CRITICAL CARE ALERT®

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INSIDE

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A Short Written Message: Improved Satisfaction After Withdrawal of Life Support

ABSTRACT & COMMENTARY

By Leslie A. Hoffman, RN, PHD

Department of Acute/Tertiary Care School of Nursing, University of Pittsburgh

Dr. Hoffman reports no financial relationship to this field of study.

Synopsis: *Families who received written information about withdrawal of life support felt more prepared and were more satisfied when contacted 2-4 weeks later.*

Source: Kirchhoff KT, et al. Preparing families of intensive care patients for withdrawal of life support: A pilot study. *Am J Crit Care* 2008;17:113-121.

THE NEED TO PREPARE FAMILIES FOR WITHDRAWAL OF LIFE SUPPORT is well known. However, the specific information needed to accomplish this goal is not well defined. In this study, 22 participants were randomized to receive a brochure with information about what to expect during withdrawal of life support, or to usual care without receiving the brochure. The brochure was provided in one of four versions. Each version of the brochure had three parts. An initial generic part included information about the uncertainty of the time interval until death, assurance that comfort measures would continue, and general information about what to expect in regard to physiological changes, e.g., breathing, skin color, and movement. The second part included additional information about anticipated physiological changes with different text depending on whether the patient would remain intubated and the anticipated duration of time before death. A final generic part provided information on actions family members could choose to take, e.g., touching, talking, and remaining with the patient.

When contacted by telephone 2-4 weeks later, families in the intervention group expressed greater satisfaction with the information they were provided ($P < 0.05$) and their understanding of how death was likely to occur ($P = 0.03$). The usual care group reported having to ask for information, not being prepared, and not understanding that time to death can differ among patients.

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COMMENTARY

ICU clinicians recognize the need to prepare families for the withdrawal of life support. A variety of studies have identified that this information should address how treatments will be withdrawn, that comfort will be ensured, how long the patient is expected to survive, and how the team will continue to provide care. Often, such information is provided in a family conference with follow-up at the bedside.

The present study had three unique features. First, information was provided in writing, as well as verbally. During this emotional experience, it is easy to forget details about the information shared. Having information available in a pamphlet appeared to provide additional support. Second, the information provided was specific with regard to what might be expected including changes in breathing, skin color, etc., and individualized as to whether patients were intubated during the process. Third, the information gave permission to show emotion, talk to, and touch the patient. Rather than being emotion-provoking, the detail proved comforting based on questionnaire responses and additional unsolicited comments.

The distress surrounding such events can be profound. Siegel and colleagues interviewed the next of kin of 51 patients who died in an ICU in a university teaching hospital, and reported that 34% of these individuals met criteria for at least one psychiatric illness, including a major depressive disorder (27%), anxiety disorder (10%), panic disorder (10%), or complicated grief disorder (5%).[1] Most participants considered the health care team to be compassionate and clear in their explanations, suggesting that communication occurred commonly and was well received. The additional step of providing a brochure, tailored to circumstances expected during withdrawal of life support, is a simple but important means of potentially decreasing grief and depression after the event. ■

Reference

1. Siegel MD, et al. Psychiatric illness in the next of kin of patients who die in the intensive care unit. *Crit Care Med* 2008;36:1722-1728.

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Noninvasive Ventilation in Acute Cardiogenic Pulmonary Edema

ABSTRACT & COMMENTARY

By Dean Hess, PhD, RRT

Respiratory Care, Massachusetts General Hospital, Department of Anesthesiology, Harvard Medical School, Boston

Dr. Hess reports no financial relationship to this field of study.

Synopsis: *In patients with acute cardiogenic pulmonary edema, CPAP or NIV produces a more rapid improvement in respiratory distress and arterial blood gases than standard therapy.*

Source: Gray A et al. Noninvasive ventilation in acute cardiogenic pulmonary edema. *N Engl J Med* 2008; 359:142-151.

NONINVASIVE VENTILATION (NIV) AND CONTINUOUS positive airway pressure (CPAP) have been shown to be of benefit in the treatment of patients with acute cardiogenic pulmonary edema (CPE). The authors conducted this study to determine whether NIV or CPAP reduces mortality in this patient population. This was a multicenter, open, prospective, randomized controlled trial. Patients were assigned to standard oxygen therapy, CPAP (5-15 cm H_2O), or NIV (inspiratory pressure, 8-20 cm H_2O; expiratory pressure, 4-10 cm H_2O). The primary endpoint was death within 7 days after the initiation of treatment.

