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Contents

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Address questions to:
Charlene Kennett, Administrative Assistant
Canadian Council of Cardiovascular Nursing
c/o Heart & Stroke Foundation Canada
Suite 1402, 222 Queen Street
Ottawa, Ontario K1P 5V9 Canada
Tel: (613) 569-4361 Fax: (613) 569-3278 ckennett@hsf.ca

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Editorial

Theresa Mirka
RN, BScN, MHSc
Editor-in-chief

This issue of the journal has a plethora of information that will help to keep cardiovascular nurses current. We have hypertension guidelines, an update on cardiac biomarkers and chest tube stumps, and a must read research rounds for all those whose abstracts have been accepted.

Karen Then has provided the journal with the 2001 Canadian Hypertension Guidelines. Because health promotion is one of the cornerstones of our practice, I know that you will find these useful when assisting your patients in adjusting to this significant health risk. The guidelines are presented in a summary format for quick reference.

The research articles in this issue highlight changes in cardiovascular nursing. First, Donna Best and her colleague from Memorial University present the newest information on the new cardiac biomarkers. I'm sure you have been wondering why all of our labs have been changing to measure troponins and myoglobin. These authors present the latest research and show how it has been integrated into practice in their institution. In addition, Gail Urkhart and colleagues present a very interesting paper on Mediastinal chest stumps. I know that many of you may have seen these in practice, and will benefit from the integration of theory and practice presented in this paper.

Rounding out this issue is a must read for all who have received a letter of acceptance regarding abstracts for the upcoming Scientific Sessions. Kathy King and Cathy Adams skillfully guide readers through tips and expectations regarding oral and poster formats. This is a wonderfully written piece that will end up posted on many walls!! Be sure to use the ideas in this article for the upcoming meetings, and you will avoid many last minute crises.

We are sure that you will find this issue helpful to your practice. Enjoy!

Theresa Mirka, ACNP, MHSc., RN
Editor-in-Chief

Clinical Rounds

The 2001 Canadian Hypertension Recommendations What's new and what's old but still important.

Canadian Hypertension Recommendations Working Group

The Canadian Hypertension Recommendations Working Group include the following:

Steering Committee: N.R.C. Campbell (Chair; CCHBPPC), R. Feldman (CHS), E. Wilson (HSFC), S. Nagpal (Health Canada), A. Chockalingam (Health Canada), T. Squires (CFPC).

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Office Measurement of BP: C. Abbott (Chair), K. Mann;
Follow-up of BP: P. Bolli;
Self-measurement of BP: D. McKay (Chair), B. Ens;
Ambulatory BP Monitoring: M. Myers, S. Rabkin;
Routine Laboratory Testing: T. Wilson;
Echocardiography: G. Honos;
Global Risk Assessment: S. Grover;
Endocrine Hypertension: E. Schiffrin;
Lifestyle Modification: E. Burgess (Chair), R. Petrella, R. Touyz;
Pharmacotherapy of Uncomplicated Hypertension: R. Lewanczuk (Chair); J. Wright, B. Culleton;
Elderly subsection: G. Fodor, P. Hamet, R. Herman;
Pharmacotherapy for Hypertension in patients with Cardiovascular Disease: F. Leenen (Chair); S. Rabkin, J. Stone;
Diabetes and Hypertension: J. Mahon (Chair), C. Jones, P. Larochelle, R. Ogilvie, S. Tobe;
Renal and Renovascular HT: M. Lebel (Chair), E. Burgess, S. Tobe;
Concordance Strategies for Patients: R.D. Feldman (Chair), J. Irvine.

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Sponsored by: The Canadian Hypertension Society, The Canadian Coalition for High Blood Pressure Prevention and Control, The College of Family Physicians of Canada, The Heart and Stroke Foundation of Canada, Adult Disease Division and Bureau of Cardio-Respiratory Diseases and Diabetes, Centre for Chronic Disease Prevention and Control Health Canada.

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Introduction

Hypertension is one of the most common reasons for an adult patient to visit a physician and is estimated to be the third leading risk associated with death worldwide (1). The last reliable data on hypertension prevalence and control in Canada is 10-15 years old (2). At that time, 22% of adult Canadians had high blood pressure, and only 16% of those with hypertension were treated and controlled. Preliminary data (Campbell, Unpublished) suggests a significant increase in prescriptions of major classes of antihypertensive agents coinciding with the introduction of the annual recommendations and implementation process. However, whether this reflects an improvement in treatment and control of hypertension is uncertain. Unfortunately, our national health surveillance is inadequate to determine whether hypertension prevalence, awareness, treatment, or control has changed. What data is available behooves all health care professionals to prioritize hypertension as a public health issue and aggressively identify, treat, and control hypertensive patients according to the best available evidence and recommendations.

