Perinatal Outreach Program of Southwestern Ontario

Partner

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Analgesia in Labour

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Introduction

ANALGESIA for labour and delivery is now safer than ever. Anesthesia-related maternal mortality has decreased from 4.3 per million live births during 1979-1981 to 1.7 per million live births during 1988-1990. The increased use of regional anesthesia for the parturient is partially responsible for this decrease in mortality (1). The goal is to provide labour analgesia, not anesthesia. Analgesia is the absence of sensation to pain, whereas anesthesia is the absence of all sensation.

Safety is the first and foremost goal of obstetrical anesthesia. For labour analgesia, a secondary goal is to minimize or eliminate maternal lower extremity muscle weakness associated with epidural and subarachnoid local anesthetics. The purpose of this article is to review analgesic techniques and medications that are currently used to provide intrapartum analgesia.

Pain in labour

During the first stage of labour, pain primarily results from dilation of the cervix and distention of the lower uterine segment, which occurs with uterine contractions. These pain impulses are transmitted by means of afferent A-delta and C fibers, which are visceral afferent nerves that accompany the sympathetic nerves and enter the spinal cord at T10 to L1. The visceral pain of uterine contractions is described as dull and aching; although severe, it is poorly localized by the patient. During the second stage of labour, pain results from distention of the pelvic floor, vagina and perineum. Pain impulses are transmitted to the spinal cord by means of somatic nerve fibers that enter spinal cord at S2to S4. Somatic pain is transmitted by rapidly conducting fibers that are more difficult to block. The pain is sharp and well localized by the patient.

Parenteral Opioids

Opioids are the most widely used systemic medications for labour analgesia. These drugs allow the parturient to better tolerate the pain of labour, but typically they do not provide complete analgesia. (Cont'd)

What's Inside…

Systemic opioids carry significant maternal side effects (i.e. nausea, vomiting, delayed gastric emptying, dysphoria, drowsiness, hypoventilation) and the potential for adverse neonatal effects. Opioids may be given subcutaneously, intramuscularly, or intravenously, either intermittently or by continuous infusion. Subcutaneous and intramuscular injections have the advantage of simplicity although, of course, are more painful. The growing popularity of Patient Controlled Analgesia (PCA) for postoperative pain relief has prompted the use of this technique for labour analgesia. PCA offers an attractive alternative for labour analgesia in hospitals in which epidural anesthesia is contraindicated or unsuccessful. The mother can tailor the administration of analgesia according to her individual needs and metabolism.

Meperidine (Demerol)

Meperidine (pethidine) is the opioid still most widely used for labour analgesia in many parts of the world. Meperidine is a synthetic opioid that readily crosses the placenta by passive diffusion. The usual dose is 25 to 50 mg intravenously or 50 to 100mg intramuscularly every 2 to 4 hours. The onset of analgesia is within 5 minutes after intravenous administration and within 45 minutes after intramuscular administration.

The timing of administration of meperidine affects the risk of neonatal depression at birth. Maximal fetal tissue uptake of meperidine occurs approximately 2 to 3 hours after maternal administration. Thus, from the pharmacologic standpoint, the best timing for birth after maternal administration of meperidine would be within the first hour or more than 4 hours after a single dose. Studies have shown that infants born 2 to 3 hours after maternal administration of meperidine have an increased risk of respiratory depression (2,3). Meperidine is metabolized in the liver to noremeperidine, meperidic acid, and normeperidic acid. Normeperidine is a pharmacologically active metabolite that is a potent respiratory depressant. Normeperidine also crosses the placenta. In addition, maternally administered meperidine is metabolized by the neonate, who produces a significant amount of noremeperidine. The half life of noremeperidine in the neonate is approximately 60 hours (4). Like other opioids, meperidine may cause decreased fetal heart rate (FHR) variability. The maximum effect on FHR variability occurs 25 minutes after intravenous administration and 40 minutes after intramuscular administration. Variability of the FHR typically recovers within 60 minutes (5,6).

