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CACCN IFC
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If you build it, he will come.

For many of us, the past few years have been particularly challenging, both on a personal and professional level. The changing tides of health care reform and fiscal restraint have affected our abilities to practice, to participate, and to perform.

We should not lose sight of the fact that health care reform and the changes implemented because of government cutbacks are two very different things, often and wrongly used interchangeably. Our health care system needs to move towards a primary health care/health promotion model and away from its current illness-centered approach. This does not mean that tertiary care is less important to our society and the health of our nation, only that more work is warranted to emphasize and address the issues for health care within our communities.

As professionals, we strive to practise to the highest standards, to give our patients the best care, and to continually ensure that our work reflects an integration of current knowledge, critical analysis, and expertise. In many cases, this has been impossible. As hospitals have been directed to implement budget cuts, their approach has generally been to try to accomplish the work with fewer and/or less-skilled people. As a result, we have all borne witness to the tensions and stress in our clinical settings as our colleagues struggle to deliver high quality care in the midst of declining resources. And we have all grown weary in the process.

Nursing has suffered and been “casualized”. Now, that may not be a real word, but I use it to try to express the demoralization and deterioration of our sense of collegiality, optimism, and unity. More nurses are working in part-time and casual positions, sometimes in two or three institutions to secure an adequate income. It means that the nurse may not have health care and disability benefits, perhaps no pension contribution. It may mean that the nurse becomes more focussed on simply securing financial remuneration than on attending to professional growth and development needs. It likely means that this nurse is not considered first when the institution offers educational and career opportunities, simply because of employment status. The nurse centers on issues of personal security rather than professional growth.

Yet, enthusiasm is out there. For critical care nursing professionals, perhaps there is no greater example than this association. This year as in previous years, at our annual Dynamics conference, I had the opportunity to speak with colleagues from across our country. We explored issues related to the quality of work life, effects on patient care, the hardships faced by individual nurses and the profession as a...
whole. The difficulties seem overwhelming, but there are similar concerns and remarkably common ground. There seems to be a great sense that we must have a collective means and process from which we can act as unified professionals, both locally and nationally. Members from coast to coast agree that CACCN is an important mechanism to address issues of concern, develop and synthesize important messages for dissemination to appropriate organizations and representatives, and enable opportunities for professional growth. There is tremendous commitment evident from our members and chapters. These nurses are facing the same issues within their work environment that render them as mentally and physically tired as any of us. Yet, many of them are finding it within themselves to invest of their time and energies to examine methods to address their concerns, and to inspire their colleagues within the context of the mission and vision of CACCN.

There is a strong belief that together we can affect greater change and that together, in an organized, strategic manner, we must work towards this goal. We must work to advance our practice and share our expertise to benefit our patients. There is a professional responsibility to maintain current in our understanding of our specialty, to continually educate ourselves and to assist in raising the standards of our practice as evidenced in the outcomes of our patients.

We have a staggering wealth of knowledge and we need to demonstrate this to decision-makers within our institutions and throughout our government hierarchies. Participating at an individual, unit, organizational, local, provincial or national level is essential to promote our issues and establish our voice as credible, valuable and worth being heard.

CACCN is working with members from across Canada to continue involving ourselves in endeavours that will assist us, individually and collectively, to establish our position(s) and develop the appropriate documents to communicate our message effectively. Together we are engaging in the initiatives that will enable us to respond effectively to issues and concerns of relevance to our patients, our practice, and our profession.

This work contributes to our ability to move beyond existence to performance. It allows us to form a powerful profile, to gather momentum, and invite partnerships which will strengthen our voices and ensure that our efforts have impact, purpose, and meaning. All of our efforts are working to create and maintain a vital, thriving, and driven organization that is seen as having an important and essential perspective. In doing so, we attract members, potential members, interdisciplinary colleagues, interested supporters and the attentions of the public, administrators, and government decision-makers. Together we continue to build a strong association and move forward to affect the changes in the best interests of health care in Canada.

Colleen Shelton, RN, BAA, CNCC(C)
President, CACCN

Letter from the clinical editor

I am writing these editorial comments having just returned from Dynamics ‘97 - and dynamic it was! Dynamics ‘97 was an outstanding conference. I was delighted to meet so many of my critical care nursing colleagues from across Canada. I was honoured to be able to listen to some very exciting presentations of what nurses are doing across the country. I was inspired by the wonderful plenary and keynote speakers. Captain Gerald Coffee (US Navy, Retired) grabbed the audience’s attention and never let it go during his two presentations. He used his experiences as a POW in the Vietnam war to establish a context for his message. His message was both simple and complex; he survived his horrendous experience through faith - faith in himself, faith in his colleagues, faith in his country, and faith in his God. Dr. Kenneth Shonk had the audience rolling in the aisles when he spoke of humour in health care. Dr. Jim Young gave us a strong message during his presentation of forensic pathology. The final speaker, Sharon Wood, spoke eloquently about reaching the summit of Mount Everest (the first North American woman to do so). She shared with the audience all of the obstacles she faced to meet that courageous goal; the greatest obstacle of all being herself. We can draw on so many of these messages during our times of struggle both professionally and personally. I came away rejuvenated and inspired.

There were many exciting moments at Dynamics ’97. My congratulations go to Cathy Mawdsley and Isobel Davis, Glenda Pack, and Jo Logan for winning the two Johnson and Johnson editorial awards for their articles published in the journal last year. The revised Standards of Critical Care Nursing Practice were released. Special thanks go to 3M for their generous contribution for the publication of these standards. All members of CACCN will be receiving a copy in the near future. The second edition of the Study Guide for Critical Care Nursing Certification was also released. Special thanks go to Hewlett Packard for their generous contribution toward the publication of this valuable study guide.

The exhibitors at the conference brought many of their exciting new products. Our thanks are extended to Hoffman-La Roche Ltd. for sponsoring the wine and cheese and Hill-Rom for sponsoring the annual dinner.

While at Dynamics I spoke with many people about the journal. What I heard over and over was that people liked the clinical focus. I am pleased to deliver a journal this quarter that is again clinically focused. Leith gives us an article about advance directives which will be very useful to those working in the clinical area. Lethbridge, Shields and Loos give us an interesting case study of a patient with a potentially fatal syndrome called neuroleptic malignant syndrome. This article has a continuing education quiz associated with it for those nurses wanting to earn CE credit hours. Riley and Daines give us another very interesting case study of a patient with systemic necrotizing vasculitis.

My last words in this issue’s editorial column are to urge you to write. I know there are great things being done across the country - I heard about them and read about them in Toronto. Finally, I am already looking forward to Dynamics ’98. It will be held in Calgary October 1, 2, and 3, 1998. I urge you to start planning now for both presenting and/or attending. It will be a time you will not want to miss.

Paula Price, RN, MN, Clinical Editor
Report from Dynamics ’97

While all the evaluations have not been tabulated yet, early reports indicate that Dynamics ‘97 - Make a Difference: Map Your Future, held in Toronto, September 17 to 20, was a huge success.

As in past years, the pre-conference day offered a large number and variety of workshops. This year, the pre-conference day was attended by a record 247 people who could choose from workshops including: Career planning, basic and advanced hemodynamic monitoring, computers in health care - clinical documentation or surfing the net, continuous renal replacement therapy and many more! And this was just the beginning!

From the opening ceremonies of Dynamics ’97 on September 18 to the closing scenes for Dynamics ’98 on the 20th, the 336 delegates were provided with opportunity after opportunity to expand their knowledge and skills, and to have a little fun along the way.

Captain Gerald Coffee’s opening keynote address: Beyond Survival, helped set the stage for the conference as he shared his personal experiences as a prisoner of war who struggled to go beyond survival to discover opportunities for personal growth and achievement.

Through attending keynote, plenary and concurrent sessions; speaking with representatives in the exhibit hall or meeting informally with other conference attendees, the delegates were able to learn from each other, bring new ideas back to their hospitals, or simply re-energize personally and professionally. Plenary sessions, 25 concurrent sessions and 18 poster presentations were provided by colleagues from across Canada and the US, to provide the latest in clinical practice, education, research and administration.

The CNCC(C) reception sponsored by the Canadian Nurses Association was

Congratulations to our certifiees!

The current CACCN members (as of September 30th, 1997) who achieved CNCC(C) credential in 1997 are:

Alberta
- Kathryn Bartlett
- Teresa Craig
- Lucille De Beaudrap
- Paulette Guenette
- Kathleen Leddy
- Holly Lee
- Janet Elinor Ohm
- Karen Schafer
- Lorie Selleck
- Jo-Anne Taylor
- Ruth Topelko

British Columbia
- Maylene Fong
- Gwen Frey
- Brent Hobbs
- Francise Kernachan
- Pamela Locke
- Diane Mainland
- Ina-Mae Meyer

Ontario
- Jackie Adcock
- Tracy Apps
- Sophia Baranik
- Judy Berry
- Barbara Bijnian
- Janine Boston
- Jill Butt
- Heather Camrass
- Maria Cancellara
- Catherine
- Chant-Gambacort
- Christine Cheeseman
- Cynthia Chilton
- Denise Cosani
- Cindy Lee Cosby
- Audrey Coukell
- Joan Craig
- Michelle d’Cambre
- Isobel Davis
- Colleen Dooks
- Mary Ann Doris
- Maryke Durrant
- Catherine Dymock
- Jeanne Elgie-Watson
- Melinda Faldas
- Jill Fediurek
- Trudy Ferguson
- Marie Frederick
- Joyce Gerber
- Claire Gillett
- Laurie Goetz
- Jeanette Guimond
- Nicole Harder
- Katherine Heddon
- Carolyn Hendry
- Tracy Houghton
- Sherry Hubbert
- Linda Hunter
- Christina
- Hurlock-Chorostecki
- Mary Lynn Iacobellis
- Andrea Jackson
- Anna Marie Kawula
- Tammy Kehoe
- Kathryn Kelly
- Trevor Koegler
- Jacqueline Kurt
- Deborah Laing
- Daisy Li
- Janet Maguire
- Karen Maguire-Winter
- Mary Maselli
- Lesley Ann Maw
- Sandra McGillis
- Barbara Metaxas
- Shari Michalewski
- Sandra Moccia
- Janet Mofford-Filion
- Robin Montgomery
- Lynda Newell
- JoAnn Noble
- Carol O’Hearn-Downey
- Valerie Papotto
- Janet Paton
- Linda Pietz
- Elizabeth Pipher
- Natalie Pum
- Lisa Regan
- Donna Regimbal
- Arlene Renn
- Paula Roberts

Prince Edward Island
- Margaret Vloet-Morrison

Saskatchewan
- Lori Garchinski
- Leslie Sparling

In total, there were 239 critical care nurses certified in 1997. Of these, 117 are current CACCN members!
a lovely opportunity to congratulate those CACCN members attending the conference for their achievement in becoming certified in the specialty of critical care nurses. The 239 nurses who became certified this year join those certified in the two previous years for a total of 572 to date! Thank you to Leslie Patry and the CNA for their ongoing support of CACCN.

