MLT Analyzer
Bulletin of the New Brunswick Society Of Medical Laboratory Technologists

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Contents:

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President's Message

By Bernadette Muise

Ah! Summer is finally here. A time most of us hope to use to re-charge and spend some time on reflection. I suspect there will be little enough "downtime" for anyone this year, so make the most of your vacation, do something "relaxing" not more "chores". As they say "life is short, eat dessert first!"

There has been a lot of activity this spring. Our registrar, Janelle B Whitlock, has met with the Canadian Medical Association at the Assembly of Health Professions meeting in February, she participated in a conference related to Internationally Educated Health Professionals (IEHP) and has attended other regulatory meetings in New Brunswick and Ontario. I also attended a workshop on Internationally Educated Health Professionals. This is becoming a very "hot" topic since the New Brunswick government is committed to increasing population growth through immigration.

As president, I also participated in a Nurse Practitioners summit sponsored by the Nurses Association of NB. It was very interesting to see the challenges they face in being integrated into the health care system. I made a presentation to the bipartisan task force formed by the NB government to reverse the decision to relocate the Canadian Blood Services Center in Saint John to Dartmouth and was included in the face to face meeting between the task force and CBS officials. A letter to the editor was written expressing the NBSMLT belief that this move by CBS could well put patient's lives at risk.

The NBSMLT, met with Roseline Pelletier who is the official responsible for the Agreement on Internal trade amendments. She has assured us that there will be plenty of notice, before any changes need to be made to our legislation, to comply with this

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federal legislation initiative. We have consulted with our legal council who also has told us that we do not need to make changes until the legislation is signed by all parties. At this juncture, our legislation remains the same. The legislation committee has been meeting regularly to study the options available so we can be prepared. Rest assured that the AIT amendment is not designed to lower the standards for technologists employed in New Brunswick and the NBSMLT is committed to ensuring that a lower entry level standard never happens.

The NBSMLT is working hard to increase our visibility within the health care community, and it seems to be paying off, since we are receiving invitations to participate in more collaborative sessions.

Election ballots for President-elect 2010 were mailed the last week of May and there has been genuine interest from the members with 239 ballots returned. The result will be announced at the AGM in October.

The Annual Provincial Scientific Convention to be held in Fredericton in October promises to be an exceptional educational, and social, event. The program is included in this newsletter. **The registration form will be e-mailed to members and available on the Website in mid-August**. If you can, **register early** so that the APSC committee can rest a little easier knowing that the members will come. I look forward to seeing you there.

From the Editor

Janelle B.Whitlock

I am proud to say that this newsletter has an excellent scientific article entitled "DIC– A coagulation nightmare". I would like to take this opportunity to congratulate Nicole Caldwell and thank her for submitting it for publication. You will find a summary of the 2008 annual report in this issue. The complete Annual report is published on the member's only section of the Website. I encourage you to go online and have a look at it; the report outlines the Society's activities during 2008.

At the member's requests, the NBSMLT has prepared a form letter which may be downloaded from the Website for those of you who wish to share your concerns with political official in your riding on the AIT . The sample letter is available on page 10. If you need contact information for MLAs or MPs in your area, please contact me at the office.

LABCON 2009 was a success. There were many participants from New Brunswick and also a few MLTs were speakers at the event. Look for articles from the NBSMLT grant winners in the next issue.

I wish you all a nice summer with your friends and family. Take time to enjoy yourselves!

