8] West Covina, CA
1/22-7/25	Principal Investigator Next Level Clinical Trials, LLC West Covina, CA
1/22-7/25	Sub-Investigator SmartCures Clinical Research, LLC Anaheim, CA

8 Not a change in employment, but reflects the legal merger of the three clinics for which I work.

7/22-7/25	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 Medicine[9] (with tenure) Department of Anesthesiology and Pain Medicine

9 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 = 41 as of 7-9-25, 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—School of Medicine
12/02-7/11	Professor of Neurobiology, Physiology and Behavior (with tenure; WOS appointment) College of Biological Sciences 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.