The wine and cheese, sponsored by Hoffman-La Roche, was held in the exhibit hall on September 18th and was well attended by all delegates. Thanks go to Dave Leitner and Frank Shannon of Hoffman-La Roche for their generous support of this event.

But the fun didn’t stop at the wine and cheese event. What would Dynamics be without the annual dinner? I suppose an even more fitting question would be, what would the annual dinner be without an opportunity to thank Doug MacDonald of Hill-Rom for the generous sponsorship of the dinner? This year’s dinner hosted street performers and included a fire eater, mime, caricaturist, tarot card reader, a juggler and of course, a magician.

**Award winners**

Dynamics is also a time to present awards to some deserving CACCN members. This year the award winners were:

• **Johnson and Johnson Editorial Awards**

  #1. Catherine Mawdsley  
  Study says critical care nurses quick to aid in death of terminally ill: a research critique  
  #2. Isobel Davis, Glenda Pack and Jo Logan  
  Promoting patient sleep: A critical but forgotten practice?

Above left, Catherine Mawdsley winner of the Johnson and Johnson Editorial Award, first prize, with representatives of Johnson and Johnson. Above right Isobel Davis and Glenda Pack were second prize winners of the Johnson and Johnson Editorial Award.

Above: Cecilia St. George-Hyslop and Maria Laslop accept the Johnson and Johnson Innovative Project Award on behalf of the paediatric critical care unit at the Hospital for Sick Children.  
Left: Heather Camrass accepts the COBE Chapter of the Year Award from COBE’s Walter Poprawa on behalf of the Ottawa Region Chapter.
• Johnson and Johnson Innovative Project Award - the Critical Care Unit at the Hospital for Sick Children in Toronto
• COBE Chapter of the Year Award - Ottawa Region Chapter
• SIMS Educational Award - Doreen Ouellett

Congratulations to all these award winners.

Corporate Contribution Program

We also added a new feature to the awards ceremony this year - recognition of those companies who contribute to CACCN, now officially known as the Corporate Contribution Program. It is only through the generous support of our corporate contributors that CACCN is able to pursue ongoing initiatives and projects, and to present a successful Dynamics conference each year. CACCN would like to thank the following companies for their support of the association and their commitment to critical care nursing across Canada:

Corporate Partners are those companies who have generously donated $5,000.00 or more in a one-year period:
- Hill-Rom Canada
- Hewlett-Packard (Canada) Ltd.
- 3M Health Care
- Hoffman-La Roche Canada Ltd.
- Royal York Hotel

Corporate Sponsors are those companies who have generously donated more than $2,000.00 but less than $5,000.00 in a one-year period:
- Baxter Health Care Corporation
- Johnson and Johnson Medical Products

Corporate Members are companies who have generously donated more than $1,000.00 and less than $2,000.00 in a one-year period:
- SIMS Canada
- Spacelabs Medical Products Ltd.
- MDS Ingram and Bell
- Fortune Financial

Corporate Supporters are companies who have generously donated more than $500.00 but less than $1,000.00 in a one-year period:
- COBE Canada Ltd.
- Hospal-Gambro Inc.

Contributions to CACCN

Sandra Matheson and Karen Palmer were recognized for their contributions to CACCN while they sat on the board of directors. Their terms were over in April of this year. Thank you Sandra and Karen for your hard work and dedication to the CACCN.

Closing address

In the blink of an eye, Sharon Wood was presenting the closing keynote presentation: Commitment to Personal Challenge. Sharon’s story of her climb to the summit of Mount Everest illustrated how an ordinary girl can beat the odds, when faced with some of the most challenging situations imaginable. Her presentation on how to achieve true potential through individual strength and teamwork, was a fitting conclusion to an exciting (and exhausting!) few days.

On behalf of the board of directors, I would like to thank the co-chairs, Karen Palmer and Colleen Shelton, and the rest of the task force, Joyce Lee, Marcia Lipsius, Doreen Ouellett and Joy Kramarich for their hard work and commitment in the many months leading up to the conference and during the conference itself. A special thank you also goes out to Heather Reid and her husband Scott who worked tirelessly behind the scenes to ensure that everything went smoothly on stage.

It’s hard to believe that another Dynamics has come and gone. The renewed friendships, the new acquaintances, the countless learning opportunities and the fun all provide memories that will last long after the bills have been paid and the work and sleep caught up on. I imagine for some of us, that will be just in time for Dynamics ’98: Critical Care Reform from a New Perspective. See you all in Calgary October 1st, 2nd and 3rd, 1998!

Helen Edwards
Director, CACCN

Ruth Haydock and Carol McKenny were two of the CACCN members making poster presentations.
Representatives from all chapters met on September 16, in conjunction with Dynamics ’97. This annual event gives all chapter presidents the opportunity to meet face-to-face, compare notes, express frustrations, share strategies, and acknowledge successes.

After introductions, the board members reported on their various activities and responsibilities. New this year is the CACCN annual report. The annual report summarizes the activities of the national board of directors for the past fiscal year. Previously, this information was only available to members attending the annual general meeting. The report will be distributed to all members.

The CACCN web page is constantly expanding. This forum is open to all chapters to post information about activities in their region. Links to research contacts and projects are a new part of the home page.

One of CACCN’s future goals is to provide support for nursing research in critical care. With this in mind, Gwynne MacDonald presented evidence-based health care to the group. Topics covered included question development, the literature review, and how to read an article critically. There was also a demonstration of the availability of research information on the Internet, and how it applies to this process. This information is available from your chapter president and on the web page.

Colleen Shelton explained the planning and organization process necessary for Dynamics each year. The growth of Dynamics has limited the number of potential sites that can be considered in the future. Some ideas concerning future Dynamics were discussed.

Corporate sponsorship is a large part of Dynamics as well as a means to provide bursaries and awards for CACCN members. Helen Edwards reported on the corporate sponsorship program and announced the companies involved. Special mention goes to 3M Canada for sponsoring the printing of the Standards for Critical Care Practice and to Hewlett Packard for sponsoring the printing of the Study Guide for Critical Care Certification. The second edition of the Standards for Critical Care Practice was presented and will be distributed to all CACCN members.

Francis Loos reviewed his involvement with the Official Journal of the Canadian Association of Critical Nurses. He announced changes coming in the future, such as changing the appearance of the cover to be in keeping with the scheme of the “Standards” and “Study Guide”.

This year CACCN has become more politically active with the position paper on “Multiskilled Workers in the ICU”. Direction for future CACCN position papers was taken from the chapter presidents. Suggestions stemmed from local and national issues. The day concluded with a renewed determination to keep critical care nursing on the forefront of health care reform.

Shelley Snider, Membership Chair, Ottawa Region Chapter
Neuroleptic malignant syndrome: A case study

by Linda Lethbridge, RN, Shelley Shields, RN, BScN and Francis Loos, RN, MN, CNCC(C)

Abstract

Neuroleptic malignant syndrome (NMS) is an extreme adverse reaction to neuroleptic drugs (e.g. haloperidol, fluoxetine HCl [Prozac]) which can bring the patient to the critical care unit. Although NMS is rare, it is potentially fatal. It is characterized by hyperthermia, muscle rigidity and tremors, altered consciousness and autonomic dysfunction. This syndrome can occur any time after beginning neuroleptics, but generally occurs a few days after drug initiation or a dosage increase. The incidence is 0.5% to 1% of patients taking neuroleptic drugs, and NMS has a fatality rate of 15 to 30%, even when treated promptly.

The authors use a case study to describe the physiology, pathophysiology, etiology and clinical presentation of NMS. The medical and nursing management of neuroleptic syndrome is described, including the use of dantrolene sodium.

Neuroleptic malignant syndrome (NMS) is an uncommon but lethal disorder which is strongly associated with neuroleptic drugs commonly used in the treatment of psychiatric disorders. It was poorly defined until Caroff’s (1980) review. No single clinical sign or laboratory test exists to identify or confirm it, as NMS is a “syndrome”. It is identified by its close association (95% of recorded cases) with four distinct clinical signs: severe muscle rigidity, hyperthermia, autonomic instability, and altered level of consciousness (Hoffman, Wax & Keyes, 1992). To make diagnosis more difficult, NMS shares signs with malignant hyperthermia, lethal catatonia, heat stroke, and central nervous system infection. If left untreated, NMS has a 100% mortality, and a death rate of 15-30% even when treated promptly (Hoffman, et al, 1992; Staab, 1994).

The authors illustrate the clinical picture of a critically ill patient with NMS through a case study. The epidemiology, pathophysiology, and clinical signs and symptoms of NMS are described. Laboratory investigations and nursing management are reviewed.

Case Study

Mrs. S.K. was a 47-year-old woman with a long psychiatric history that included depression and obsessive-compulsive behaviour, compounded by non-compliance with medication. Her medications included clomipramine, amilzide, lorazepam, nadolol, and flurazepam. There had been a recent increase in the dosage of clomipramine.

Following the dosage increase, S.K became increasingly agitated and aggressive at home. While S.K.’s husband was driving her to see the physician, she had a seizure, became unresponsive and was admitted to the local hospital. On arrival at the hospital, S.K. was hypertensive, tremorous, febrile, and had a decreased level of consciousness. Because of the severity of her condition, she was transferred to the intensive care unit at a tertiary centre to rule out sepsis and drug overdose.

S.K. was admitted to the intensive care unit at 2015hr. She was hypotensive and her temperature was 40.9°C. Her Glasgow Coma Scale (GCS) was 9 and her pupils were non-reactive at 6 mm. Her skin was ashen, cool and she was rigid. A computerized axial tomography (CT) scan of the head was normal. Creatinine phosphokinase (CK) was 1437 with a MB fraction of 0.9%.