Next submission date: November 6, 2009

Submit your texts or comments/questions to:

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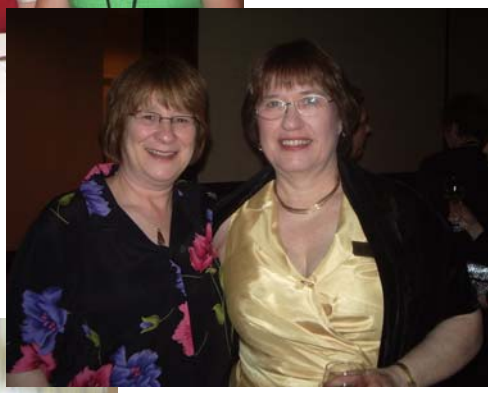
Editorial policy
The purpose of this newsletter is to provide a means of communication between the members of the NBSMLT and its Board of Directors. The opinions expressed in the MLT Analyzer are those of the contributors and do not constitute official policy of the NBSMLT. The editor reserves the right to edit submissions as required.

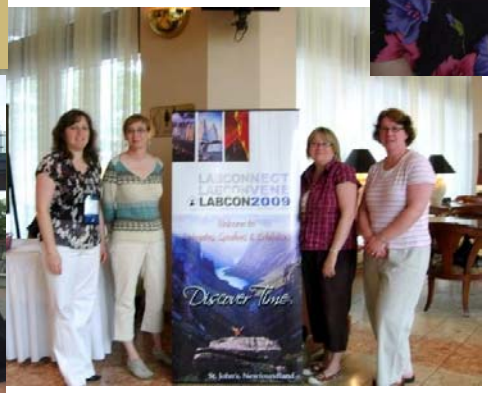
LABCON 2009

St John's Newfoundland

New Brunswick participants:

Lucille Griffin (provincial Vet lab)
Anne Galloway
Mary Hamilton
Coral MacRae
Susan Findlater
Pierre Leveille
Randi Hayes
Robert Adam
Christine Contant (presenter)
Janet Reid (presenter)
Hope MacKenzie (presenter)
Renelle Brun
Serge Pierre Tremblay
Janelle B. Whitlock
Bernadette Muise
Susan Atkinson
Sylvie LeBreton





2008 Annual Report Summary

- The 2008 Annual General Meeting was held in Edmundston on October 18, 2008. New business included a presentation of three year strategic plan, a discussion on temporary license for new graduates and an overview of the CIHI data collection for health human resource planning.
- The Public Relations chair summarized the two day Marketing and Communication meeting held at the CSMLS head office. Key discussion points included media relations, national med lab week and advocacy.
- The ACR & PP chair was pleased to report that less than 1% of MLTs were non compliant to the PDP regulation. The volunteer committee reviewed 371 applications during the year. It was noted in the report that disorganized submissions were very time consuming and some were sent back to the applicant. The Board of Directors approved a processing fee for applications sent after the October 15th deadline. A fee was also set at $25 for duplicate PDP certificates. Attendance and evaluation templates are available in both official languages on the Website. The committee was commended for their dedication and patience.
- The Legislation committee met twice in 2008 and communicated extensively through e-mail. Some of the items discussed were: the reinstatement process and fee structure, recommendation to review the re-entry guidelines, policy for advertising on the Website and newsletter, numerous position statements, complaint document template (available on Website) and a review of the Terms of Reference.
- The Fredericton Academy celebrated Med Lab Week by having a special lunch and displays in the hospital. The provided grants for Maritech and were busy planning for 2009 APSC.
- The Saint John Academy held two meetings. They awarded two one hundred dollar grants to two technologists for CE. Various activities for med lab week were held including a fund raiser which was lead by the NBCC SJ students to purchase nets for malaria prevention in Africa. Two lunch and learns and a teddy bear fair were well attended.
- The Moncton Academy held two meetings. Ten grants were given out for Maritech. There were plenty of activities during Med lab week including a presentation on the International Federation of Biomedical Laboratory Science Congress given by Susan Atkinson.
- The North Shore Academy was not very active in 2008. Members of the executive are encouraging technologists to get involved. Ms. Carlene McCaffrey will be the Director for 2009.
- Edmundston organized a successful one day event for the APSC. They Academy also organized an education day during Med lab week.
- The APSC 2008 took place at Château Edmundston. There was a social event planned on the Friday night followed by a full day of educational sessions. A total of 60 delegates attended the sessions and most of them stayed for the NBSMLT`s AGM.
- The Registrar highlighted some NBSMLT initiatives in 2008. A new type of temporary license called: *Restricted Temporary* is now available for new graduates who fail the national certification exam (at the discretion of the Admission committee). There was a total of 674 practicing members working in New Brunswick in 2008. Twenty three (23) new graduates became licensed to work and an estimated twelve (12) retired from the profession.
- The financial statements indicated a surplus of $12,550 for 2008.