This is the third year that Canada has comprehensively updated its hypertension recommendations (3, 4, 5, 6, 7). The recommendations are linked to an expanding implementation effort (8). This is a brief summary of the 2001 recommendations, highlighting those recommendations that are new, revised, or important to improve blood pressure control in Canada. New recommendations of specific interest include an updated section on management of hypertension in persons with diabetes. Also included are new recommendations for initial therapy and a new recommendation to lower blood pressure following the acute phase of strokes or transient ischemic attacks. The arbitrary classification of old and young persons at age

sixty has been removed. Evidence for an age effect is required as opposed to the previous requirement for evidence in the specific-age categories. This has resulted in a more aggressive threshold for initiating therapy in those over age sixty. The recommendation to switch first line therapies when there is inadequate response has been changed to a recommendation to combine first line therapies, thereby recognizing the need for multiple drugs to control hypertension as well as the sequential method of adding medications used in major therapeutic trials. There are also new comprehensive sections on management of patients with pheochromocytoma and hyperaldosteronism.

The purpose of this summary is to provide a rapid update to the 2001 hypertension recommendations. A full publication of the comprehensive recommendations has been published (6,7). The latter publication is intended to be a scientific reference and not a clinical practice guideline. A slide kit and clinical practice algorithms supporting the full 2001 recommendations are available to download at www.chs.md.

The methods for producing the recommendations have been published previously (9), but there have been some revisions. In 2001, a separate meeting of those involved in the production of recommendations was held to discuss new, changed, or controversial recommendations and evidence. While a voting process adopted in 2000 to exclude recommendations where 30% or more of those involved with the subgroups, central review committee, and steering committee disagreed was continued, individuals with direct conflict of interest on specific recommendations were excluded from voting on those recommendations. Those with conflict of interest participated in the discussions following disclosure. The recommendations were based on the results of literature searches (to at least March 2001), personal knowledge of published literature, contact with authors, and major clinical trials published prior to November 2001.

Diagnosis

Although there are no substantive changes to this section, the diagnosis is critical to the management of hypertension. The recommendations highlight the importance of assessing the blood pressure of all adults using proper measurement technique at all appropriate visits. Hypertension can be diagnosed immediately if there is a hypertensive urgency or crisis, and in three visits in the presence of target organ damage in patients who are clinically stable. However, diagnosis requires up to five visits if there is no target organ damage and the initial blood pressure is <180/105 mm Hg. Although the recommendation for five visits represents substantive work, those whose blood pressure falls to less than <140/90 mm Hg with observation have a normal prognosis and can avoid labeling and interventions that may harm them (10). Self-measurement and 24-hour ambulatory measurement continue to be recommended for consideration in assessing office-induced blood pressure elevation, and the former to improve patient compliance. Only devices meeting international standards should be used (11). Daytime blood pressures <135/85 mm Hg with ambulatory and self-measurement are associated with a normal prognosis.

Laboratory Investigation

Routine laboratory assessment should be performed at diagnosis, and should include blood for electrolytes, creatinine, fasting glucose, complete blood count, and lipid profile (total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides); a urinalysis; and an electrocardiogram. Criteria for screening patients for renovascular hypertension with a post captopril renogram (Table 1) and pheochromocytoma (Table 2) with a 24-hour urine for metanephrines and creatinine is provided. Screening patients for pheochromocytoma by assessment of urinary vanilmandelic acid (VMA) is inadequate. More recent studies have confirmed that hyperaldosteronism is relatively common. Screening for hyperaldosteronism should include assessment of a plasma aldosterone, and plasma renin activity measured in morning samples taken from patients in a sitting position after resting at least 15 minutes. (Antihypertensive drugs with the exception of aldosterone antagonists may be continued prior to testing.) The criteria for selecting patients to be screened for hyperaldosteronism is shown in Table 3. Comprehensive

Table 1:

Patients with the characteristics listed below and who are candidates for angioplasty or revascularization should be screened for renovascular hypertension with a post-captopril renogram
• Uncontrolled hypertension despite therapy with three or more drugs, • Deteriorating renal function, • Recurrent episodes of flash pulmonary edema

Table 2:

Patients with the following characteristics should be considered for screening for pheochromocytoma with a 24-hour urine for metanephrines and creatinine*
• Paroxysmal and/or severe sustained hypertension refractory to usual antihypertensive therapy;
• Hypertension and symptoms suggestive of catacholamine excess (two or more of headaches, palpitations, sweating, etc.);
• Hypertension triggered by beta-blockers, monoamine oxidase inhibitors, micturation, or changes in abdominal pressure;
• Incidentally discovered adrenal adenoma;
• Multiple endocrine neoplasia (MEN) 2A or 2B; von Recklinghausen's neurofibromatosis, or von Hippel-Lindau disease.