She was immediately intubated and placed on a T-piece. A radial arterial line and triple lumen catheter were inserted. She was given saline boluses for hypotension and a Dopamine drip was initiated. This was quickly titrated up to 7.5 µg/kg/min. Epinephrine 500 µg was administered intravenously (IV) for a systolic blood pressure of 53 mmHg. An epinephrine drip was commenced at 1 µg/kg/min and rapidly titrated as high as 24 µg/kg/min over the next two hours with intermittent boluses of 500µg of epinephrine given as required. Midazolam and benzotropine mesylate (cogentin) were given IV push to reduce muscle rigidity. A lumbar puncture (LP) was performed and reported as normal.

S.K.‘s muscle rigidity was so severe that it was difficult to obtain an acceptable quality 12 lead ECG. The elevated temperature and muscle rigidity prompted several boluses of dantrolene sodium. A pulmonary artery catheter was inserted for further monitoring. At this point S.K.‘s cardiac output was 3.23 l/min, systemic vascular resistance (SVR) was 817 dyne-sec/cm² and systolic blood pressure was 78 mmHg.

Arterial blood gases (ABGs) revealed hypoxia and both respiratory and metabolic acidosis. At 2250 hours S.K. experienced a respiratory arrest necessitating hyperventilation and hyperoxygenation with 100% oxygen. She was placed on an SIMV rate of 10, with a TV 700 ml, FiO₂ 0.5, PEEP +5 cmH₂O and pressure support of 12 cm H₂O. Epinephrine and dopamine required frequent titration to attain and maintain a systolic blood pressure above 100 mmHg. Subsequent ABGs revealed continued hypoxia. The FiO₂ was increased to 0.60 and subsequently 0.70.

By 0100 S.K.‘s temperature had dropped from a high of 41.9°C to 35.4°C and a warming blanket was applied. Her CKs reached 1910. The epinephrine infusion resulted in SVRs of 2000 to 3200 dynes-sec/cm² and blood pressure of 74 mmHg. Sodium nitroprusside was added to decrease the SVR, which resulted in an increased cardiac output.

Continuing problems with hypotension on day one necessitated a change in therapy. Amrinone was added at 5 µg/kg/min. The dopamine was weaned to 2.5 µg/kg/min, epinephrine to 12 µg/kg/min and nitroprusside off.
Urinary output and high pulmonary artery wedge pressure (PAWP) were treated with furosemide. Weaning from the ventilator was begun. A coagulopathy developed and was treated with vitamin K, platelets and red blood cells. Low urine output and high pulmonary artery wedge pressure (PAWP) were treated with furosemide. Weaning from the ventilator was begun.

Hypertension and tremors complicated the clinical picture for the next several days. On day eight mobilization was attempted but was unsuccessful. S.K. was unable to lift her head, had no torso control, and became dusky and hypertensive. Her GCS remained at 10. S.K.’s eye movements were abnormal, deviating to the right. She had facial tremors and twitching. An electroencephalograph (EEG) performed on day eight revealed no focal abnormality or asymmetry of focal discharge. S.K.’s GCS remained at 10 or 11.

On day nine, a second attempt at mobilization was made. Again, this was unsuccessful. S.K. became hypertensive (systolic BP of greater than 190 mmHg), bradycardic and hypoxic (SpO₂ 30%). A decreased level of consciousness was quickly resolved by ventilation with a 100% bag-valve system.

On day 11, an unsuccessful extubation resulted in reintubation and use of external CPAP of +5 cmH₂O within one half hour. A percutaneous tracheostomy was performed. S.K. continued to require critical care nursing for the next month. Her GCS returned to 15; although motor strength was poor. Additional complications included minor seizure activity and a right arm deep venous thrombosis (DVT). S.K. was eventually transferred to the neuroscience ward. A week later she was transferred to her home hospital with a percutaneous enteral gastric feeding tube and tracheostomy in place. She had been weaned from mechanical ventilation but remained on anticoagulant therapy.

**Neuroleptic malignant syndrome: Epidemiology and pathology**

Neuroleptic agents such as haloperidol and Prozac are commonly used in the treatment of psychiatric disorders. They act on the brain’s dopaminergic neurotransmitter system, antagonizing the effects of dopamine in the basal ganglia. The results include a reduction in symptoms by sedating, tranquilizing, and blunting emotional expression or aggressive and impulsive behaviour in patients receiving therapy (Petersdorf, 1994).

The incidence of NMS has been reported as 0.5% to 1% of patients receiving either one or more neuroleptic agents (Staab, 1994). Haloperidol has been the sole agent in 28% of cases and associated with 50% of all reported cases. Other drugs used concomitantly with haloperidol are lithium, tricyclic and monoamine oxidase inhibiting antidepressants, antiparkinsonian agents, and benzodiazepines (Caroff & Mann, 1993; Petersdorf, 1994). Neuroleptic malignant syndrome is not the result of drug overdose, as it occurs predominantly in patients whose blood levels are within normal therapeutic range (Caroff & Mann, 1993).

Studies suggest that the mean age of patients diagnosed with NMS is estimated to be 40 years. Data also show that NMS is more common in males. However, these two variables may be confounded by the different ways in which neuroleptics are used in the treatment of men and women, and also among patients of different age groups (Caroff & Mann, 1993).

Heredity does not appear to play a role in the occurrence of NMS. Certain disorders such as mood swings, catatonia, schizophrenia and pre-existing abnormalities in the state of brain dopamine activity or receptor function have been associated with a predisposition to the development of NMS (Caroff & Mann, 1993). Exhaustion, dehydration, and agitation were found to precipitate the diagnosis of 75% of reported cases of NMS (Caroff & Mann, 1993). It is unclear whether these are risk factors or in fact early symptoms of NMS.

Neuroleptic malignant syndrome usually occurs within 24 to 72 hours of the initiation of neuroleptic drug therapy or an increase in drug dosage (within therapeutic range), and 90% of all cases occur within 10 days (Caroff & Mann, 1993; Heiman-Patterson, 1993; & Staab, 1994). As neuroleptic drugs are not easily dialyzable, a delay in resolution of symptoms for five to 10 days after the discontinuation of oral neuroleptic therapy is not uncommon (Petersdorf, 1994).

Disruption of the basal ganglia resulting in the blockage of dopaminergic receptors in the striatum causes skeletal muscle contractions. Loss of temperature control occurs with disruptions in the hypothalamus. Four prominent clinical signs found in 95% of NMS patients are severe muscle rigidity,
hyperthermia, autonomic instability, and an altered level of consciousness. These signs must be examined and differentiated from lethal catatonia, malignant hyperthermia, heat stroke, and central nervous system (CNS) infection.

**Clinical signs and symptoms**

Severe muscle rigidity, in which the patient appears to have a “lead pipe-like” stiffness (joints are movable but resistance is similar to a soft lead pipe) occurs in 98% of cases (Caroff & Mann, 1993; Staab, 1994). Complications of this symptom are poor chest wall expansion leading to hypoxia, respiratory compromise and failure, rhabdomyolysis (muscle breakdown) with metabolic acidosis and renal damage complicated by resultant myoglobinuria and dehydration. Muscle rigidity differs from lethal catatonia because catatonic patients do not present with autonomic compromise, fever or leukocytosis as do NMS patients. Tetanus, also characterized by muscle rigidity, dysphagia, and respiratory complications, should be included in the differential diagnosis of NMS (Kerns, 1992).

Another sign of NMS is hyperthermia, which is pyrexia of 38.0°C to 43.0°C. As a result of hyperthermia, cellular energy demands outstrip available energy supplies leading to membrane dysfunction and cellular injury (Hoffman, et al, 1992). Unlike malignant hyperthermia (also characterized by muscular rigidity, temperature elevation, and metabolic acidosis) which occurs predominantly in response to the introduction of anesthetics (Heiman-Patterson, 1993), hyperthermia in NMS occurs because of severe and continuous contraction of skeletal muscles. Hyperthermia coincides with the blockade of the central dopaminergic mechanism required for the dissipation of heat (Dilsaver, 1993). Diaphoresis commonly occurs with hyperthermia leading to severe dehydration that is compounded by dysphagia and decreased oral intake (Staab, 1994). Heat stroke, which may also be confused with hyperthermia in NMS does not present initially with muscle rigidity.

Autonomic instability includes clinical signs such as tachycardia, dysrhythmias, labile blood pressure, tachypnea, diaphoresis, urinary incontinence, and exaggerated deep tendon reflexes (Kerns, 1992; Staab, 1994). Aggressive treatment is necessary to prevent these from progressing quickly to shock, and respiratory and/or cardiac arrest.

Another sign of NMS, an altered level of consciousness, involves such early clinical signs as agitation or akathisia, and stupor that alternates with lethargy and depression. Progression towards a comatose state will occur. It is very important to note that these early behaviours (such as agitation) may be manifestations of the initial psychiatric disorder. A mistake in treatment may involve the addition of, or increase in, neuroleptic agents to treat what is believed to be an exacerbation of the underlying psychiatric disorder. This of course will only aggravate NMS (Staab, 1994). Neuroleptic malignant syndrome may resemble a CNS infection such as meningitis, since alterations in level of consciousness occur in both conditions. This must be ruled out early since prompt treatment is critical.

**Laboratory investigations**

While there is no definitive laboratory test to confirm NMS, diagnosis of the syndrome can be supported by a variety of tests. Hallmarks of NMS include an elevation of CK as well as metabolic acidosis. Myoglobinuria, secondary to muscle rigidity and rhabdomyolysis may also occur. If left unchecked, rhabdomyolysis leads to decreased renal function resulting in increased blood urea nitrogen and creatinine levels.

An LP does not confirm NMS but may be necessary to rule out CNS infection such as meningitis and should be done in conjunction with a complete septic investigation (Hoffman, et al, 1992). Arterial blood gases reveal a metabolic acidosis from muscle rigidity, hypoxemia, respiratory acidosis associated with poor chest wall expansion, and respiratory compromise or failure.

Although NMS is not caused by overdose, drug screens and levels should be drawn for baseline data and to confirm the existence or absence of neuroleptic agents (Staab, 1994). Ingesting and intoxication from street drugs such as phenycyclidine (PCP) should be ruled out as this may cause signs and symptoms similar to NMS (Hoffman, et al, 1992).