Of 1069 patients enrolled, 367 were assigned to standard oxygen therapy, 346 were assigned to CPAP, and 356 were assigned to NIV. There was no significant difference in 7-day mortality between patients receiving oxygen therapy (9.8%) and those receiving NIV (9.5%, $P = 0.87$). There was no significant difference in the combined endpoint of death or intubation within 7 days between the patients who were assigned to NIV (11.1%) and those assigned to CPAP (11.7%, $P = 0.81$). Compared to oxygen therapy, NIV was associated with greater improvements at 1 hour after the beginning of treatment in patient-reported dyspnea ($P = 0.008$), heart rate ($P = 0.004$), pH ($P < 0.001$), and hypercapnia ($P < 0.001$). There were no treatment-related adverse events. The authors concluded that, in patients with acute CPE, NIV induces a more rapid improvement in respiratory distress and metabolic disturbance than standard oxygen therapy, but has no effect on short-term mortality.

COMMENTARY

Over the past 10 years, there has been increasing use of NIV in patients with acute respiratory failure. Many randomized controlled trials have been published and these have been combined into several meta-analyses. In fact, meta-analyses have reported benefit for NIV and CPAP for acute CPE, concluding that this therapy reduces the risk of intubation.

The first meta-analysis was by Pang et al in 1998.[1] They found that CPAP (3 studies) was associated with a decrease in the need for intubation (risk difference, -26%; 95% confidence intervals [CI], -13% to -38%) and a trend for a decrease in hospital mortality (risk difference, -6.6%; 95% CI, +3% to -16%) compared with standard therapy alone. In this meta-analysis, there was insufficient evidence to comment on the effectiveness of NIV compared with either standard therapy or CPAP and standard therapy.

The second meta-analysis was published in 2005 by Masip et al.[2] In the combined analysis of CPAP and NIV, there was a significantly reduced mortality by nearly 45% compared with conventional therapy (RR, 0.55; 95% CI, 0.40-0.78; $P < 0.001$). Although mortality was reduced for CPAP (9 studies; RR, 0.53; 95% CI, 0.35-0.81; $P = 0.003$), this was not the case for NIV (6 studies; RR, 0.60; 95% CI, 0.34-1.05; $P = 0.07$). Both CPAP and NIV showed a significant decrease in intubation compared with conventional therapy; CPAP (RR, 0.40; 95% CI, 0.27-0.58; $P < 0.001$), NIV (RR, 0.48; 95% CI, 0.30-0.76; $P < 0.001$), and together (RR, 0.43; 95% CI, 0.32-0.57; $P < 0.001$).

A third meta-analysis by Peter reported that CPAP was associated with a significantly lower mortality rate than standard therapy (12 studies; RR, 0.59; 95% CI, 0.38-0.90; $P = 0.015$).[3] There was a nonsignificant difference in mortality for the comparison between NIV and standard therapy (7 studies; RR, 0.63; 95% CI, 0.37-1.10; $P = 0.11$). The need for intubation was reduced with CPAP (RR, 0.44; 95% CI, 0.29-0.66; $P = 0.0003$) and with NIV (RR, 0.50; 95% CI, 0.27-0.90; $P = 0.02$), compared with standard therapy.

Finally, a meta-analysis by Winck et al showed a 22% absolute risk reduction (ARR) in intubation (95% CI, -34% to -10%; $P < 0.001$) and 13% in mortality (95% CI, -22% to -5%; $P = 0.003$) for CPAP compared to standard therapy (10 studies).[4] For 6 studies of NIV compared to standard medical treatment, there was an 18% ARR in intubation (95% CI, -32% to -4%; $P = 0.01$) and 7% in mortality (95% CI, -14% to 0%; $P = 0.06$).