Morphine

The usual dose for maternal analgesia is 2 to 5 mg intravenously or 5 to 10 mg intramuscularly. The onset of analgesia is within 3 to 5 minutes after intravenous administration and within 20 to 40 minutes after intramuscular administration. The duration of action is quite long at 4 to 6 hours. Morphine is conjugated in the liver to one of two metabolites: Morphine-3-glucuronide is pharmacologically inactive, whereas morphine-6-glucuronide produces analgesia and depression of ventilation (7). Elimination of morphine glucuronides may be impaired in patients with renal failure, causing an accumulation of metabolites and unexpected ventilatory depressant effects of small doses of opioids.

Currently morphine is administered infrequently during labour and vaginal delivery.

Fentanyl

Fentanyl is a highly lipid-soluble, highly protein-bound synthetic opioid with an analgesic potency 75 to 100 times that of morphine and 800 times that of meperidine. Its use for obstetric analgesia/anesthesia as an alternative to epidural analgesia developed because of its rapid onset, short duration of action, and lack of active metabolites. Because of its lipid solubility, fentanyl crosses biologic membranes (e.g., placenta) rapidly.

Morley-Forster and Weberpals (8) retrospectively reviewed the outcomes of 32 neonates whose mothers received PCA fentanyl during labour. Only three infants required naloxone but fourteen (44%) of the infants had a 1-minute Apgar score of less than 6. At five minutes, all infants, except those who had required naloxone, seemed normal with an Apgar score greater than 7. Gestational age, birth weight, method of delivery, PCA duration, time from last dose to delivery, and dose and rate of fentanyl

infusion were not predictive of low 1-minute Apgar scores. However, in the subgroup given naloxone, there was a higher total maternal fentanyl dose (770 +/- 233 mcg vs. 300 +/- 287 mcg, p=0.027).

In our institution at St. Joseph' Health Care, we provide PCA fentanyl with an initial loading dose of 50 mcg IV, followed by a dose of 10-20 mcg, lockout time of 6 minutes, and an hourly maximum of 100 mcg. A basal rate of 10 mcg per hour adds greater comfort.

Nalbuphine (Nubain)

Nalbuphine is a mixed agonist/antagonist opioid analgesic. Nalbuphine and morphine result in similar respiratory depression at equianalgesic doses. However, nalbuphine demonstrates a ceiling effect for respiratory depression with increasing doses. Maximal respiratory depression occurs with a 30 mg dose in the average adult. Nalbuphine results in no further increase in respiratory depression with doses greater than 30mg IV.

Nalbuphine is approximately 0.7 to 0.8 times as potent as morphine for the relief of acute pain (9). The usual dose is 10 to 20 mg every 4 to 6 hours. The onset of analgesia occurs within 2 to 3 minutes after intravenous administration and within 15 minutes after intramuscular or subcutaneous administration. The duration of analgesia ranges from 3 to 6 hours. Nalbuphine is associated with less maternal nausea and vomiting than meperidine but produces more maternal sedation and dizziness.

Non-steroidal Anti-inflammatory drugs

Ketorolac is a prostaglandin synthetase inhibitor that is most often administered for postoperative analgesia. In theory, ketorolac may suppress uterine contractions and may cause closure of the fetal ductus arteriosis leading to pulmonary hypertension. [**prostaglandin synthetase inhibitors should not be used for management of intrapartum pain**] There are few data on the administration of ketorolac during labour.

Nitrous Oxide Analgesia

Intermittent inhalation of nitrous oxide has been used for more than 100 years to provide safe analgesia for labour, but is not a particularly effective analgesic. When properly applied, inhalation of 50% nitrous oxide: 50% oxygen (Entonox) may provide significant pain relief in as many as 90% of parturients (10). Nitrous oxide does not interfere with uterine activity (11). To achieve substantial pain relief with nitrous oxide, maternal cooperation is required. The patient is encouraged to breathe the mixture of 50% nitrous oxide in oxygen from 30 seconds prior to the contraction and to continue until the end of the contraction. Peak concentrations in the alveoli occur 60 seconds after the start of administration. An apparatus that limits the concentration of nitrous oxide (e.g. a nitrous oxide/oxygen blender or a premixed 1:1 cylinder) is required and must be checked periodically to prevent the unintentional administration of a high concentration of nitrous oxide and a hypoxic concentration of oxygen.

A recent survey from Ontario found that nitrous oxide analgesia was available in 75% of hospitals. Hospitals without the availability of epidural analgesia were more likely to have nitrous oxide analgesia than those with epidural analgesia (89% versus 70%) (12).