A complete blood count is done to assess for leukocytosis which occurs in 70 - 80% of NMS patients (Heiman-Patterson, 1993). Metabolic complications of rhabdomyolysis include hyperkalemia, hyper/hypophosphatemia, hyper/hypocalcemia, hypoalbuminemia, and hyperuricemia (Hoffman, et al., 1992), so electrolyte levels are measured. Coagulation tests are useful, as disseminating intravascular coagulation is a further complication of rhabdomyolysis.

Liver function tests are done because the drugs involved in both the development and management of NMS may affect the liver. In over 50% of NMS patients, EEGs showed a nonfocal, generalized slowing of activity (Caroff & Mann, 1993). Pulmonary artery catheter monitoring and derived parameters can guide the complicated treatment regimen. Normally, a patient with severe pyrexia would have vasodilation as a compensatory mechanism, however, NMS patients do not demonstrate this, but have severe vasoconstriction. Therefore, the SVR of NMS patients is elevated.

**Nursing management**

The care of a patient with NMS is challenging. The severity of this syndrome necessitates prompt nursing intervention. Three primary goals in the treatment of NMS patients are fluid replacement, reduction of temperature and support of cardiac, respiratory and renal functions.

Once other disease processes have been ruled out and all neuroleptic drugs discontinued, a head-to-toe assessment should be carried out while steps are taken to stabilize the patient. Arterial, central venous, and pulmonary artery catheters may be inserted to monitor the patient’s response to treatment as hypertension, tachycardia, tachypnea and orthostatic hypotension may be present (Dilsaver, 1993). Isotonic crystalloids, colloids, starches and albumin may be needed to replace volume lost because of diaphoresis. Inotropic support, such as dopamine or dobutamine and vasopressors such as epinephrine may be necessary to correct decreased cardiac output and to maintain adequate cerebral and renal perfusion.

The administration of IV fluids is necessary to maintain adequate preload and renal perfusion. Intake and output must be monitored. Observing blood tinged or tea-coloured urine can indicate if rhabdomyolysis is present.
Dantrolene sodium (dantrolene), a skeletal muscle relaxant, and bromocriptine, a dopamine agonist are effective in the treatment of malignant hyperthermia, clinical spasticity as well as NMS (Hetherington, 1992). Dantrolene, while acting on skeletal muscle, has little or no effect on cardiac or smooth muscle. It works in the skeletal muscle by inhibiting the release of calcium (Heiman-Patterson, 1993). Neurotransmitters are not affected. Central nervous system effects such as drowsiness and dizziness can occur, but respiratory effort is not affected. When mixed, dantrolene should be kept in a darkened area and used within six hours. The IV dosage is 1 to 10 mg/kg/day with repeated oral doses of 4 to 8 mg/kg/day (Vallerand & Deglin, 1996). Body temperature usually returns to normal within hours of the initial dose. Thrombophlebitis has been reported during IV administration (Heiman-Patterson, 1993).

Dantrolene may be used in conjunction with bromocriptine. The usual dose of bromocriptine is 2.5 to 10 mg three times a day, although higher doses of 20 mg four times a day have been reported (Heiman-Patterson, 1993). Hydration and antipyretic medication may be given to reduce fever. Other cooling techniques such as cooled IV fluids, ice packs, cooling blankets, fans or cooling baths may also be used (Waggoner & Hanson, 1992). Vital signs and temperature must be monitored continuously.

Lungs must be auscultated for such complications as pneumonia, aspiration or poor air entry related to muscle rigidity. Signs and symptoms of tension pneumothorax or pulmonary emboli may be evident. Ventilator settings should be adjusted to maintain uniform gas exchange at the tissue and capillary levels. Arterial blood gas monitoring and optimal pulmonary toileting are mandatory. The head of the bed should be elevated to facilitate chest expansion. Pancuronium or vecuronium may be used if the patient is resisting the ventilator. When neuromuscular blocking agents are used, it is important to remember that patients need to be sedated while paralyzed. Patient safety must be guarded by ensuring alarms are on, settings are correct and all tubings are properly connected. Nutritional support must be considered. Patients may be unable to swallow or be in an obtunded state. A nasogastric tube may be inserted to provide enteral feeding or total parenteral nutrition may be considered. Once feeding is instituted, appropriate laboratory values, weight, and fluid balance must be monitored. The patient should remain NPO until swallowing has returned (Hetherington, 1992). Infusion and skin breakdown are highly possible because of immobilization; therefore the patient must be monitored for signs of infection and receive antibiotics as ordered. Basic nursing care such as turning and repositioning, massage, air mattresses, heel and elbow pads, mouth care and eye lubrication should be provided (Staab, 1994). Calf compression devices should be used to decrease the incidence of DVTs. Seizures, though very rare, are another complication. Appropriate precautions should be taken as per unit policy.

The nurse also plays a large role in assisting the family to cope with the stress of the situation. Frequent communication between the health care team and family members before and during therapy can help prevent misinterpretation of information. The machinery, tubes and lines may overwhelm and frighten the family when they see their loved one for the first time. The family should be both allowed and encouraged to express their fears and concerns. The nurse should answer any questions and give appropriate information, without generating unrealistic expectations. Explanations and reinforcement by the nurse as to the purpose of the therapies helps to reassure the family that the patient is comfortable and receiving good care. Social work, pastoral care and other disciplines may be consulted to provide holistic care to the patient and family.

**Conclusion**

Neuroleptic malignant syndrome is a potentially fatal complication of neuroleptic drug use. The patient with NMS can quickly become critically ill and challenge the skills of the critical care nurse. The problems of muscle rigidity, hyperthermia, autonomic instability, and an altered mental status must be assessed and managed efficiently to promote the optimal outcome for the patient.

Linda Lethbridge, RN and Shelley Shields, RN, BScN are staff nurses in intensive care and Francis Loos, RN, MN, CNCC(C), is clinical development educator in the intensive care units at Plains Health Centre, Regina Health District, Regina, Saskatchewan.
Continuing Education (CE) hours from this article will be granted by CACCN. The CE hours can be applied to recertification in the critical care specialty [CNCC(C)] as designated by the Canadian Nurses Association certification program.

Quiz Topic: Neuroleptic malignant syndrome: A case study

Educational Objectives:
Based on the content of the article, you should be able to: 1. Describe the clinical presentation of neuroleptic malignant syndrome. 2. Explain the etiology. 3. Describe the pathophysiology associated with neuroleptic malignant syndrome. 4. List the appropriate interventions.

Instructions:
To receive CE hours for this quiz, mark your answers on the enrollment form. Complete the form and submit it with the $12.00 processing fee to CACCN, P.O. Box 22006, London, Ontario, N6C 5Y3. This enrollment form must be postmarked by January 1, 1999. Three weeks after the enrollment form and payment are received by CACCN, a corrected answer form will be sent to you. If you receive a passing score, a CE hour certificate will be enclosed.

Quiz Writers: Francis Loos, RN, MN, CNCC(C), Linda Lethbridge, RN, & Shelley Shields, RN, BSN

Credit: You can earn 2.0 CE hours with a passing mark of 11 (79 percent) correct answers on this quiz (ID #CACCN98-1).

Questions:
1. Which one of the following is a drug implicated in the development of neuroleptic malignant syndrome?  
a. Epinephrine; b. Bromocriptine;  
c. Haloperidol; d. Dantrolene

2. Which of the following is associated with the development of neuroleptic malignant syndrome (NMS)?  
a. A decrease in the dose of a neuroleptic agent; b. An overdose of a neuroleptic agent;  
c. Initiation of, or an increase in, the dose of neuroleptic agent; d. A hereditary predisposition to NMS

3. Which of the following is a clinical sign of NMS?  
a. Hyperthermia; b. Hypokalemia;  
c. Autonomic stability; d. Muscle flaccidity

4. Which of the following is the first line treatment for NMS?  
a. Increase the dose of neuroleptic agent; b. Dialysis to remove the neuroleptic agent;  
c. Volume expansion to encourage excretion of the agent; d. Stop the neuroleptic agent

5. Agitation, akathisia and stupor are early signs of which of the following problems associated with NMS?  
a. Altered level of consciousness; b. Exacerbation of the underlying disease;  
c. Development of a coexisting CNS disease; d. Low serum sodium

6. Arterial blood gases in the NMS patient with muscle rigidity reveal which of the following?  
a. Metabolic acidosis and hypoxemia; b. Metabolic alkalosis and hypoxemia;  
c. Metabolic acidosis and low PaCO₂; d. Respiratory alkalosis and hypoxemia

7. Which of the following are laboratory findings supporting the diagnosis of NMS?  
a. Lumbar puncture CSF with a high white blood cell count; b. Normal serum potassium;  
c. Leukocytosis; d. Normal serum creatinine kinase

8. Which of the following is the mechanism by which dantrolene controls the muscle rigidity of NMS?  
a. Dantrolene inhibits the release of calcium in skeletal muscle; b. Dantrolene stops the release of neurotransmitters at the neuromuscular junction;  
c. Dantrolene relaxes smooth muscle; d. Dantrolene opens the calcium channels in the muscles

9. Which of the following causes hyperthermia in NMS?  
a. Disruptions of the hypothalamus; b. Dehydration;  
c. Excitation of the dopaminergic receptors in the striatum; d. Use of anesthetic agents

10. Autonomic instability of NMS differentiates it from which of the following problems?  
a. Lethal catatonia; b. Tetanus; c. Heat stroke; d. Meningitis

11. Which of the following is a primary goal in the treatment of NMS?  
a. Fluid restriction; b. Reduction of temperature;  
c. Increase of the neuroleptic agent; d. Increase muscle tone

12. Which of the following is a complication of rhabdomyolysis?  
a. Hypokalemia; b. Hyperaluminaemia;  
c. Hypouricemia; d. Hypercalcemia

13. Mr. K is becoming increasingly frustrated by his wife’s lack of response to the treatment of her NMS. Which of the following responses would be most appropriate?  
a. Encourage communication between the medical team and Mr. K.; b. Encourage the family to express concerns and fears;  
c. Call for a meeting of the health team immediately; d. Tell the husband you will talk to the physician

14. Which of the following is the mortality rate of NMS despite prompt treatment?  
a. 5%; b. 12%; c. 30%; d. 45%
CE Quiz Enrollment Form  
CACCN # 098-1  
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Photocopy this form and mark your answers in the appropriate spaces. 
This form expires on January 1, 1999.