Meta-analyses have also compared CPAP with NIV. Masip et al reported no differences in intubation or mortality rates in the analysis of studies comparing the 2 techniques.[2] Comparing NIV to CPAP, Peter et al found no difference in mortality risk ($P = 0.38$) or intubation rate ($P = 0.86$).[3] Winck et al found a nonsignificant 3% ARR in intubation (95% CI -4% to 9%) and 2% in mortality (95% CI, -6% to 10%) with NIV compared to CPAP.[4] Ho et al conducted a meta-analysis specifically comparing NIV and CPAP.[5] From 7 studies, intubation (RR, 0.80; 95% CI, 0.33-1.94; $P = 0.62$) and hospital mortality (RR, 0.76; 95% CI, 0.32-1.78; $P = 0.52$) were similar between patients treated with CPAP and those treated with NIV.

Following the study by Mehta et al,[6] there was concern about the safety of NIV, specifically for new myocardial infarction, in patients with acute cardiogenic pulmonary edema. However, this has not emerged as a significant concern in subsequent studies or meta-analyses.

How should this new study by Gray et al be viewed in the context of these meta-analyses? First, the intubation rate was extremely low in the Gray study: 0.8% in the standard therapy group, 0.3% in the CPAP group, and 1.1% in the NIV group. It's hard to imagine how any therapy can improve on the need for intubation when the overall intubation rate of the enrolled patients is so low. This suggests that the patients in this study were less acutely ill than those in previous studies included in the meta-analyses. The absence of a mortality benefit in this study is consistent with the meta-analyses described above, most of which failed to report a mortality benefit for NIV. This may be related to the relatively low mortality in this patient population. In the Masip meta-analysis,[2] for example, the overall mortality

was 15%. A quick power analysis reveals that a sample size of more than 1500 patients (about 700 in each group) is required to detect a reduction in mortality from 15% to 10% with a power of 0.8. So it should be no surprise that a meta-analysis, let alone a single study, is unlikely to show a mortality benefit in this patient population.

Given the results of prior studies, many of us have adopted the use of NIV or CPAP as first-line therapy in patients presenting with acute CPE. This is supported not only by the results of randomized controlled trials, but also by a sound underlying physiology. Increasing the intrathoracic pressure with NIV or CPAP reduces preload and afterload, which supports the failing heart while definitive therapies are administered. This should translate into improvements in heart rate, respiratory rate, dyspnea, and arterial blood gases. In fact, these benefits were reported in the study by Gray et al.

The study by Gray et al should not change the recommendation for CPAP of NIV as a first-line therapy in patients presenting with acute CPE. Similar outcomes are likely with CPAP or NIV; this is probably more of an academic than a practical point, given that the same equipment is used for either therapy in modern practice. CPAP or NIV produces a more rapid improvement in respiratory distress than standard therapy alone. CPAP and NIV reduce the need for intubation in patients sick enough to be at risk for intubation, which is supported by several meta-analyses. ■

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Special Feature

Critical Care Management of the Patient with Subarachnoid Hemorrhage

By Andrew M. Luks, MD

Pulmonary and Critical Care Medicine, University of Washington, Seattle

Dr. Luks reports no financial relationship to this field of study.

SPONTANEOUS SUBARACHNOID HEMORRHAGE (SAH) IS associated with high morbidity and mortality. Although a significant proportion of the deaths occur at the time of or shortly following the initial hemorrhage—10% of patients die before hospitalization while 25% die within the first 48 hours[1]—getting through this initial period of illness does not ensure good patient outcomes: Patients remain at risk for complications and severe neurologic insults for up to two weeks following the initial bleeding event. As a result, the ultimate outcome of those who survive the initial hemorrhage depends to a large extent on the quality of critical care they receive following admission.

Management of SAH patients has traditionally fallen within the purview of the neurosurgeons and neurointensive care specialists, but with increasing efforts to assure intensivist involvement in the management of all ICU patients, it is expected that more general intensivists will be caring for these patients. The purpose of this special feature is to review the critical care management of patients with SAH from the general intensivist's perspective. The review will begin by examining those issues that arise before the aneurysm has been secured and then consider issues that arise later in the course of illness when the risk of non-bleeding complications remains high.