It is important to remember that when using nitrous oxide, you should maintain a well-ventilated area to allow safe use. According to the National Institute For Occupational Safety and Health (NIOSH), it is recommended that waste anesthetic exposure should not exceed a time weighted average over 8 hours of 25 ppm. (13)

Epidural and spinal analgesia / anesthesia

Epidural analgesia is the most effective method of intrapartum pain relief in current practice (13). Epidural analgesia provides excellent analgesia in the majority of labouring women (14) and reduces maternal plasma concentration of catecholamines. Decreased alpha-and beta-adrenergic receptor stimulation may result in improved uteroplacental perfusion and more effective uterine activity. (15, 16)

There are a number of problems with labour epidural analgesia that have prompted concern and investigation. First, the time from epidural catheter placement until the patient is comfortable is variable, but depending on

the local anesthetic used, can take up to 30 minutes. Other disadvantages of labour epidural analgesia include maternal hypotension, inadequate analgesia (10-12%)(17) and lack of ambulatory ability with motor block, even with the very dilute local anesthetic solutions (18).

Subarachnoid opioid offers rapid, intense analgesia with minimal changes in blood pressure (19) or motor function (20). The opioid is usually administered as part of a combined spinal epidural (CSE) technique.

Patient controlled epidural analgesia (PCEA) is growing in popularity. This technique allows the patient to self-medicate, thereby controlling her own analgesia. Compared with continuous infusion or intermittent bolus techniques, PCEA is associated with fewer anesthesiologist interventions and less motor block (21, 22). When using Bupivaciane 0.125% and Fentanyl 2 mcg/ml the usual parameters are Dose: 4-5 mL, Lockout 15 minutes, Basal: 5 mL/hr, and total hourly dose of 25 mL.

In summary, techniques and drugs available for the pain of labour are vastly superior to what existed previously. The future of obstetric anesthesia lies in making analgesic techniques safer, more effective and more widely available.

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Hyperemesis Gravidarum: In-Hospital Treatment

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NAUSEA AND VOMITING OF PREGNANCY (NVP) is the most common medical condition in pregnancy affecting 50-90% of pregnant women [1]. Hyperemesis gravidarum (HG) is the extreme of NVP affecting 5 in 1000 pregnancies. It is defined as intractable vomiting associated with electrolyte imbalances, ketosis and a pre-pregnancy weight loss of greater than 5% [2]. Prior to modern medicine, HG resulted in high rates of maternal mortality. Fortunately, today this disorder rarely causes death, however it still has a significant impact on a woman's quality of life during her pregnancy and in severe cases can also affect fetal health. The exact etiology for this disorder is still unknown. Although guidelines for treating NVP have been created, a standard protocol for patients requiring admission to hospital has not yet been established.

The clinical coarse of HG begins at 5 to 6 weeks of gestation and peaks at approximately 9 weeks [2]. Symptoms usually resolve by 16 to 18 weeks. However, in approximately 5% of cases symptoms will continue until term [2]. Also severe HG, if not treated can result in severe complications such as Wernicke's encephalopathy and central pontine myelinolysis [3]. HG also has been associated with negative psychological effects including isolation and depression. In extreme cases, terminations for wanted pregnancies have been requested [1]. As well, loss of a woman's work productivity is common with 30-50% of women reporting decreased job efficiency, loss of work time and loss of house-work and child rearing time. In terms of fetal outcome, the prognosis is positive. Several studies have shown no differences in birth weight or mean gestational age at birth between those with this disorder and those without. However, those women who are refractory to standard treatment and require multiple hospital-izations do have difficulty catching up in terms of weight gain and have an increased incidence of infants whose weights are under the 10th percentile for gestational age [4].

A specific stimulus for the etiology of HG has not been identified and at present a multifactorial etiology is favoured. Human chorionic gonadotropin (HCG) has been linked to HG for several reasons. Firstly, there is a close temporal association between the peak of HCG production in pregnancy and the peak of symptoms. As well, certain conditions with increased levels of HCG production such as multiple gestations and gestational trophoblastic disease are more likely to be associated with severe NVP [2]. More recently increased rates of seropositivity for the bacteria, Helicobacter pylori has been demonstrated in patients with HG. It is suggested that it should be considered as a cause in refractory cases [6].