Quiz Topic: Neuroleptic Malignant Syndrome: A Case Study

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**Program Evaluation**

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<td>Easy</td>
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Address:___________________________________________ City: _________________________________
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CNA Certification #: ________________________________ Specialty: ___________________________________
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Systemic necrotizing vasculitis: A case of Wegener’s granulomatosis

Lynn Riley, RN, and Patricia Daines, RN, BScN, CNCC(C)

Abstract

Wegener’s granulomatosis, a systemic necrotizing vasculitis, includes a triad of destructive inflammation involving the upper and lower respiratory tracts and the kidneys. Its cause is unknown. Management is often complicated by pulmonary hemorrhage and renal failure that occur as a result of the disease pathophysiology. Forty years ago, a diagnosis of Wegener’s granulomatosis was considered fatal, with a mean survival time of five months from onset. Today, with early recognition and new treatment options, remission is possible. A case study approach is used to illustrate the many facets of the disease process and treatment. Nursing management, which focuses on understanding the complex nature of the disease and its various treatment options, is explored.

Marie, a 37-year-old female, was admitted to our intensive care unit (ICU) with a one-week history of general malaise and fever. She had been experiencing hemoptysis for the past five days with a drop in hemoglobin from 102 g/L to 79 g/L. On admission, she was awake and alert, receiving oxygen by face mask at 40%, and breathing at a rate of 30 breaths per minute.

Her chest x-ray showed diffuse bilateral infiltrates and she continued to produce bloody sputum. Marie reported that one week prior to admission she had a migraine episode with transient hemiplegia. At that time, she experienced some
dyspnea, had a productive cough and a fever. She recalled having an eye infection two to three weeks prior to admission, and complained of pain and itching in her feet which she described as “walking on pins and needles”.

On day two in the ICU, Marie required intubation and ventilation for deteriorating respiratory function. She required 100% oxygen and was maintained on pressure control ventilation. The gross hemoptysis continued and it became increasingly difficult to maintain adequate oxygenation. At this point, Marie was sedated and chemically paralyzed.

On day three, the nephrology service was consulted. On examination, a urinalysis was grossly positive for red blood cell (RBC) casts. It was presumed that Marie had a renal-lung syndrome, and a blood specimen was sent for anti-neutrophil cytoplasmic autoantibodies (ANCA) testing. Later that day, Marie was seen by rheumatology specialists who concurred that the diagnosis was indeed a renal-lung syndrome, likely Wegener’s granulomatosis (WG).

Wegener’s granulomatosis is one of several systemic necrotizing vasculitic diseases. Its presentation and course most often involve the upper and lower respiratory tracts and the kidneys. A renal-lung syndrome, usually seen in the ICU setting, consists of pulmonary hemorrhage and acute renal failure due to glomerulonephritis. Prior to this experience with Marie, we were unfamiliar with WG. A review of the literature revealed much medical information in textbooks and journals, but no nursing literature was found. Through the use of this case study, the complex nature of WG will be explored along with the challenges it poses for nurses in the ICU.

**History and prevalence**

The history of WG spans some 60 years. In the late 1930s, Dr. Friederich Wegener described a systemic vasculitic disease which included the upper and lower respiratory tracts and kidneys in three patients (Geiger, Garrison & Losh, 1992). By the 1950s, WG was known as a fatal disease with a mean survival time of five months (Walton, 1958). While the last 40 years have brought new knowledge about WG’s presentation, course, and treatment, its cause remains unknown. Today WG is considered an uncommon, but not rare disease. With early detection and treatment, survival and remission are possible.

Wegener’s granulomatosis occurs in patients of all ages (9-78 years) with a mean of 41 years. Both genders are equally represented. Prevalence has been described as 3.0 per 100,000 persons. It has been suggested that WG’s prevalence has been underestimated due to its variability and course as well as evidence of mild forms of the disease (Duna, Galperin, & Hoffman, 1995).

**Pathophysiology**

In order to better understand WG, it is helpful to explore the pathogenesis underlying systemic vasculitis. Vasculitis is a process which is characterized by inflammation of blood vessels. The inflammation associated with systemic vasculitis can involve any blood vessel in the body, and may result in dysfunction of any organ system (Haynes, Allen & Fauci, 1986). Although some forms of vasculitis have unknown causes, others have been associated with infection, or medications, such as quinidine (Falk & Jennette, 1992). Vasculitis may also occur as part of certain systemic disease processes (eg. systemic lupus erythematosus, rheumatoid arthritis).

Several diagnostic categories currently exist which classify the vasculitides. Wegener’s granulomatosis is one of several disease processes categorized under the heading of systemic necrotizing vasculitis. These processes have in common the features of vascular inflammation, vascular necrosis, and varying degrees of ischemia in target organs. They are believed to be immunologically mediated, but rarely have causative agents been identified (Calabrese, Hoffman, Guillemin, 1995).

Vasculitis is a prominent feature of WG. Three distinct forms can be seen such as: microvasculitis, granulomatous vasculitis, and necrotizing vasculitis. Microvasculitis occurs as a result of infiltration and destruction of capillaries, venules and arterioles by neutrophils (Sneller, 1995). Microvascular lesions can occur in the lungs, resulting in diffuse alveolar hemorrhage, or on the skin where palpable purpura is seen. When the microvascular lesions are found in the kidney, they are associated with necrotizing glomerulonephritis (Sneller, 1995). Granulomatous vasculitis refers to a form of vascular lesion, or granuloma, resulting from inflammation and involving small or medium sized arteries and veins (Anderson & Anderson, 1990). Granulomatous lesions may be discrete or ill-defined. They are composed of granulocytes (specifically neutrophils), multinucleated giant cells, and necrotic debris (Jennette, 1991).

Granulomas are often seen in the

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**Table One: Organ involvement in Wegener’s granulomatosis**

<table>
<thead>
<tr>
<th>Organ</th>
<th>Frequency, %</th>
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<tbody>
<tr>
<td>Lung</td>
<td>95+</td>
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<tr>
<td>Paranasal sinuses</td>
<td>90+</td>
</tr>
<tr>
<td>Kidneys</td>
<td>80-85</td>
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<tr>
<td>Nasopharynx</td>
<td>65-90</td>
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<tr>
<td>Joints</td>
<td>55-65</td>
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<tr>
<td>Skin</td>
<td>45</td>
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<tr>
<td>Ear</td>
<td>40-60</td>
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<tr>
<td>Eye</td>
<td>40-60</td>
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<td>Nervous system</td>
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<td>Larynx</td>
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upper and lower respiratory tracts and lungs, in association with inflammation of lung parenchyma. Necrotizing vasculitis is characterized by necrosis, fibrosis, and proliferation of the inner layer of the vessel wall (Anderson & Anderson, 1990). It involves small to medium-sized arteries or veins and can occur in any organ system (Sneller, 1995). Any of these three forms of vasculitis can result in thrombosis and occlusion of blood vessels leading to tissue necrosis (Sneller, 1995).

**Clinical presentation and diagnosis**

As with other forms of systemic necrotizing vasculitis, clinical signs and symptoms of WG may be vague, mimic other systemic processes, and therefore make a definitive diagnosis difficult. Clinical features which distinguish WG from other forms of vasculitis are involvement of the upper and lower respiratory tracts in association with glomerulonephritis (Bennett, 1996). Although some patients present with what has been described as a limited form of WG (involving only the upper airways), this discussion is confined to the classic or generalized presentation.

It is believed that WG begins as a localized process. After a variable period, the disease becomes more aggressive and the classic systemic picture develops with progressive renal involvement (Geiger et al., 1992). Most patients with WG first exhibit upper and lower airway symptoms, as shown in Table One. Sinusitis, nasopharyngitis, epistaxis and otitis media are common upper airway symptoms (Balau & Fauci, 1993). Pulmonary involvement is manifested by cough, dyspnea, chest pain and hemoptysis. On chest x-ray, nodules or nodular infiltrates which may cavitate can be seen (Sneller, 1995). Renal involvement consists of glomerulonephritis. Approximately 20% of patients will manifest this initially, along with respiratory symptoms (Sneller, 1995). Usually a microscopic hematuria and low grade proteinuria can be demonstrated clinically (Bennett, 1996).

The diagnosis of WG or any other vasculitis is challenging in the ICU setting where multisystem disease of varying origins is common. Clues to a possible vasculitis in the ICU patient include fever, pulmonary infiltrates, altered mental status, abdominal pain, seizures, congestive heart failure, arthralgia, hypertension, skin lesions, limb ischemia, weakness, and renal failure (Houk, 1992). As well, certain laboratory markers provide a potentially powerful diagnostic aid such as anti-neutrophil cytoplasmic autoantibodies (ANCA) (Geiger et al., 1992).

**ANCA testing**

In the 1980s it was discovered that many patients with systemic vasculitis had anti-neutrophil cytoplasmic autoantibodies present in their circulation (Hoffman, 1994). These autoantibodies are specific for antigens in the cytoplasm of neutrophils and monocytes (Jennette, 1991). Two types of ANCA have been described. Type p-ANCA produces a perinuclear staining pattern using direct immunofluorescence techniques on neutrophils that are ethanol-fixed. Type c-ANCA produces a cytoplasmic staining pattern using the same technique. In most studies it was found that both the sensitivity and the specificity of the typical c-ANCA pattern for active WG is approximately 90% (Hoffman, 1994). Thus, approximately 90% of patients with active WG will demonstrate positive c-ANCA test results, making it a helpful tool for diagnosis (Hoffman, 1994; Falk & Jennette, 1992). ANCA’s role in the pathogenesis of WG is currently unknown and continues to be studied (Duna et al., 1995). When diagnosing WG, it is important to note that some false positive ANCA results have been reported; hence, the benefit of positive tissue diagnosis along with ANCA results.

For those patients who have been diagnosed with WG, ANCA measurements are useful in determining if the disease is active or quiescent. If ANCA titers become elevated, it is likely that the disease has exacerbated; negative ANCA test results in those diagnosed with WG indicate remission (Falk & Jennette, 1992). Titers rise and fall with exacerbations and remissions (Geiger et al., 1992).