Disseminated Intravascular Coagulation

A Coagulation Nightmare

Submitted by
Nicole Caldwell

The focus of this article is to explain the severe consequences of a coagulation imbalance, and the critical role of the transfusion medicine department in providing a diverse supply of blood products. Included is the case study of a 29 year old postpartum patient who presented in the emergency department with heavy bleeding following the delivery of twins at a neighbouring hospital.

Overview

Disseminated intravascular coagulation (DIC) is a condition which is known under many different names such as disseminated intravascular thrombosis, defibrination syndrome, consumptive coagulopathy, and secondary fibrinolysis. It is difficult to correctly define a condition that varies so greatly in symptoms and severity. A reasonable definition for DIC is an acute or chronic event that complicates a variety of clinical conditions, any of which produces the activation of the haemostatic mechanism, the production of intravascular fibrin, and also secondary fibrinolysis[6].

DIC frequently manifests itself as bleeding, but it will usually begin as a result of an uncontrolled local or systemic activation of coagulation caused by the complication of an underlying disorder[1]. This underlying disorder is the trigger that causes the coagulation imbalance; therefore, when this underlying condition is removed, haemostasis should be restored. Such disorders can include massive bleeds, sepsis, or vascular disorders.

The inciting events are numerous, but generally involve either overwhelming release of tissue factor by cellular, vascular, or hypoxemic injury, or by the presence of endogenously or exogenously derived procoagulant molecules (such as bacterial lipopolysaccharide, proteins produced by neoplastic cells etc.) [1].

DIC is generally due to a pathologic generation of thrombin in the vascular compartment leading to intravascular coagulation with the consumption of coagulation factors, thrombin generation, and secondarily, the consumption of platelets[5]. Severe DIC can lead to widespread tissue hypoxia and multiorgan dysfunction with possible hepatic, neurologic, cardiac, and renal impairment[1]. It should be noted that multiorgan failure is associated with an extremely high mortality rate[1]. On the contrary, some less severe forms of DIC will only produce a slight imbalance of haemostasis, allowing the body to compensate without any clinical symptoms being detected.

In a healthy individual, the body has several mechanisms which maintain a proper balance between clot formation (thrombosis) and clot dissolution (fibrinolysis). Clotting is needed to prevent excessive bleeding when damage occurs, but haemostasis ensures that any unnecessary clots are removed from the circulation. This prevents excessive thrombi formation which could be disastrous by impeding blood flow in the vasculature, and consequently giving rise to organ failure. Loss of blood flow to the organs is one of the causes of morbidity in the most severe cases of DIC[9].

A Review of Coagulation

The pathogenesis of DIC is based on tissue factor-mediated initiation of systemic coagulation activation that is insufficiently contained by physiologic anticoagulant pathways and amplified by impaired endogenous fibrinolysis[9]. The normal response to tissue damage is a contained thrombin generation at the site of injury, which results in the coagulation of blood on the surface of damaged microvessels in order to stop the blood loss[2].

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In DIC, there is an unregulated thrombin explosion that causes the release of free thrombin into the circulation[2]. In an attempt to maintain vascular patency, the body releases excess plasmin so that systemic fibrinogenolysis as well as local fibrinolysis can occur and remove excess thrombi[2]. It is the uncontrolled generation of both free thrombin and plasmin within the circulation that lead to the clinical features of DIC, both of which are responsible for the thrombotic and haemorrhagic manifestations, respectively[2].