Critical Care Issues Prior to Securing the Aneurysm

Once a patient presents with spontaneous SAH, nothing can be done to reverse the damage of the initial bleeding event. It is imperative, however, to prevent further bleeding episodes, which are a major source of morbidity and mortality.[1] Toward this end, the majority of patients undergo a definitive procedure to "secure" the aneurysm. A full discussion of the optimal method (surgical vs endovascular) for this procedure is beyond the scope of this review, but it is important that intensivists at facilities lacking neurosurgical services

arrange early transport to neurosurgical centers in order that the definitive procedure can occur with the now-preferred 72-hour window.

Until the aneurysm is secured, the rebleeding risk can be reduced through adequate blood pressure control. Although data are lacking to support particular blood pressure targets, efforts should be made to bring systolic blood pressures below 140 mm Hg. Nicardipine drips are commonly used for this purpose and recent evidence suggests that use of this agent is associated with less blood pressure variability and fewer dose adjustments when compared to intravenous labetalol in patients with various forms of stroke including SAH.[2] In the course of lowering blood pressure, however, care must be taken to avoid provoking hypotension, as cerebrovascular autoregulation is lost following SAH and any hypotensive episodes could provoke cerebral ischemia.

Rebleeding risk can also be reduced by reversing any preexisting coagulopathy, although the routine use of antifibrinolytic therapy (e.g., aminocaproic acid) should be avoided because the positive effect on rebleeding rates is offset by an increased incidence of cerebral ischemia.[3]

During this early period of SAH, patients are also at risk for acute hydrocephalus and its resultant complications. As a result, ventriculostomy is indicated in those patients with intraventricular blood, poor aneurysm grade, or in intubated patients in whom neurologic exam is not possible. Concern has been raised that external ventricular drains increase the rebleeding risk but methodological issues limit the data underlying these claims[4] and a more recent study suggests they may have no impact on rebleeding.[5] Because external ventricular drains increase the risk of meningitis and/or ventriculitis, strict infection control practices should be implemented when these devices are used.[6]

Once the Aneurysm Has Been Secured

The risk of rebleeding declines dramatically once the aneurysm has been secured, but patients remain at risk for a variety of other complications that adversely affect outcomes and, in some cases, lead to severe neurologic deterioration.

Vasospasm

In addition to rebleeding and acute hydrocephalus, cerebral vasospasm is another leading cause of morbidity and mortality in those who survive the initial hemorrhage, and extensive effort should be directed toward identifying and treating this complication, which may occur anywhere from 3 to 14 days following the initial hemorrhage. Although angiography remains the gold standard for the detection of cerebral vasospasm, the cost and invasiveness of the procedure limit its widespread application in vasospasm monitoring. Instead, clinicians must rely on a combination of the clinical exam—the onset of vasospasm is often heralded by a change in the patient's level of consciousness—and a variety of imaging modalities including serial transcranial Doppler ultrasonography and SPECT scanning to identify when more invasive testing (angiography) or therapeutic intervention is necessary.

All patients should be placed on prophylaxis with nimodipine (60 mg every 4 hours for 21 days), the lone strategy in vasospasm management that has Level 1 evidence demonstrating a beneficial effect on patient outcomes.[7] Many institutions also rely on "Triple-H" therapy (hypertension, hypervolemia, hemodilution) to prevent and treat vasospasm, but a meta-analysis of the four prospective trials that investigated this strategy revealed no evidence of benefit in this regard[8]; the strategy may also cause further complications such as pulmonary edema from volume overload. While some data suggest that the hypertension component of Triple-H therapy may be of benefit when used alone,[9] at a minimum, efforts should be made to avoid volume depletion and hypotension in the two weeks following SAH. To this end, diuretic use should be avoided.

In situations where vasospasm does occur, blood pressure and central venous pressure should be raised through the use of vasopressors and intravenous fluids, although prospective, randomized trials have not confirmed that this improves outcomes relative to normovolemia and normotension. Patients with angiographically confirmed vasospasm may also undergo a variety of interventions, such as balloon angioplasty or vasodilator administration; a discussion of these issues is beyond the scope of this review.