*Assessment of urinary VMA is inadequate.

Table 3:

Screening for hyperaldosteronism should be considered for at least hypertensive patients with the following characteristics*
• Spontaneous hypokalemia
• Profound diuretic-induced hypokalemia (<3.0 mmol/L)
• Hypertension refractory to treatment with three or more drugs;
• Incidental adrenal adenomas.

* Screening for hyperaldosteronism should include assessment of a plasma aldosterone and plasma renin activity measured in morning samples taken from patients in a sitting position after resting at least 15 minutes. Antihypertensive drugs with the exception of aldosterone antagonists may be continued prior to testing.

Table 4:

Cardiovascular risk assessment methods based on Framingham data can be performed by many ways:
• for information about the use of desk top computers, see www.hyp.ac.uk/bhs/managemt.html;
• for information about palm-type devices, see www.statcoder.com/cardiac.htm;
• for information about the use of risk charts, see www.hyp.ac.uk/bhs/managemt.html or *Journal of Hypertens* (1999) 17:151-83 (10);
• for information about calculators with the incorporated formulae, see JAMA 2001: 285:2486-97 (11), or Circulation 1999, 100: 1481-1492 (12) or J Human Hypertens 1999, 13:569-92 (13) or BMJ 2000, 320:709-710 (14).

recommendations for diagnosis and management of pheochromocytoma and hyperaldosteronism will be published with the detailed 2001 hypertension recommendations.

Risk Assessment

It is recommended to assess quantitatively a patient's cardiovascular risk and adopt a multi-factorial approach for treating hypertension. A variety of methods can be used (Table 4 and references 12-16).

Lifestyle Modification

Individualized lifestyle modification is recommended for all patients with hypertension and those at risk for developing hypertension. A diet consistent with Canada's Guide to Healthy Eating (i.e., high in fresh fruit and vegetables and low fat dairy products and low in saturated fat) and limitation of salt additives and foods with excessive added salt will lower blood pressure. Other lifestyle changes that are effective at reducing blood pressure include weight loss (4.5 Kg minimum) in those who are overweight, regular physical activity (optimum 45-60 minutes of moderate activity (brisk walking 4-5 times a week), and low risk alcohol consumption (0-2 drinks per day; less than 14 drinks per week in men, less than 9 drinks per week in women). Cognitive behavior modification for stress management is effective in some individuals. Because smoking is a major cardiovascular risk factor, has greater than additive risk in hypertensive persons, and reduces or abolishes the beneficial outcomes associated with antihypertensive therapy, smoking cessation should be strongly encouraged in all hypertensive patients.

Pharmacotherapy

Drug treatment is recommended if the diastolic blood pressure

is greater than 90 mmHg and if there is cardiovascular disease or other target organ damage or cardiovascular risk factors. Most hypertensive patients have additional risk factors or target organ damage. However, if these are not present, the lower cardiovascular risk has resulted in a recommendation to treat diastolic blood pressure ≥100 mm Hg or systolic blood pressure ≥ 160 mm Hg. Initial drugs for diastolic and combined systolic and diastolic hypertension include diuretics, long acting dihydropyridine calcium channel blockers, and angiotensin converting enzyme inhibitors. Beta-blockers are recommended as first line therapy in those under but not age sixty or over. Alpha-blockers are not recommended as first line therapy and short acting calcium channel blockers should not be used as antihypertensive agents. For isolated systolic hypertension, initial therapy should be with a low dose thiazide diuretic or a long acting dihydropyridine calcium channel blocker.

In persons with diabetes mellitus, angiotensin converting enzyme inhibitors are recommended as first line therapy in all situations. Low dose thiazide diuretics and long acting dihydropyridine calcium channel blockers are recommended as alternative first line agents in isolated systolic hypertension. Angiotensin II receptor blockers are recommended as alternative first line agents to angiotensin converting enzyme inhibitors in the presence of diabetic renal disease (e.g., microalbuminuria >30 mg/24 hours).