In normal coagulation, the extrinsic coagulation pathway is activated following vascular endothelial cell injury. Such an injury causes the exposure of tissue factor which is a receptor protein on the cells that underlie the endothelium[4]. Tissue factor and plasma factor VII bind to form a TF:VII complex, which is already enzymatic, but becomes even more enzymatic when factor VII becomes activated by factors such as IXa and Xa to form a TF:VIIa complex[4]. TF:VIIa along with Ca^{2+}, activates factor X to the serine protease Xa which initiates the common pathway[4]. In the common coagulation pathway, factor Xa along with a cofactor, Va, lipid, and Ca^{2+} converts prothrombin (II) into thrombin (IIa) [4]. Thrombin then converts fibrinogen to fibrin which is then stabilized by factor XIII[4].

The body's natural response to control this thombin explosion is the activation of the fibrinolytic pathway. Plasmin is the molecule involved in the fibrinolytic coagulation pathway which causes clot dissolution. This pathway is usually activated once the injury has healed in order to restore proper blood flow. Damaged tissue releases a substance called a plasminogen activator which activates the inert and circulating precursor plasminogen[4]. Plasminogen is normally found in plasma, and is adsorbed by fibrin which allows plasminogen to be automatically incorporated into the fibrin clot[4]. Haemostasis is therefore maintained by allowing a condition that triggers fibrin formation to also initiate plasmin formation. The activated form of plasminogen is called plasmin which degrades fibrin as well as factors I, V, VIII and XIII which are also part of the fibrinogen group of factors[4]. These are the most labile factors consumed in coagulation, and they are the only group of factors that act as a substrate for the enzyme plasmin[4].

Evidently, DIC lacks the checks and balances that normally regulate thrombin and plasmin; therefore, both these powerful proteolytic ezymes meet little resistance to their lytic attack on whatever factor VIII, factor V, fibrinogen and platelets that survive the coagulation process[5]. All major natural anticoagulant pathways (i.e. antithrombin III, the protein C system, and tissue factor pathway inhibitor) seem to be impaired[9]. Antithrombin III is probably the most important inhibitor of thrombin[9]. Antithrombin III will complex with the thrombin to neutralize it; however, in the absence of its cofactor heparin, antithrombin III reacts fairly slowly. Consequently, if the rate of thrombin generation exceeds the capacity of antithrombin to neutralize it, the thrombin activity will remain unchecked and thrombin production consequently becomes out of control[7]. The heparin-ATIII complex can also inactivate factors XIIa, XIa, and Xa, Kallikrein, and plasmin[9].

If plasmin is generated in amounts exceeding the capacity of the available antiproteases, excessive systemic fibrinolysis might occur[7]. Such unrestricted plasmin action can lead to further decreases in levels of fibrinogen and factors V and VIII, as well as depletion of antiplasmins in plasmin-antiplasmin complex formation and depletion of plasminogen by conversion to plasmin[7].

Protein C is an important regulator protein which has a dual function of being both a potent inhibitor of coagulation, and also enhances fibrinolysis. It is activated by thrombin, and this reaction is greatly enhanced by thrombomodulin, a cofactor found in the surface of endothelial cells[7]. In the presence of another cofactor, protein S, the complex destroys factor Va and VIIIa through proteolysis.

The protein C-S complex has also been shown to increase plasminogen activator activity by neutralizing the inhibitor of plasminogen activation[7]. In DIC, there is impairment of the fibrinolysis pathway caused by elevated levels of plasmino-

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gen activator inhibitor since protein C levels are decreased due to increased consumption.