Other Critical Care Issues

Blood Transfusions

It has been argued that SAH is the "heart attack of the brain," and that transfusions should be employed as they would be in patients with acute coronary syndrome, to maintain the hematocrit above 30%. While anemia (Hb < 10 g/dL) is, in fact, associated with adverse outcomes in SAH,[10] red blood cell transfusion is also associated with adverse outcomes including death and severe disability.[11] The link between transfusion and adverse outcomes may be strongest in those who have no evidence of vasospasm, suggesting that we should tailor transfusion practices based on the clinical characteristics of the patients, although such practices have not been tested in a prospective manner. Until the

results of such studies are available, care should be taken to avoid unnecessary transfusions, and arbitrary transfusion thresholds should not be rigidly applied to all patients.

Glucose Control

Multiple studies have established that hyperglycemia is associated with adverse outcomes following SAH, including mortality, vasospasm, and poor neurologic status.[10,12] Although studies have suggested a benefit from intensive insulin therapy (IIT) in other post-surgical patient populations,[13] there are, as yet, no data demonstrating that IIT improves outcomes in SAH patients.[14] In light of data suggesting possible harm from IIT in critically ill patients that may be related to insulin itself or an increased incidence of hypoglycemia,[15] care should be taken to avoid implementing protocols with overly strict glucose thresholds until further data become available.

Fever

Temperature > 38.3° C occurs in up to 41% of SAH patients, and is associated with both increased risk of vasospasm and poor neurologic outcomes.[16] In up to 25% of cases, no infectious source can be identified and the fever is presumed to be central in origin.[16] Efforts should be made to identify all reversible infectious and non-infectious causes of fever, including periodic sampling of cerebrospinal fluid in patients with external ventricular drains, limiting the use of invasive catheters when not necessary (e.g., avoidance of central lines in patients not requiring vasopressors or 3% NS), and maintenance of vigorous infection control practices during repeated handling of devices such as external ventricular drains. Given the adverse impact on patient outcomes, it makes sense that treating fevers aggressively would have a beneficial impact. However, while studies have shown that various medicines (acetaminophen, ibuprofen) and cooling systems are capable of reducing fever in SAH patients, large, controlled trials have yet to demonstrate a positive impact on patient outcomes.

Sodium Balance

Hyponatremia occurs in up to 40% of patients with SAH. It is most commonly due to cerebral salt wasting syndrome (CSWS), but in some cases, results from inappropriate secretion of antidiuretic hormone (SIADH). Distinguishing between the two causes is important, as different therapies are required in each case; SIADH is managed with fluid restriction, while CSWS is managed with sodium administration in the form of saline or salt tablets. The disorders share several common laboratory findings including elevated urine osmolality and urine sodium and low serum uric acid concentrations but can be distinguished by the fact that CSWS patients are typically volume-depleted with normal or elevated serum osmolality while SIADH patients are either euvolemic or slightly hypervolemic and have low serum osmolality. Given the increased risk of diabetes insipidus and the fact that many patients also receive 3% normal saline or mannitol during their treatment, patients should also be monitored for hypernatremia, which has been associated with increased risk of cardiac complications and death following SAH.[17]

Cardiopulmonary Complications

A detailed discussion of this extensive topic is beyond the scope of this review, but the general intensivist should be aware that SAH patients are at increased risk for cardiac complications including the development of reversible myocardial stunning. Electrocardiograms should be obtained in all patients. Abnormalities such as symmetrical T-wave inversions and ST changes can be seen and should be followed up with serial troponin levels. Those patients with elevated troponins or persistent hypotension should be evaluated with echocardiography. Because left ventricular dysfunction and impaired cardiac output increase the risk of vasospasm and cerebral ischemia, any patients with evidence of cardiac dysfunction may require more intensive monitoring (e.g., arterial line and pulmonary artery catheter) and the administration of dobutamine to ensure adequate cardiac output and maintain cerebral perfusion.

Pulmonary complications such as neurogenic or cardiogenic pulmonary edema, aspiration pneumonia, and volume overload from Triple-H therapy are also common in SAH. In addition, up to 27% of SAH patients develop acute lung injury, an outcome associated with increased mortality and ICU length of stay.[18] While patients should receive standard treatment for these problems, a few issues warrant additional attention. Although there is theoretical concern that high levels of positive end-expiratory pressure (PEEP) may decrease cerebral venous return and lead to elevated intracranial pressure, there is evidence to suggest that both PEEP levels of 15 cm H_2O and recruitment maneuvers can be applied safely in these patients.[19,20] Strong consideration should be given to monitoring with jugular venous oximetry, brain tissue PO_2, and/or intracranial pressure in patients with severe hypoxemia requiring higher levels of PEEP. Low tidal volume ventilation in acute lung injury may lead to hypercarbia and subsequent increases in cerebral blood flow, which may exacerbate intracranial pressure problems. At the same

time, routine hyperventilation is no longer used in this patient population, as the associated hypocarbia decreases cerebral perfusion and may provoke ischemia.