Controlling High Blood Pressure

As in the previous year, one of the most important aspects of the recommendations is the need to control blood pressure in treated patients. It is recommended to reduce blood pressure to less than 140/90 mmHg in most patients including the elderly and less than 130/80 mmHg in patients with diabetes mellitus or renal dysfunction. Lowering blood pressure to less than 125/75 mm Hg is recommended in patients with renal dysfunction and >1g/day proteinuria. Two new changes are the recommendation to use combinations of medication if the initial choice is ineffective, and to switch to alternate first line agents only if there is intolerance or adverse effects. The average blood pressure lowering of a single drug is about 10/5 mm Hg. In the large outcome trials, stepwise addition of antihypertensive medications were prescribed to achieve blood pressure targets, and the use of multiple agents was necessary in a large proportion of patients. Table 5 indicates combinations of first line agents that have additive hypotensive effect when used in combination for the treatment of uncomplicated hypertension. Other first line dual agent therapies have less than additive hypotensive effects and are recommended only for specific indications (e.g., b-blockers and angiotensin converting enzyme inhibitors after myocardial infarction). In uncomplicated hypertension when using triple or quadruple therapy, all potential antihypertensive combinations of first line agents are effective. Individual physicians need to assess their personal skill and experience in determining the need for specialty consultation for resistant hypertension. In patients who have little response to appropriate single or combination medications, consider non-adherence, secondary hypertension, interfering drugs, lifestyle and/or office induced increases in blood pressure (white coat effect).

In specific patient subgroups, there are further treatment recommendations (see Table 6). A notable change is the recommendation to consider strongly antihypertensive therapy in persons who have had a non-disabling stroke or TIA after the acute phase. A recent trial (PROGRESS) demonstrated a reduction in recurrent cerebrovascular events when blood pressure was lowered in both hypertensive and normotensive persons (17).

Patient's compliance is still a major challenge and should be addressed by health care professionals at each medical visit.

There were 105 recommendations produced and 38 eligible voters (subgroup, central review committee, and steering committee membership). For 22 recommendations, there was no disagreement; 74 recommendations had one vote in

Table 5: For additive hypotensive affect in dual therapy combine an agent from column one with any in column two*

Column 1	Column 2
Low dose thiazide diuretics	β-blocker
Long acting dihydropyridine calcium channel blocker	ACE Inhibitor **

*Dual combination of agents within column 1 and within column 2 have less than additive hypotensive affect but may be indicated in specific settings (e.g. column 2 drugs in patients following myocardial infarction). In uncomplicated hypertension when using triple or quadruple therapy, all potential antihypertensive combinations of first line agents are effective.
** Angiotensin receptor blockers are an alternative initial choice in patients with diabetes and nephropathy.

disagreement; 7 recommendations had 2 votes in disagreement; and 2 recommendations had 3 votes of disagreement. The recommendation that had the greatest disagreement was voted against by 8% of the eligible voters. It is important that individuals involved in the recommendations

Table 6: Considerations in the individualization of antihypertensive therapy

Risk factor/disease	Initial Therapy	Second Step Therapy	Notes/cautions*
Uncomplicated hypertension	Low dose thiazide-like diuretics, Beta blockers, ACE inhibitors or long acting dihydropyridine calcium channel blocker	Combinations of first line drugs. See table 5 `	α blockers are not recommended as initial therapy. β blockers are not recommended as initial therapy in those over age 60. Hypokalemia should be avoided by using K sparing agents in those prescribed diuretics
Isolated systolic hypertension	Low dose thiazide-like diuretics, or long acting dihydropyridine calcium channel blocker		Hypokalemia should be avoided by using K sparing agents in those prescribed diuretics
Diabetes mellitus with nephropathy	ACE inhibitors alternatively angiotensin II receptor blockers	One or more of low dose thiazide like diuretics, cardioselective b-blockers, long acting calcium channel blockers	If the serum creatinine is > 150 umol/L, a loop diuretic should be used as a replacement for a low dose thiazide diuretics if volume control is required
Diabetes mellitus without nephropathy	ACE inhibitors	One or more of Angiotensin II receptor blockers, low dose thiazide-like diuretics, cardioselective b-blockers, long acting calcium channel blockers	
Diabetes mellitus without nephropathy, with systolic hypertension	ACEI, alternatively, low dose thiazide diuretics long acting dihydropyridine calcium channel blockers		
Angina	Beta blockers (consider ACE inhibitors as add on therapy)	Long acting calcium channel blockers	
Prior myocardial infarction	Beta blockers and /or ACE inhibitors	Combinations of additional agents	
Systolic dysfunction	ACE inhibitors (thiazide or loop diuretics, beta blockers, spironolactone as additive therapy)	Angiotensin II receptor blockers hydralazine/isosorbide dinitrate, Amlodipine	Avoid non-dihydropyridine, calcium channel blockers (diltiazem, verapamil)
Past cerebrovascular accident or TIA	Strongly consider blood pressure reduction after the acute phase		Blood pressure reduction reduces recurrent cerebrovascular events
Renal disease	ACE inhibitors (diuretics as additive therapy)	Combinations of additional agents	ACE inhibitors if bilateral renal artery stenosis
Left Ventricular Hypertrophy	Does not affect initial treatment recommendations	Does not effect initial treatment recommendations	Avoid hydralazine, and minoxidil
Peripheral arterial disease	Does not affect initial treatment recommendations	Does not effect initial treatment recommendations	Avoid beta blockers with severe disease
Dyslipidemia	Does not affect initial treatment recommendations	Does not effect initial treatment recommendations	