As fibrin dissolves the clot, there is the formation of fibrin degradation products (FDP). These FDPs are significant because they can severely impair the haemostatic process and are presumably a major cause of haemorrhage in intravascular coagulation and fibrinogenolysis[4]. The FDPs demonstrate antithrombin activity, interfere with stabilization of the fibrin monomer, and also interfere with platelet function[4]. These FDPs have an affinity for coating platelet membranes and therefore can cause a clinically significant platelet dysfunction by inhibiting aggregation. Fibrinolysis can be detected by the D.dimer test which measures fibrin products that have been cross-linked by activated factor XIII[1]. Fibrin monomers are not normally present in the plasma, but when these monomers complex with FDPs, this gives rise to the pathological fibrinolytic state which is characteristic of DIC[4].

Diagnosis

As difficult as DIC is to define, it is even more troublesome to diagnose. It remains a challenge for the clinician to try and separate the possible symptoms caused by the disseminated intravascular coagulation while also trying to diagnose the underlying condition. Symptoms will vary depending on the severity of the DIC. Mild cases can have a slight case of coagulation imbalance, while severe cases will present with bleeding and symptoms of shock.

The varying degrees of severity are influenced by many factors. Some of these include the platelet and clotting factor levels prior to the onset of the DIC; the rate and amount of the trigger stimulus; the adequacy of the natural control mechanisms; the capacity of the bone marrow, liver, and endothelial cells to compensate for platelet and clotting factor consumption through increased production; and the effectiveness of the fibrinolytic mechanism in dissolving fibrin deposits and maintaining vessel integrity[7]. Some clinicians have even proposed a scoring system which assigns a point value based on the various clinical and laboratory findings, and the total score will conclude a DIC diagnosis[3].Obviously, there is no laboratory test specific for the diagnosis of DIC, but several tests in combination will often lead to an assumption of a DIC complication which can then be monitored.

Laboratory Findings

The classical blood picture is often similar to that of a microangiopathic haemolytic anaemia with decreased platelets, fibrinogen, factors II, V, VIII, XIII, and antithrombin III[7]. These decreased levels will cause a prolonged PT, APTT and TT. The breakdown of fibrin will cause the presence of fibrin degradation products which will cause a positive DDimer test [7]. In brief, excess thrombin generation will be detected by prolonged PT, PTT and TT, a decrease in platelet count, and a decrease in fibrinogen levels. Excess plasmin generation will be detected by an increase in fibrin degradation products. This positive DDimer test is specific for fibrin degradation, which indicates that thrombosis has occurred before the fibrinolysis, which is a key in trying to diagnose DIC. As the patient recovers, these DDimer levels will progressively decrease.

The peripheral blood smear will most likely have the presence of schistocytes. Fibrin strands in the microcirculation can cause mechanical damage to the red cells, leading to the presence of these fragmented red blood cells in the circulation[14]. Platelets will be decreased because of intravascular clearance due to activation and aggregation at the sites of local prothrombotic reactions[13].

Chemistry tests should include renal, liver, and respiratory function tests. Other tests such as electrolytes and bicarbonate can be used to monitor the acid-base balance of the patient which can become disrupted in severe cases of DIC.

Other than coagulation, haematology testing will include the CBC to monitor the haemoglobin, as well as the platelet counts. These will help determine the transfusion needs of the patient.

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Microbiology tests might include blood cultures and testing of antibiotic sensitivities.

Treatment

The clinical and laboratory manifestations of DIC should resolve with correction of the inciting disorder, such as by effective administration of antimicrobials in sepsis, treatment of malignancy, surgery to repair an aneurymal dilatation, or removal of conceptus and placenta[13].