On day four, one day after Marie’s blood was sent for ANCA testing, a positive c-ANCA result was reported. Marie’s clinical picture remained consistent with the preliminary diagnosis of WG. Her chest x-ray showed diffuse alveolar hemorrhage and she had active urinary sediment. A sinus biopsy showed evidence of chronic inflammation but no signs of vasculitis. [The amount of tissue available for biopsy in this area is not always sufficient to demonstrate vasculitic changes (Duna et al., 1995).] It was thought that a renal biopsy would be helpful to determine

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**Table Two: Key points to consider when assessing and monitoring the patient with Wegener’s granulomatosis**

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<td>• Arterial and mixed venous gases</td>
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<td>• Airway obstruction due to hemoptysis and tissue sloughing</td>
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<tr>
<td>• Signs of barotrauma (elevated ventilating pressures)</td>
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<td>• Need for sedation and chemical paralysis</td>
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<th>Hemodynamics</th>
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<tr>
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<tr>
<td>• Need for inotropes and vasopressors</td>
</tr>
<tr>
<td>• Signs of hypovolemia due to pulmonary hemorrhage and extravascular fluid shifts</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Kidney function</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Signs of renal failure (urine output, laboratory values)</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Effects of immunosuppressive treatment</th>
</tr>
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<tr>
<td>• Leukopenia</td>
</tr>
<tr>
<td>• GI bleeding</td>
</tr>
<tr>
<td>• Opportunistic infections</td>
</tr>
</tbody>
</table>
Marie’s diagnosis, but her condition was too unstable to allow such a procedure.

**Treatment**

Treatment is aimed at halting disease progression by interrupting the inflammatory process and suppressing the immune response. As knowledge of WG has evolved over the last 40 years, treatment protocols have changed. Corticosteroids were used as early as the 1950s to control the inflammation associated with WG (DeRemee, 1995). Prior to this, most patients died within one year (Walton, 1958). Therapy with corticosteroids alone may have decreased inflammation and improved systemic and upper airway systems temporarily; however, they failed to control significant pulmonary or renal disease (Fauci & Wolff, 1973).

In the early 1970s, cytotoxic drugs were added to existing treatment protocols using corticosteroids alone. Cytotoxic drugs modify the disease activity through their effects on the immune system (Sack, 1989). The use of cyclophosphamide, an alkylating agent, dramatically increased survival time for some patients, and resulted in remission of WG in others (Fauci & Wolff, 1973).

Cyclophosphamide can be given orally or intravenously in patients with fulminant disease. The initial daily dose is maintained for 10 days to two weeks. Dosage is increased at this point if a favourable clinical response is not seen. The rate of increment is continued until evidence of a clinical response is seen or signs of toxicity appear, such as leukopenia. Clinical response is measured by a decrease in respiratory signs and symptoms, remission of peripheral manifestations of vasculitis, and improvement in renal function. Cyclophosphamide therapy is usually continued at a maintenance dose for one year after all traces of disease disappear (Fauci & Wolff, 1973).

Methotrexate, another cytotoxic immunosuppressive agent, has been used in low dosage for some patients with WG. This antimetabolite has been used effectively in patients who have not responded to treatment with cyclophosphamide or have experienced cyclophosphamide-related toxicity (Duna et al., 1995).

Cyclosporine, a noncytotoxic immunosuppressive agent, may be considered for use in patients who have responded poorly to other cytotoxic agents. Within a certain dose range, cyclosporine has been effective in maintaining disease control; however, associated nephrotoxicity limits its benefit for WG patients (Duna et al., 1995).

Most patients with WG develop respiratory tract infections at some point during the course of their disease. Antimicrobial agents, specifically trimethoprim/sulfamethoxazole, have been identified as a possible solution to the relapse of disease activity experienced in patients related to infectious organisms (Duna et al., 1995).

Intravenous immunoglobin (IVIg) is used in a variety of inflammatory and autoimmune diseases as it has a theoretical ability to interact with the idiopathic network of immunoregulation (Calabrese et al., 1995).

Plasmapheresis is described as an unproven treatment for some patients with WG. It may prove useful in those patients with severe renal insufficiency or massive pulmonary hemorrhage. Each exchange replaces 3-4 litres of a patient’s plasma (Falk & Jennette, 1992). The benefit of plasmapheresis is based on the rationale that it removes immune complexes and low affinity antibodies and helps to restore the clearing function of the reticuloendothelial system (Houk, 1992).

Patients with rapidly progressive renal failure may require intermittent hemodialysis. The prognosis is generally not favourable in these cases. Reversibility of renal disease depends on the severity of necrosis and the extent of irreversible sclerosing lesions (Balau & Fauci, 1993).

Marie was started on a five-day course of IVIg prior to a confirmed diagnosis of WG. Cyclophosphamide and corticosteroids were added as soon as her positive c-ANCA result was received. On day five, trimethoprim/sulfamethoxazole was started as antimicrobial prophylaxis particularly against pneumocystis carinii pneumonia for which she was thought to be at risk. Marie did not respond well to immunosuppressive therapy; by day 12, her oxygenation had worsened and high frequency jet ventilation was initiated. Pulmonary hemorrhage continued, and a milrinone infusion was started for its inotropic/vasodilator action in an attempt to lower her high pulmonary artery pressures. By decreasing right ventricular pressures and right ventricular afterload, it was thought that the pulmonary hemorrhage would decrease. In addition to her deteriorating pulmonary status, Marie continued to have red blood cell casts in her urine. Serum blood urea nitrogen (BUN) and creatinine continued to rise. Cyclophosphamide was discontinued for her pancytopenia and granulocytopenia colony stimulating factor (GCSF) was starting to stimulate WBC production. Vitamin E was added to her chemical treatment for its theoretical antioxidant effects in the face of continued high fraction of inspired oxygen.

**Nursing challenges**

Marie’s care required constant attention to maintain a balance between the effects of treatment and those of the disease (Table Two). Immunosuppressive drugs were administered in an attempt to reverse the pulmonary and renal effects of WG. These same drugs are known to cause serious adverse effects (e.g. gastrointestinal bleeding, leukopenia, inability to fight infection/risk of opportunistic infection). Airway and breathing management meant being alert to and monitoring for any changes in respiratory status. Intermittent bleeding from upper airways required frequent suctioning to maintain a patent airway. An in-line suctioning system was used to facilitate frequent suctioning, and minimize the need for disconnection from the ventilator. Oxygen saturation was monitored frequently, as were arterial and mixed venous blood gases in order to continuously evaluate the effects of the many ventilator changes and other interventions aimed at improving ventilation. The risk of barotrauma related to high airway pressures and increased positive end expiratory pressure...
pressure requirements was an ever present concern, requiring close monitoring for evidence of pneumothoraces.

Marie’s unstable hemodynamic picture demanded constant vigilance and assessment for the need to titrate inotropes and add vasopressors. While sedation and chemical paralysis with neuromuscular blockers helped to stabilize Marie’s hemodynamic and respiratory swings, their use created other potential problems. The inability to move, in addition to generalized edema, posed a threat to skin integrity. Marie was placed on a three inch foam mattress, her heels were kept off the bed, and hydrogel wafers were applied to her elbows. Turning and repositioning were difficult as they resulted in desaturation and a decrease in blood pressure. Passive range of motion was performed regularly and was better tolerated.

Marie had a strong support system in her husband, parents, and siblings who took turns caring for the children and maintaining a 24-hour vigil at her bedside. Her inability to communicate with her family, and the change in her appearance posed different challenges. The presence of gross generalized edema, blood oozing from nose and mouth, and hair loss due to the cyclophosphamide greatly altered how she looked. Marie’s husband decided it was best for their three small children not to visit her. Photographs of Marie with her husband and family were placed in her room. Great care was taken to manipulate her long blond hair as little as possible in an attempt to preserve it. Her own nightgowns were brought from home and laid over her to normalize her appearance as much as possible. Her family were encouraged to be at Marie’s bedside throughout her illness. Although her condition remained very unstable, whenever possible they participated in her care.

Possibly the greatest challenge in caring for Marie was understanding a disease with which the health care team had no previous experience. Information sharing among the team and with Marie’s husband and family was a continuous and vital process. Knowing what to expect, the constant need to keep one step ahead, and maintaining hope under extraordinary circumstances presented a personal challenge for all those who were part of Marie’s care.

On day 36, there was a marked deterioration in Marie’s condition. She remained on high frequency jet ventilation and had chest tubes inserted for bilateral pneumothoraces. In a final attempt to improve ventilation, Marie was placed in a prone position to maximize ventilation and perfusion of the anterior lung areas. Five weeks had passed since Marie was admitted to the ICU. Tragically, all efforts to support her were futile and she died surrounded by her parents and husband who never left her side.

**Conclusion**

Through Marie’s experience the authors have endeavoured to portray the many aspects and challenges of caring for a patient with Wegener’s granulomatosis. Despite the progress made over the last 50 to 60 years in the diagnosis and treatment of WG, its cause remains unknown. Although the literature shows little hope for a cure at the present time, with early detection and treatment, remission and survival are possible.

Lynn Riley, RN, is a staff nurse, critical care unit, at Sunnybrook Health Science Centre in Toronto, Ontario. Patricia Daines, RN, BScN, CNCC(C), is an educator, critical care unit at Sunnybrook Health Science Centre.

**References**


Advanced directives in critical care

By Beverly Leith, RN, BScN, CNCC(C)

Abstract

There have been great advances in critical care medicine which now permit the extensive use of life-sustaining treatments. However, not all patients want to receive life-prolonging therapies for every health care crisis. Critically ill patients are often unconscious or incompetent to indicate their treatment preferences. Advance directives (ADs) promote patient autonomy and self-determination by permitting individuals to document their health care preferences in the event that they become incompetent to make health care decisions in the future. The concept of ADs raises several potential issues for health care professionals and patients. This article provides a review of ADs, describes the status of ADs in Canada, explores the issues relating to ADs in the critical care setting, and then identifies potential roles for critical care nurses with respect to ADs.

The concepts of advance directives (ADs) and living wills are receiving much attention in the popular press and professional literature. This author provides a review of ADs, describes the status of ADs in Canada, and explores the issues related to ADs in the critical care setting. Potential roles for critical care nurses with respect to ADs are identified.