It is important to remember that HG is a diagnosis of exclusion. There are many other disorders that present similarly to HG and it is imperative that they be ruled-out. The differential diagnosis includes GI disorders (i.e. hepatitis, cholelithiasis), metabolic problems (i.e. diabetes, parathyroid disease), neurologic disorders (i.e. migraines, tumour), drug toxicity, infection or psychiatric illness. At the time a patient presents, there are

several standard investigations required to help determine the cause of her symptoms as well as the severity of her vomiting. Patients with HG may have hypokalemia, metabolic alkalosis, abnormal liver function tests (LFT) (usually in the 100s) and hyperbilirubinemia. As well, due to hemoconcentration from fluid loss, the hematocrit will be elevated.

In most cases, patients respond to re-hydration and a short period of gut rest followed by re-introduction of a diet rich in carbohydrates and low in fat. Advice has traditionally revolved around dietary changes such as small and frequent meals. However, evidence has not substantiated its success. As well, avoidance of environmental triggers such as specific odours, heat, humidity, visual or physical motion have been recommended. Although many non-pharmacological treatments have minimal evidence to support their efficacy, it is important for health practitioners to be aware of what patients are using. Ginger, a common spice found in food and beverages, in doses of 1000mg/day is used in many cultures for nausea and vomiting. Small randomly controlled trials have shown a clinical significant improvement in those using ginger versus placebo [7]. Patients should be cautioned that ginger is a non-regulated food product and most preparations are of uncertain purity and composition. Some women resort to acupuncture and acupressure even though its efficacy is difficult to prove. However, acupuncture is safe and there is no theoretical concern regarding its safety with regards to the fetus.

In cases when nausea and vomiting are severe or conservative measures have been tried and failed, pharmacological intervention is warranted. As of yet, no medication for HG has been approved by the U.S Food and Drug Administration. This is most likely an after effect of the thalidomide tragedy. A case in point is when Bendectin (a drug created for HG) was removed in 1983 due to legal costs based on teratogenicity claims, which were subsequently proven to be unsubstantiated [8]. Hospitalizations for NVP increased significantly after this drug was removed from the market and an estimated 20 million dollars in hospital care could have been saved [8]. In Canada, Diclectin, a combination of Doxylamine and pyridoxine, was introduced in the early 90s with excellent clinical results. As its use increased, hospitalization in this country decreased. Today, Diclectin is readily prescribed and it is considered the standard drug for NVP since it has the greatest evidence to support its efficacy and safety.[7] It is made up of Doxylamine, an H1 receptor antagonist and pyridoxine, vitamin B6. Currently, the maximum dose recommended is 4 tabs/ day, however 5 to 8 tabs are safely being prescribed and is starting to be used for patients with a larger body mass index.

When oral intake is not tolerated, Diclectin cannot be continued or initiated. In these cases, the vomiting is most likely severe enough that dehydration has become an issue and the patient requires admission to hospital for rehydration. This is accomplished with IV crystalloid fluids to replenish estimated fluid loss as well as to continue maintenance requirements. Vitamins, electrolytes and minerals also need to be replenished at this time. In patients who have been vomiting for more than 3 weeks, thiamine needs to be given to prevent neurological sequelae. In most patients, symptoms will improve within 1 to 2 days of rehydration.

There are many other safe anti-emetic drugs that are beneficial at the time of hospital-ization if Diclectin is not tolerated. Dimenhydrate (Gravol) is available in both parenteral and suppository formulations; it works well for treating acute or breakthrough episodes of NVP. Its effects start within 1 hour and lasts for 4 to 6 hours [7]. Caution needs to be taken, however, in preventing anticholinergic side effects. Once a patient is taking a full dose of Diclectin, the Gravol dose should not exceed 200 mg per day [7]. Phenothiazines (phenergan) are dopamine antagonists and can also be taken parenterally. Several RCTs have demonstrated positive therapeutic effects [7].