Treatment should be focused towards preventing hypotension and tissue ischemia. Replacement therapy focuses on the building blocks of the thrombus (platelets and fibrinogen), and secondarily on other coagulation factors, including factors VIII, XIII, V, and II[14]. Thus, in bleeding patients, platelet transfusion is indicated when the platelet count falls below 50,000/µL, and cryoprecipitate is needed if the fibrinogen level falls below 100mg/dL[14]. Cryoprecipitate is administered since it is an efficient means of fibrinogen replacement as it contains a minimum of 150 mg of fibrinogen, and it also contains a minimum of 80 IU of factor VIII. Fresh frozen plasma is indicated in the setting of haemorrhage that results from DIC once the fibrinogen is above 100 mg/dL[14]. Once an acceptable fibrinogen level has been achieved, the bleeding patient can effectively receive fresh frozen plasma since this contains a dose of all of the coagulation factors rather than only specific ones. Evidently, if the patient is bleeding, anaemia might warrant the transfusion of red blood cells since these will increase the oxygen carrying capacity.

After reading an abundance of literature, the consensus seems to be the same that although studies have shown that product concentrate such as protein C, fibrinogen or even factor XIII help in re-establishing specific coagulation imbalances, these are mostly used for congenital deficiencies rather than to treat DIC patients. Although theoretically it would make sense to treat the procoagulation pathway, the effects vary so greatly between patients that there is fear of causing too many clots, and obviously creating an even bigger problem[8].

The only factor concentrate that seems to be used when all else fails is activated recombinant factor VIIa. Some studies suggest that this product may increase thrombin generation via two pathways; one being tissue factor-dependent and another being an activated platelet-dependent manner; therefore, such a treatment may prove to be suboptimal in patients without functional platelets[11, 15]. Although there are successful cases of treating DIC with recombinant activated factor VIIa, the efficacy and safety of this treatment DIC is unknown[9, 15].

Case Study

In this case study, a post partum patient presented in the emergency department with an uncontrollable bleed and a critical fibrinogen level. An obstetrical complication most likely triggered a severe case of DIC. Obstetrical complications include things such as postpartum haemorrhage, placental abruption, pre-ecclampsia, retained dead fetus syndrome, retained placental fragments etc.

This case of DIC was most likely triggered by the tissue thromboplastin derived from the placenta entering the maternal circulation. Such an acute case of DIC usually results from the generation of a large amount of thrombin in a short period of time. Acute DIC might initially manifest itself as a hypercoagulable state and lead to thrombosis; nevertheless, it can be followed by the development of a so-called hypocoagulable phase caused by depletion of clotting factors[12]. The term acute defibrination has often been applied to this condition, owing to the sudden and massive depletion of fibrinogen as a result of coagulation activation and resultant overwhelming fibrinolysis[6].

It is crucial for such a patient with DIC to be treated immediately. Once the body enters a state of shock, haemostasis is lost, organ systems begin to fail, and the patient's life becomes jeopardized. Acute renal failure is a common occurrence in severe DIC, and can be disastrous[8]. This was a concern with this patient since she presented with hypovolemia and thrombosis.

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Kidneys are vital organs that perform a variety of important bodily functions. The most prominent functions are the removal of unwanted substances from the plasma (both waste and surplus), homeostasis (the maintenance of equilibrium) of the body's water, electrolyte and acid-base status, and participation in the endocrine regulation[10]. Some acute cases can be treated and will recover, but if renal damage is too severe, chronic renal failure might occur[10]. Other organ systems will usually follow renal shutdown since they can all be affected by thrombosis[10].

Since this patient's fibrinogen level is severely decreased, cryoprecipitate should be given since this product would transfuse the most amount of fibrinogen. Fresh frozen plasma will be needed as well in order to restore the other factors being consumed in the coagulopathy. If the patient is bleeding heavily, red cell concentrates might be needed to increase the haemoglobin and promote oxygen distribution to the organs and tissues. Also, if the platelet count is low and she is bleeding, platelets might be part of the treatment as well.

Once a patient has a compromised coagulation, they can certainly use a massive amount of product until the bleeding is brought under control. It is crucial for the transfusion medicine department to be well prepared and always have enough products available for the patient. This includes avoiding any delay for the patient such as making sure there are always red cell concentrates crossmatched, plasma units thawed, cryoprecipitate pooled, and above all, ensuring an adequate inventory of products for the patient's needs. Good communication between the transfusion medicine and the patient's care unit is the key in this process.