The term “living will” was first used in 1969 to describe a document in which a competent adult sets forth directions regarding medical treatment in the event of his or her future incapacitation (Kutner, 1969). It is called a “living” will because it takes effect prior to death as opposed to a last will and testament which takes effect only after an individual’s death. Living wills include general statements about refusing “heroic life-saving measures”. This type of non-specific advance directive has been criticized as being vague and difficult to apply in medical practice.

In an attempt to become more specific, a different form of AD was proposed by Emanual and Emanual (1989) and is called the “medical directive”. The medical directive describes four hypothetical clinical scenarios which are defined by prognosis and disability in incompetent patients. For each scenario, the patient indicates preferences regarding specific life-sustaining interventions (e.g. cardiopulmonary resuscitation, mechanical ventilation, artificial nutrition and hydration, surgery, blood products, antibiotics, pain medications).

In Canada, Singer (1995) has developed a similar format of AD called the “living will” with nine health care situations (e.g. stroke, dementia, terminal illness) for which the individual indicates his or her decision for seven life-sustaining treatment options. These forms of specific ADs have been criticized as difficult to complete without medical knowledge as well as being relevant to only the specific scenarios described (Brett, 1991).

Douglas and McCullough (1991) suggested that the strength of ADs can be enhanced by the use of a “values...
The legal status of advance directives

In 1988, Nova Scotia was the first Canadian province to enact legislation which related to ADs. According to a June 1995 report by the Senate of Canada, Quebec, Ontario, Manitoba, and Nova Scotia had passed different types of legislation relating to ADs. British Columbia, Alberta, New Brunswick, Newfoundland and the Northwest Territories were developing guidelines for ADs. Prince Edward Island, Saskatchewan, and the Yukon had no current legislation. This same report suggested that all provinces and territories should adopt legislation on ADs (Senate of Canada, 1995). It is therefore foreseeable that all Canadian provinces have already or will soon develop different types of legislation recognizing the validity of ADs. Health care professionals must be familiar with the current legislation of their individual province.

Despite the concepts of patient autonomy and self-determination, which support an individual’s right to refuse life-prolonging medical treatment, some health care professionals still fear legal consequences from following a patient’s AD. To date, the issue of ADs has not been directly addressed in Canadian courts. However, if health care professionals ignore a patient’s AD, they could potentially be held liable for battery or assault.

Advance directives in Canada

The limited Canadian studies about ADs indicate a lack of knowledge about ADs. One Canadian study found that only 16 per cent of Canadian outpatients knew about living wills, 11 per cent knew about durable powers of attorney for health care, and only four per cent knew the term AD (Sam & Singer, 1993). While 19 per cent had discussed their health care preferences, none had written them down.

The perceived barriers to completing an AD were found to be the inability to write, the belief that an AD was unnecessary, a fatalistic attitude, previous discussion of preferences, a desire to leave the decision to doctors, uncertainty about preferences, a desire to discuss preferences rather than document them, a desire to wait until the situation arose, a desire to write down preferences in the future, and a desire to avoid thinking about preferences or ADs (Sam & Singer, 1993).

A study of Canadian physicians and nurses found that participants favoured the use of ADs, although physicians had a less positive attitude than nurses.

Table One:
Types of advance directives

- LIVING WILLS: Living wills are a form of advance directive which include general statements about refusing heroic life-sustaining measures in the event of terminal illness or permanent unconsciousness.
- INSTRUCTIONAL DIRECTIVE: Instructional directives allow individuals to identify what or how health care decisions are to be made if they become incompetent. Instructional directives include general living wills, the “Medical Directive”, the “Values History” and other forms of written or verbal descriptions.
- PROXY DIRECTIVE: Proxy directives allow individuals to specify who is to make health care decisions in the event that they become incompetent. Proxy directives include durable power of attorney for health care and proxy decision maker for health care.
- COMBINED DIRECTIVES: A combined advance directive includes both an instructional and proxy directive in one document. It has been recommended that a living will with both general and specific statements should be combined with a durable power of attorney for health care to provide the best assurance that patients’ desires concerning medical treatments will be respected (Silverman, Vinicky & Gasner, 1992).
limited number of American studies have related to ADs in critical care; however, a hospital had a policy on ADs (Rasooly only 2.6 per cent of responding Canadian (p.1435). However, one study found that ADs could be beneficial in facilitating discussions about foregoing life-sustaining treatment at an earlier time in the hospitalization.

Another study evaluated the use of ADs in the ICU of an American tertiary care cancer centre (Ewer & Taubert, 1995). The researchers found that many problems may have been avoided and considerable cost savings might be achieved by the early presentation of ADs and the evaluation of clinical goals on an ongoing basis. Sixteen per cent of patients admitted to the ICU for non-surgical acute care medical problems presented ADs; however, 26 per cent of these ADs were made known to the health care team only one to two days after admission to ICU. Some patients feared that acknowledging the presence of an AD may make them less likely to be eligible for ICU. In other cases, patients and families believed there was no need to acknowledge the existence of an advance directive before the onset of critical illness. In five cases, the attending physician indicated that the patient might not have been admitted to the ICU had the AD been presented earlier.

A third American study found that ICU patients with documented ADs were 3.6 times more likely to have life-sustaining interventions withheld from them than ICU patients without documented ADs (Kollef, 1996). However, a documented AD did not ensure the withdrawal of life sustaining interventions in all patients. Thirty-eight per cent of the patients who had documented ADs did not have life-sustaining interventions withdrawn prior to death.

Issues relating to advance directives in critical care

Advance directives are important issues in critical care because this is the area where the majority of life-sustaining treatment takes place. Critical care health care professionals must be aware of several issues related to ADs. Unfortunately, this article does not provide sufficient space for a detailed discussion of the ethical and moral issues related to ADs. The major issues are briefly described below.

• Competency in critical care patients. Critically ill patients are often unable to participate in decision making due to confusion because of acute illness, centrally active drugs, and sensory or sleep deprivation. Other critically ill patients may be unconscious or in a coma state. Advance directives are potentially an important mechanism to allow incompetent critically ill patients to identify their health care preferences. Unfortunately, conversations about future medical care and ADs rarely occur in an outpatient setting, and patients enter ICU without prior opportunities to discuss treatment preferences (Reigle, 1992).

• Advance directives and informed decision making. Advance directives are intended to promote patient autonomy and self-determination by allowing individuals to indicate their health care preferences in the event that they become incompetent. Some health care professionals are concerned that when individuals complete an AD they do not have adequate information to make such decisions about their future health care and can therefore not make a reliable, informed decision. Because ADs involve life or death issues, many health care professionals prefer to be overcautious and not withdraw life-sustaining treatment due to concerns about the accuracy of an AD or fear of litigation.

• Withholding or withdrawing treatment. In general, ethicists agree that there is no ethical difference between withholding or withdrawing life-sustaining medical treatments from a critically ill individual (Snider, 1995). Many health care professionals feel more comfortable withdrawing rather than withholding treatments. Withdrawing a medical treatment means that it has been attempted and failed, so the health care professionals can withdraw the therapy with the certainty that it is not beneficial for the patient. When a health care professional withholds medical treatment, they cannot have the same certainty that it will not be beneficial to the patient.

treatment in the critically ill. They suggest that foregoing therapy should be discussed “when the patient has a diagnosis with a grave prognosis, when the burdens of therapy outweigh the benefits, and when the quality of the patient’s life is expected to be unacceptable to the patient” (Task Force on Ethics of the Society of Critical Care Medicine, 1990, p.1435). They indicate that treatments which offer no benefit and serve to prolong the dying process should not be employed and that health care professionals have no obligation to offer, begin, or maintain treatment which is considered futile.

- **The concept of futility.** Historically, health care professionals have been taught how to care for patients but are often unclear on when it is appropriate to change from providing aggressive treatment to compassionate, palliative care. Health care professionals can be uncomfortable with the concept of futility because it implies that any further medical care, and therefore their training and skills, would no longer be beneficial to the patient. Health care professionals often find it difficult to determine when a treatment becomes futile because of different concepts for quality of life, different opinions on prognosis, and different definitions for futility. The question “when does a patient become hopelessly ill without any realistic chance of recovery?” can be difficult to answer with certainty.

- **Economic considerations of advance directives.** Society has a legitimate concern about the cost of life-prolonging therapies and “futile” treatments. While economic considerations should not be the basis for health care decisions, they do present valid concerns. Escalating health care costs from advancing technologies, the aging population, and diminishing health care funds must be considered.

    Some American studies have found that patients with documented ADs have 35 to 68 per cent lower mean terminal hospitalization charges and spend significantly less days in ICU than those without advance directives (Chambers, Diamond, Perkel, & Lasch, 1994; Weeks, Koford, Wallace, & Welch, 1994). These studies imply the potential for an enormous cost savings to Canadian society. If more patients completed ADs, significant health care dollars could be saved by preventing inappropriate and unwanted life-sustaining treatment. However, the extent of economic benefits from an increase in completion and implementation of ADs will be difficult to document and may in fact prove to be less than imagined.

### The critical care nurse’s role with advance directives

Nurses have the potential to play a crucial role in determining the existence of ADs and acting as the patient’s advocate by upholding patients’ wishes in interactions with the family members and the entire health care team (Singleton, Dever, & Donner, 1992). Nurses can also have a major role in educating patients and family members about ADs because they are often more readily available and accessible than other health care team members.

“Ideally, physicians and nurses in outpatient areas will begin to initiate discussions about ADs with patients during routine physical examinations or during office visits” (Martin & Vitello, 1992, p. 17A). “However, a window of opportunity for discussions about ADs may arise after the patient’s condition has improved and before transfer from ICU” (Dooley & Marsden, 1994, p. 342).

Nurses have a moral obligation to ensure that a patient receives accurate information about the purpose, advantages, and limitations of ADs (Reigle, 1992). It has been proposed that critical care nurses should be responsible to inquire about the presence of ADs (Reigle, 1992). However, one 1994 study found that nurses believed they neither had the necessary knowledge about ADs nor the time to have meaningful discussions with their patients (Silverman, Fry, & Armistead, 1994).

White (1997) explored the role of critical care nurses in counseling families about ADs. She suggests that critical care nurses alert the family to the existence of ADs, describe the role that he or she has seen ADs play in patients’ care in the ICU, share observations of the patient’s current experience and his or her sense of the range of experiences that the patients might have in the future, encourage the family to discuss ADs with the patient’s physician, and encourage the family of a patient with a good prognosis to think about how to raise the issue of ADs in the future (White, 1997). However, White also identifies that the critical care nurse must refrain from giving any advice about the manner in which an AD should be interpreted.