In cases when symptoms are not completely resolved, other adjuvant anti-emetics may be added to the regimen. Metoclopromide (Maxeran) is an upper GI motility stimulant used extensively in Europe. It can be administered orally or parenterally and is helpful when oral fluids and solids are reintroduced. Odansetron is a seratonin 5-hydroxytryptophan antagonist used extensively in those with severe nausea and

vomiting particularly in patients undergoing chemotherapy. Evidence for its efficacy and safety in pregnancy is not established and it is significantly more expensive. Until further studies prove its benefit, it should not be advocated for first line use [7]. Corticosteroids are currently being suggested with severe and refractory cases although there are no controlled studies to demonstrate its effectiveness in HG. A recent study comparing Prednisone to placebo did not show a clinically significant difference in response [9]. Currently, guidelines state that steroids should be avoided under 10 weeks gestation[9].

Esophageal reflux is commonly associated with NVP and can severely affect a woman's quality of life. Antacids and H2 receptor antagonists can be used at recommended doses and have been shown not to be teratogenic to the fetus. Proton pump inhibitors have limited studies and are currently not recommended. In patients who continue to be refractory despite maximum treatment, parenteral nutrition may be required to provide necessary calories for fetal energy, balance and growth [2]. For long-term treatment, central access is preferred. As well, other etiological causes should be sought and investigations including H. pylori serology, abdominal ultrasound or thyroid levels need to be performed.

The group Motherisk (Hospital for Sick Children, Toronto) acts as a counseling service to women and health professionals concerning drugs, chemicals and infections during pregnancy and lactation. They have produced a protocol for NVP [8]. The following table is to be used to investigate and manage women who require admission to hospital with hyperemesis gravidarum.

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In-Hospital Management for Hyperemesis Gravidarum

When to admit to hospital

- Dehydration
- Unable to tolerate fluids
- Electrolyte imbalance

Admission

- Complete blood count
- Electrolytes
- Liver function tests
- H. pylori (if persistent nausea and vomiting)
- Obstetrical Ultrasound

Fluid Resuscitation

- NPO
- IV crystalloid (Normal Saline, Ringers Lactate)
- 1-2 litres to replenish losses
- Maintenance rate 125-150 cc/hr

+

Electrolytes and Vitamins

- KCL 20-40 meq/L PRN (maintenance)
- Multivitamin 1 amp daily
- Thiamine if vomiting >3 weeks
- Repeat blood work 24 hrs post rehydration

Anti-emetics

- Gravol 50-75 mg IV/PR q 4-6h or
- Phenergan 10-25 mg IV q4-6h

and/or

- Maxeran 5-10 mg IV q 8h
- Stemetil 5-10 mg IM q8h

Anti-reflux

- Ranitidine 50 mg IV q8h or 150 mg po bid

and/or

- Diovol 30-60 ccs po bid

No or minimal improvement

- Odansetron 4-8 mg IV q12h

Improvement

- Re-start oral fluid intake
- Reduce Gravol dosage
- Switch to Diclectin 1 in am, 1 ac meals and 2 at night
- Slowly introduce bland solids

No improvement

- No nutritional intake > 1 week, consider parenteral nutrition
- Investigate other causes, ie. H. pylori, psychiatric, neurological disorders

YOU ASKED US:

Q:... The Perinatal Outreach Program has recently become aware that there seems to be confusion regarding the management of women with GBS bacteriuria. Some family physicians seem to be reluctant to treat it antenatally, and the new SOGC guidelines on GBS only address intrapartum management. What is the appropriate course of action?

A:... The Centers for Disease Control (CDC) recommend that women who are pregnant should have a urine culture done in early pregnancy to determine if there is GBS in their urine, as GBS bacteriuria can increase the risk for preterm labour and transmission of the bacteria to the baby. Providers need to remember to note on the laboratory requisition that the woman is pregnant, also any drug allergies she might have. Any growth of GBS in the urine, regardless of symptoms, should be treated with the appropriate antibiotics, and then a urine culture done after the completion of the course of treatment to ensure that the treatment was effective. Women who have had GBS bacteriuria will require intrapartum antibiotic prophylaxis and do not need to be swabbed at 35 – 37 weeks gestation, as GBS in the urine is a marker for heavy colonization. Even with antibiotic therapy during pregnancy, recolonization is likely to occur and these women are assumed to be GBS positive at the time of birth.

Reference

Prevention of Perinatal Group B Streptococcal Disease. MMWR Morb Mortal Wkly Rep 2002;51:RR-11.