Laboratory results will play an important role in monitoring this patient. Everything from coagulation to organ function tests should be clearly monitored in order to notice any improvements or collapse of the patient's condition.

Conclusion

Disseminated intravascular coagulation is a condition that complicates many disorders. The key to recovery seems to be an early diagnosis with an immediate treatment. The transfusion medicine department definitely has a critical role in providing life-saving products for a haemorrhaging patient with disseminated intravascular coagulation.

NBSMLT Awards Presented to MLT Graduates

NBSMLT executive director and registrar, Janelle B. Whitlock presented two awards to Stéphanie Caissie both academic excellence, and clinical excellence in June, 2009.

In addition, Stéphanie's name was engraved on a commemorative plaque.

Also, we wish congratulate Allison Ahier from NBCC Saint John, who received the General Proficiency Award this year.

Congratulation Graduates

Make your voice heard!

As many of you are aware, the Federal Government has introduced an agreement to amend Chapter seven pertaining to labour mobility across Canada. Even though the new legislation is not yet effective in New Brunswick, if it does become effective, the NBSMLT will have to license any technologist who is already licensed in another regulated province regardless if they have successfully completed the CSMLS certification exam. Most provinces have the CSMLS certification as their main requirement for licensure except for Québec who have their own process. The NBSMLT has sent their concerns with regards to the removal of the exam as a requirement for licensure to the Premier, the Minister of Health and to the Senior policy advisor at the Department of Post-Secondary Education, Training and Labour.

The following is an example of a form letter that is currently available on the NBSMLT Website. Members can send this letter to their local MPs or MLAs to make their voice heard.

Your name:

Your address:

Date:

MP/MLA name

MP/MLA address

Dear *name of official*:

As an active member of the New Brunswick Society of Medical Laboratory Technologists, I would like to take the opportunity to express my concern regarding the amendment to Chapter 7 (Labour Mobility Clause) of the Agreement on Internal Trade.

I understand the intent was to increase labour mobility, but, I believe that removing the requirement for successful completion of the professional competency examination; which has been recognized by nine (9) provinces as an acceptable entry to practice standard; is ill advised. The current professional competency examination provides labour mobility because it is the recognised occupational standard for entry to practice medical laboratory technology for most of Canada.

This entry to practice standard has provided a national benchmark against which **all** medical laboratory technologist candidates may be measured. Removing this standard will result in increased costs, as each province must then develop another framework to ensure that the level of training meets the high standards previously provided by the common professional competency examination (CSMLS certification exam).

I urge you to reconsider support of the proposed amendment to Chapter 7, Labour Mobility, of the Agreement on Internal Trade.

Sincerely;

Your name

Annual Provincial Scientific Convention 2009

The X Factor

*e**X**amine the facts...e**X**pand your mind...e**X**tend your knowledge*

October 14-17, 2009

Beaverbrook Crowne Plaza

659 Queen St.

Fredericton NB

Tentative Scientific Program

Wednesday, October 14, 2009

Flowcytometry: Aline Mansour (Sysmex) "The Future is Now" Presentation and Discussion

Safety Workshop: "Transportation of Dangerous Goods Certification Course" (DECRH) Coral MacRae MLT