Nurses should explore their own feelings and attitudes toward death and quality of life, then identify their own personal, religious, and professional views about ADs (Anderson, Gladue, Laurie, Skotniski, & Tramer, 1991). “When any health care professional has conscientious objections to directives that run counter to their personal code

### Table Two: Basic information about advance directives

**Provincial legislation may vary, but in general:**

- ADs do not apply to individuals who are unable to appreciate the nature and consequences of their decisions (e.g. children under 16 years of age or mentally disabled individuals)

- ADs should be signed by the individual, dated, and witnessed by two adults or notarized (witnesses should be people not related to the individual, who will not benefit from the death of the individual, and who are not the appointed proxy)

- A proxy must be an adult and cannot be a member of the attending health care team

- The AD only becomes effective when the patient is incompetent to make health care decisions

- A copy of the AD should be placed in a prominent location of the patient’s chart and all those involved with the care of the patient should be made aware of its existence

- An advance directive may be revoked verbally or in writing at any time
of values, they have the right to withdraw from non-emergent cases and transfer the care of the patient to another provider” (Kelner et al., 1993, p.1336).

Preparing an AD for oneself can be an effective method for nurses to explore the issue of ADs (Reigle, 1992). Ethics committees and professional associations can provide further information and guidance about ADs (Anderson et al., 1991). A large amount of information relating to advance directives is available from journal articles, texts and the internet. The University of Toronto Joint Centre for Bioethics provides a description of advance directives with instructions on how to complete an advance directive and a sample advance directive through the internet at “http://www.utoronto.ca/jcb”. (Also, refer to Table Two.)

**Conclusion**

The incidence of ADs in Canadian health care will invariably increase. There is a great need for both public and professional education. Critical care nurses must be prepared to take an important role to support and promote the use of ADs by educating themselves, their patients, and their patients’ family members.

**About the author**

Beverly Leith, RN, BScN, CNCC(C) is an MICU/CCU staff nurse at the Health Sciences Centre in Winnipeg, Manitoba. She is currently completing her Masters of Nursing degree at the University of Manitoba.

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Awards available to CACCN members

COBE Chapter of the Year Award Program

Purpose

To recognize the effort, contributions and dedication of a chapter of CACCN in carrying out the purposes and goals of the association.

A. Criteria for the award program
1. The award program will be for the period of July 1 to June 30 each year.
2. Chapters may win the award for one year followed by a two-year lapse before entering again.
3. A point system has been developed to evaluate chapter activities during the year. The chapter with the most points will be the winner of the Chapter of the Year Award. CACCN reserves the right to adjust points depending upon supporting materials submitted.
4. The award winner will be announced at Chapter Connections Day.

B. Conditions for the award program
1. All chapters of CACCN are eligible to participate provided they have on file at national office all of their financial (quarterly) and activity (bi-annually) reports required for the qualifying period.
2. Chapters must submit their entry form and accompanying binder to national office postmarked by July 31.
3. Each chapter is required to record their activities and their total points awarded for each activity.
4. Supporting materials are to be typewritten in a looseleaf binder, separated by category.
5. Each entry must contain a cover sheet listing the points accrued in each category and the total overall points.

If the above conditions are not met, the entry will be disqualified.

The winning chapter will receive a plaque and cheque for $500.00 that will be presented at that year’s Dynamics. Announcement of the winner will be published in CACCN publications. The winning chapter’s binder will be displayed at that year’s Dynamics.

C. Categories and their corresponding points
1. List the educational programs, with an accompanying brochure or pamphlet, that occurred during the period of July 1 - June 30.
2. Submit the minutes of business meetings held during the qualifying period.
3. Provide member attendance sheets for each program and/or meeting, and calculate your points based on percentage of members that attend the program and/or meeting out of the total chapter membership.
4. Submit a list of new members recruited from July 1 to June 30 during the qualifying period, and include national CACCN membership numbers. Calculate your points based on the percentage of new members recruited as compared to the total membership of July 1 (prior to the qualifying period). The points will be calculated on a percentage of money spent on that member in relation to the current financial account at that time. Please provide proof that the member attended.
5. List projects that provide public education, community service and/or promote the image of critical care nursing. These projects must be presented under the auspices of the CACCN chapter.

In the case of a tie, CACCN reserves the right to determine the winner.

Good luck in your endeavours!

Johnson & Johnson Editorial Awards

The award

The Johnson & Johnson Editorial Awards will be presented to the author(s) of two written papers in the Official Journal of the Canadian Association of Critical Care Nurses which demonstrate the achievement of excellence in the area of critical care nursing. A $750.00 award will be given to the author(s) of the best article, and $250.00 given to the author(s) of the runner-up article. It is expected that the money will be used for professional development. More specifically, the funds...
 must be used by the recipient:
1) within 12 months following the announcement of the winners, or within a reasonable time;
2) to cover and/or allay costs incurred while attending critical care nursing-related educational courses, seminars, workshops, conferences or special programs or projects approved by the CACCN, and
3) to further one’s career development in the area of critical care nursing.
Eligibility
1) The author(s) is a Canadian citizen
2) The primary author(s) is an active member of the Canadian Association of Critical Care Nurses (minimum of one year).
3) The author(s) is prepared to present the paper at Dynamics of Critical Care (optional).
4) The paper contains original work, not previously published by the author(s).
5) The award may not be presented to the same author(s) two consecutive years.
6) Members of the CACCN board of directors, awards committee or editorial committee of OJCACCN are excluded from participation in these awards.
Criteria for evaluation
1) The topic is approached from a nursing perspective.
2) The paper demonstrates relevance to critical care nursing.
3) The content is readily applicable to critical care nursing.
4) The topic contains information or ideas that are current, innovative, unique and/or visionary.
Style
The paper is written according to the established guidelines for writing a manuscript for the Official Journal of the Canadian Association of Critical Care Nurses.
Selection
1) The papers are selected by the awards committee in conjunction with the CACCN Board of Directors.
2) The awards committee reserves the right to withhold the awards if no papers meet the criteria.
Presentation
1) The awards are presented by a representative of Johnson & Johnson at the Dynamics of Critical Care Conference.

SIMS Educational Awards
The CACCN Educational Awards have been established to provide funds ($750.00 each) to assist critical care nurses to attend continuing education programs at the baccalaureate and masters nursing levels. All critical care nurses in Canada are eligible to apply, except members of the CACCN Board of Directors and the awards committee.

Criteria for application
• be a Canadian citizen
• be an active member of CACCN in good standing for a minimum of one (1) year.
• demonstrate the equivalent of one (1) full year of recent critical care nursing experience in the year of the application.
• be an active member (minimum of one [1] year) of a CACCN committee(s) and/or participate in other chapter-related activities. Past participation is acceptable.
• submit a letter of reference from his/her current employer.
• be accepted to an accredited school of nursing or recognized critical care program of direct relevance to the practice, administration, teaching and research of critical care nursing.
• contribute to CACCN in return for the award - successful applicant must submit an article to the official journal within the next year after receipt of the award.
• incomplete applications will not be considered; quality of application will be a factor in selecting winners.
Application process
• submit completed CACCN educational award application forms to national office (forms package can be requested from national office).
• obtain a minimum of 200 merit points (preference will be given to members with the highest number of merit points).
• keep a record of his/her own merit points (form included in forms package).
• submit all required documentation outlined in criteria - candidate will be disqualified if documentation is not submitted with application.
Post-application process
• all applications will be acknowledged in writing from awards committee.
• unsuccessful applicants will be notified individually by awards committee.
• winners will be acknowledged at Dynamics of Critical Care and published in the official journal.
Deadlines for receipt of applications in national office are: September 1 and January 31 of each year.

INTRODUCING...
The Johnson & Johnson Innovative Project Award
The Johnson & Johnson Innovative Project Award will be presented to a group of critical care nurses who develop a project that will enhance their professional development. The primary contact person for the project must be an active member of CACCN (for at least one year). Applications must be received in CACCN national office on or before July 1. Presentation of the award will be made at Dynamics.

Applications will be judged according to the following criteria:
1) the number of nurses that will benefit from the project,
2) the uniqueness of the project, and
3) the relevance to critical care nursing.
Within one year, the winning group of nurses is expected to publish a report that outlines their project in the Official Journal of the Canadian Association of Critical Care Nurses. Do you have a unique idea?

Recognition, Recruitment and Retention Award

This CACCN initiative was established to recognize members and the chapters for their outstanding achievements with respect to recruitment and retention. Individual members will be recognized for long-standing service to the association as well.

Recognition Initiative
Members will receive recognition at Dynamics for their long service to CACCN. This will be in the form of a pin that will be given to people with five years, 10 years, 15 years and 20 years of continuous membership in the association. Membership must be renewed within a two-month window in order to qualify for continuous membership. Note: In the new national membership database, all members’ “date of joining” is March 1996 or after.

In addition, new members from the previous 12 months prior to each Dynamics will be given ribbons on their name tags if they attend Dynamics that year.

Recruitment Initiative
This initiative will benefit the chapter if the following requirements are met:
• If the chapter recruits 25-49 new members from February 1 to January 31 of the next year, they receive one full tuition to Dynamics of that year.
• If the chapter recruits 50-100 new members from February 1 to January 31 of the next year, they receive one full tuition plus $100.00 to Dynamics of that year.

Retention Initiative
This initiative will benefit the chapter if the following requirements are met:
• If the chapter has 100% renewal of its previous year’s members, the chapter will receive $250.00.
• If the chapter has greater than 80% renewal of its previous year’s members, the chapter will receive $150.00.
• If the chapter has greater than 60% renewal of its previous year’s members, the chapter will receive $100.00.
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HILL-ROM INTRODUCES THE TOTALCARE™ BED SYSTEM

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• The Shearless Pivot™ patient position mechanism which combines articulation of the frame, surface and patient
• The FlexAfoot™ retractable foot mechanism to customize the overall length of the bed system
• The OneStep™ siderail release mechanism

The TOTALCARE™ Bed System - designed to help you provide the ultimate care!

Hill-Rom® CANADA

For more information, contact your local Hill-Rom representative or call 1-800-267-2337.