Resources for care providers

Centers for Disease Control:
2002 GBS Guidelines

- http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5111a1.htm

CDC'S GBS Internet Page:

- http://www.cdc.gov/groupbstrep

SOGC Information for patients:

- http://www.sogc.org/pub_ed/groupb/index_e.shtml

Upcoming Events:

Mark Your Calendar!

Fetal Health Surveillance Workshop

March 22, 2005
Location: Strathroy Middlesex General Hospital
Contact: Mary Robertson
(519) 245-1550

March 31, 2005
Location: Norfolk General Hospital, Simcoe
Contact: Dodie Trimble
(519) 426-0750

Maternal Newborn Nurse Education Course

London:
Mondays: April 4 – May 16, 2005
St. Joseph's Health Care, London

London:
Mondays: Sept. 12 – Oct. 31, 2005
St. Joseph's Health Care, London

Contact:
Gwen Peterek
Perinatal Outreach Program
Phone: (519) 646-6100 ext 65901
Fax: (519) 646-6172
Gwen.peterek@sjhc.london.on.ca

Regional Nurse Manager's Meeting
(for entire region)
Thursday, Friday, June 2-3, 2005
Location: Windsor Regional Hospital

Contact: Perinatal Outreach Office
(519) 646-6100, ext. 65859

PSYCHIATRIC UPDATE 2005
(focus on women's mental health)
Friday, June 3, 2005
Location: Stoneridge Inn, London

Contact: London Regional Mental Health
Wendy Spenler
(519) 455-5110 x 47555
Julie Franklin
(519) 455-5110 x 47397

9TH Annual Breastfeeding Conference
(Ottawa Valley Lactation Consultants)
June 16-17, 2005
Embassy West Hotel, Ottawa
For more information, contact Sheryl Hamilton
(613) 224-3528 or Sheryl.Hamilton@rogers.com

Type 1 Diabetes

Type 1 Diabetes

Can we protect our children?

Are you or your partner pregnant?

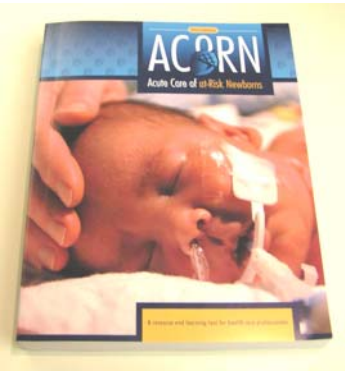
Do you, your partner or any of your children have Type 1 Diabetes?

Recent diabetes research has pointed to a possible link between infant nutrition and the development of Type 1 (insulin–dependent, or juvenile) diabetes in childhood. TRIGR is an international study looking at infant nutrition to find out whether the number of children who develop Type 1 diabetes can be reduced.

For more information please contact:
Lynda Bere,
Study Coordinator,
St. Joseph's Health Care,
London, Ontario.
(519) 646-6000 ext. 65996

We did it!

The new ACoRN Program (Acute Care of Resuscitated Newborns) was launched nationally at the Lamplighter Inn, London, ON on February 14-15, 2005. Congratulations to Jill Boulton and other members of the ACoRN editorial board for their exceptional work in developing this long awaited program and manual. Plans are underway for rollouts in various locations throughout the southwest region. See ***Upcoming Events*** *for* details.



ACoRN REGIONAL ROLL-OUT

Windsor - September 7-8, 2005
Owen Sound - October 5-6, 2005
Chatham – November 3-4, 2005
Stratford – November 17-18, 2005
London - January 19-20, 2006

Contact: Perinatal Outreach Office
(519) 646-6100, ext. 65859
Watch our webpage for further details:
www.sjhc.london.on.ca/sjh/profess/periout/periout.htm

20TH ANNUAL PERINATAL OUTREACH CONFERENCE
"STRADDLING THE FAULT LINE"

Wednesday, September 21, 2005
Location: Lamplighter Inn, London

Contact: Perinatal Outreach Office
(519) 646-6100, ext. 65859
Watch our webpage for further details:
www.sjhc.london.on.ca/sjh/profess/periout/periout.htm

ALARM COURSE

Toronto: November 27-28, 2005
(in conjunction with ON CME)

For more information, contact the SOGC
1-800-561-2416 / www.sogc.org
Or contact Linda Kollesh CME/ALARM Program office at: lkollesh@sogc.com
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