Thursday, October 15, 2009

Keynote Speaker: Kathleen Howard

EXHIBITS OPEN

Concurrent Sessions

Clinical Chemistry:	Jeffrey Moore MD (Cardiologist) TBA
	Janet Walker RN BN "Hemodialysis"
	Daniel Hughes MD "Cystic Fibrosis"
Haematology:	James Henderson MD "Biologic Treatment for Inflammatory Arthritis"
	Akmal Ghafoor MD "Afebrile Neutropenia"
	Pharmacist from SJRH "POC for INRs"
Microbiology:	James Goltz DMV "Ticks in NB"
	Daniel Hughes MD "Cystic Fibrosis"
	Christine Contant MLT "Influenza"
Transfusion Medicine	Sue Smith CBS "OneMatch Stem Cell and Marrow Network"
	Dorothy Harris MLT CBS "Supply and Demand of Blood Products"
	Dorine Belliveau "Hemophilia"
Histopathology:	Mihkel Oja MD (Plastic Surgeon) TBA
General:	Colleen O'Connell PhM "Helping Hands Canada"
	Roberto Sgrosso WorkSafeNB TBA
	John Swanwick BSc MLT "One Patient, One Report"
	Tim Christie MD (Clinical Ethicist) TBA

Social Program

Thursday

6:30 - 8:30 pm

Exhibitor's Night

9 pm

Haunted Walk

(through Historic downtown Fredericton)

Friday

8 pm "BOO" Bash

DJ, dance, prizes

Bring your Costume!

Friday, October 16, 2009

Concurrent Sessions

Clinical Chemistry:	Angela McGibbon MD (Endocrinologist) TBA
	Kimberly Butt MD "Maternal Serum Screening"
	Jamie Reynolds RT "Blood Gas Review"
	Alfred Robichaud MD " Conceptia Clinic" (English &French)
Haematology:	Martha Mills MD "Coagulation in Pregnancy"
	Janet Milne "Coagulation Workshop" and "Herbal Remedy Presentation"
	Travis Quigley "Bone Marrow Recipient"
Microbiology:	Duncan Webster MD "TB ... an Old Disease with New Diagnostics & Treatment"
	Jacqueline Badcock DVM "Lyme Disease in NB "
	Barry Wecker MD "Tropical Diseases in Developing Countries"
	Gordon Dow MD "Toxic Shock Syndrome" and "Infections Acquired in the New Brunswick Outdoors"
Transfusion Medicine:	Hany Elgendy MD "Factor VIII Inhibitors"
	Danny Lemieux "Molecular Testing in Transfusion Medicine"
	Diane Burella "Automation in Transfusion Medicine"
	TBA "LEAN Transformation in TM"
	Claude Gilks "Alternatives to Tranfusion and a Major Religion's Work and Influence in Advancing Its Development"
Histopathology:	Jane Agar MLT "MOHS"
	David Barnett MD "Mass Spectrometry at the Atlantic Cancer Research Institute"
	Ken Obenson MD TBA
General:	Heather Hamilton RN BN "Mentoring New Employees"
	Thelma Greene Research & Productivity Council TBA
	LaLena Stary MLT "MLT in Afganistan"
	Moira McLaughlin MA "Forensic Case Studies"
	Cathy Moran – Robinson "New MLT Program"

SPONSORS to date

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- *Hologic
- *Immucor Gamma
- *InterMedico
- *Inverness Medical Canada
- *NB Hemophilia Society
- *Ortho Clinical Diagnostics
- *Radiometer Canada
- *Research and Productivity Council
- *RHA B Zone 3 Dept. of Laboratory Medicine
- *RHA B Zone 3 DECRH
- *Roche Diagnostics
- *Sarstedt Inc
- *Seimens Canada
- *Somagen Diagnostics Inc
- *Sysmex Canada Inc

Saturday, October 17, 2009

General Sessions:	Darren Martin "Kidney Transplant Recipient"
	Sandra Rooney MLT "Risk Management"
Cytopathology:	Dan Fontaine MD "HPV" and TBA
	Eshwar Kumar MD, Rejean Savoie MD "NB Cervical Cancer Screening Program"
	Shannon Nardin "Gynecologcal Liquid Based Pap Tests"
Noon:	**Annual General Meeting**
Cytopathology:	Shannon Nardin: Workshop at DECRH "Gynecological Liquid Based Pap Tests"

Registration Form to Follow By Email ....Watch NBSMLT website for updates and Registration Form