Nutrition

Depending on the severity of the SAH, patients may develop catabolic responses marked by the presence of markedly elevated resting energy expenditures and negative nitrogen balance.[21] As a result, it is important to begin early enteral nutrition where feasible with careful attention to preventing aspiration in those who may have impaired mental status and swallowing abilities.

Conclusion

In considering the issues described above, it is important to recognize that solid Level 1 evidence is lacking for many of the management strategies used with SAH patients. As a result, there can be a considerable degree of inter-individual and inter-institutional variation in the care of these patients. Although the proper means for dealing with the various complications may not be clear, the complications and their risk factors are well established and careful attention to these details can go a long way toward assuring better outcomes for patients with this potentially devastating disorder. ■

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CME / CE Questions

27. Providing a brochure that described patient response during life support withdrawal:
a. increased anxiety for next of kin who were older.
b. had no effect on the majority of participants.
c. led to depressive disorders in 5% of families.
d. eliminated the need for family conferences.
e. improved family satisfaction following the event.

28. Which of the following is correct related to the use of CPAP and NIV in patients presenting with acute cardiogenic pulmonary edema?
a. NIV is associated with a more rapid improvement in respiratory distress than standard therapy.
b. Meta-analyses report reduced intubation with the use of NIV compared to standard therapy.
c. Clinical outcomes for CPAP and NIV are comparable.
d. All of the above

29. Which of the following has been associated with an increased risk of adverse neurologic outcomes following subarachnoid hemorrhage?
a. Anemia
b. Fever
c. Hyperglycemia
d. Hypernatremia
e. All of the above

30. Which of the following variables can be used to distinguish cerebral salt wasting syndrome from SIADH?
a. Urine osmolality
b. Urine sodium
c. Serum uric acid concentrations
d. Volume status
e. Serum sodium

Answers: 27. (e); 28. (d); 29. (e), 30. (d).

CME / CE Objectives

After reading each issue of *Critical Care Alert*, readers will be able to do the following:

- Identify the particular clinical, legal, or scientific issues related to critical care.
- Describe how those issues affect nurses, health care workers, hospitals, or the health care industry in general.
- Cite solutions to the problems associated with those issues.

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7. Complete Mailing Address of Known Office of Publication *(Not Printer) (Street, city, county, state, and ZIP+4)*
3525 Piedmont Road, Bldg. 6, Ste. 400, Atlanta, Fulton County, GA 30305
Contact Person: Robin Salet
Telephone: 404/262-5489

8. Complete Mailing Address of Headquarters or General Business Office of Publisher *(Not Printer)*
AHC Media LLC, 3525 Piedmont Road, Bldg. 6, Ste. 400, Atlanta, GA 30305

9. Full Names and Complete Mailing Addresses of Publisher, Editor, and Managing Editor *(Do Not Leave Blank)*
Publisher *(Name and Complete Mailing Address)*
Robert Mate, President and CEO
AHC Media LLC, 3525 Piedmont Road, Bldg. 6, Ste. 400, Atlanta, GA 30305
Editor *(Name and Complete Mailing Address)*
Paula Cousins, same as above
Managing Editor *(Name and Complete Mailing Address)*
Coles McKagen same as above

10. Owner *(Do not leave blank. If the publication is owned by a corporation, give the name and address of the corporation immediately followed by the names and addresses of all stockholders owning or holding 1 percent or more of the total amount of stock. If not owned by a corporation, give the names and addresses of the individual owners. If owned by a partnership or other unincorporated firm, give its name and address as well as those of each individual. If the publication is published by a nonprofit organization, give its name and address.)*

Full Name	Complete Mailing Address
AHC Media LLC	3525 Piedmont Road, Bldg. 6, Ste 400 Atlanta, GA 30305

11. Known Bondholders, Mortgagees, and Other Security Holders Owning or Holding 1 Percent or More of Total Amount of Bonds, Mortgages, or Other Securities. If none, check box → ☐ None

Full Name	Complete Mailing Address
Thompson Publishing Group Inc.	805 15th Street, NW, 3rd Floor Washington, D.C. 20005

12. Tax Status *(For completion by nonprofit organizations authorized to mail at nonprofit rates.) (Check one)*
The purpose, function, and nonprofit status of this organization and the exempt status for federal income tax purposes:
☐ Has Not Changed During Preceding 12 Months
☐ Has Changed During Preceding 12 Months *(Publisher must submit explanation of change with this statement)*

PS Form **3526**, September 1998 *See instructions on Reverse)*

13. Publication Name: Critical Care Alert
14. Issue Date for Circulation Data Below: September 2008

15. Extent and Nature of Circulation			Average No. of Copies Each Issue During Preceding 12 Months	Actual No. Copies of Single Issue Published Nearest to Filing Date
a. Total No. Copies *(Net Press Run)*			642	498
b. Paid and/or Requested Circulation	(1)	Paid/Requested Outside-County Mail Subscriptions Stated on Form 3541. *(Include advertiser's proof and exchange copies)*	380	303
	(2)	Paid In-County Subscriptions *(Include advertiser's proof and exchange copies)*	2	0
	(3)	Sales Through Dealers and Carriers, Street Vendors, Counter Sales, and Other Non-USPS Paid Distribution	17	19
	(4)	Other Classes Mailed Through the USPS	11	21
c. Total Paid and/or Requested Circulation *(Sum of 15b(1) and 15b(2)*			410	343
d. Free Distribution by Mail *(Samples, Complimentary and Other Free)*	(1)	Outside-County as Stated on Form 3541	16	19
	(2)	In-County as Stated on Form 3541	1	0
	(3)	Other Classes Mailed Through the USPS	0	0
e. Free Distribution Outside the Mail *(Carriers or Other Means)*			20	20
f. Total Free Distribution *(Sum of 15d and 15e)*			37	39
g. Total Distribution *(Sum of 15c and 15f)*			447	382
h. Copies Not Distributed			195	116
i. Total *(Sum of 15g, and h.*			642	498
Percent Paid and/or Requested Circulation *(15c divided by 15g times 100)*			92%	90%

16. Publication of Statement of Ownership
Publication required. Will be printed in the November 2008 issue of this publication. ☐ Publication not required

17. Signature and Title of Editor, Publisher, Business Manager, or Owner: President and CEO — Date: 9/28/08

I certify that all information furnished on this form is true and complete. I understand that anyone who furnishes false or misleading information on this form or who omits material or information requested on the form may be subject to criminal sanctions (including fines and imprisonment) and/or civil sanctions (including multiple damages and civil penalties).

Instructions to Publishers

1. Complete and file one copy of this form with your postmaster annually on or before October 1. Keep a copy of the completed form for your records.
2. In cases where the stockholder or security holder is a trustee, include in items 10 and 11 the name of the person or corporation for whom the trustee is acting. Also include the names and addresses of individuals who are stockholders who own or hold 1 percent or more of the total amount of bonds, mortgages, or other securities of the publishing corporation. In item 11, if none, check the box. Use blank sheets if more space is required.
3. Be sure to furnish all circulation information called for in item 15. Free circulation must be shown in items 15d, e, and f.
4. Item 15h., Copies not Distributed, must include (1) newsstand copies originally stated on Form 3541, and returned to the publisher, (2) estimated returns from news agents, and (3), copies for office use, leftovers, spoiled, and all other copies not distributed.
5. If the publication had Periodicals authorization as a general or requester publication, this Statement of Ownership, Management, and Circulation must be published; it must be printed in any issue in October or if the publication is not published during October, the first issue printed after October.
5. In item 16, indicate date of the issue in which this Statement of Ownership will be published.
6. Item 17 must be signed.

Failure to file or publish a statement of ownership may lead to suspension of second-class authorization.

PS Form **3526**, September 1999 *(Reverse)*