* When using two drugs specifically to lower blood pressure, use table 5 to maximize the hypotensive effect. Short acting calcium channel blockers are not recommended in the treatment of hypertension.

process or in a subgroup may have personally opposed specific recommendations. Therefore, acknowledgement of an individual's contribution to the hypertension recommendations process does not indicate personal support for any specific recommendation.

Acknowledgements

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Cardiac Biomarkers: New Kids on the Block

Donna G. Best, Memorial University of Newfoundland, St. John's, Newfoundland
Patricia Grainger, Center for Nursing Studies, St. John's, Newfoundland

Early diagnosis and treatment are imperative in the management of acute coronary syndrome (ACS). The World Health Organization guidelines for the diagnosis of myocardial infarction (MI) include diagnostic ECG changes, characteristic chest pain, and abnormally elevated serial cardiac enzymes. Because of the delay in elevated CK-MB serum levels after MI (6-8 hours), its use as an early diagnostic tool is limited. Other cardiac markers (CK and AST) lack specificity for myocardial injury. New biochemical markers are now used along with the standards of ECG and patient symptoms to diagnose ACS.

Myoglobin and troponin I are two markers used in our institution. Myoglobin has been detected in patients two hours after the onset of chest pain and has been shown to have high specificity for detecting MI. Although not perfect, myoglobin is the earliest significant indicator of breakdown of myocardial cells. Troponin I is very specific for myocardial injury as it is found only in myocardial cells.

Nursing knowledge in this area is very important in relation to the collection of medical histories, timing of laboratory tests, and the interpretation of patients' laboratory results. In addition, the nurse's role in performing bedside assays used to facilitate early treatment is expanding. A knowledge of diagnostic tests commonly used in the care of patients with suspected ACS is imperative if patients are to receive appropriate, timely, and cost effective care.

Research related to the use of biomarkers is ongoing. Biomarkers have been used in detecting reperfusion after thrombolytic therapy and angioplasty. Their use in detecting MI during and following open heart surgery is also being investigated.

Contact Information:
Donna G. Best is an Assistant Professor at Memorial University School of Nursing and Patricia Grainger is a Nurse Educator at the Center for Nursing Studies in St. John's, NF
Correspondence should be addressed to Donna G. Best, Assistant Professor Memorial University School of Nursing, St. John's, NF, A1B 3V6, Tel. No. (709) 777-7103, Fax: (709) 777-7037, email: dbest@mun.ca

The management of individuals with acute chest pain continues to present many challenges for the health care system. Early and accurate diagnoses are necessary in order to facilitate the optimal treatment of acute coronary syndrome (ACS) such as myocardial infarction (MI) as well as cost-effective management of non-cardiac problems. Acute coronary syndrome is a pathophysiologic continuum that results from rupture of an atherosclerotic plaque and the formation of an associated thrombus. It can result in clinical presentations ranging from entirely asymptomatic to unstable angina to myocardial infarction (Fuster & Badimon, 1999). In the past, MI was diagnosed using the World Health Organization guidelines that included the presence of two of the following criteria: characteristic chest pain, diagnostic electrocardiograph (ECG) changes, and elevated serial cardiac markers (World Health Organization, 1979). Unfortunately, diagnosis may be difficult and time to treatment prolonged as symptoms can be non-specific; laboratory results may not be available; and ECG changes may not be evident upon presentation (Christenson & Duh, 1999). Recent advances in research and technology relating to biomarkers are influencing the diagnosis and treatment of ACS.

History of Cardiac Biomarkers

The use of serum enzymes as a means of diagnosis of MI began nearly 50 years ago. Enzymes, found within the cell, are released into the circulation upon injury. Detection of elevated amounts of the enzyme indicates damage to the tissue for which the enzyme has specificity (Vaughn, 1999). The first cardiac enzyme used in the diagnosis of MI was aspartate aminotransferase (AST) in 1954 followed by lactate dehydrogenase (LD) and creatine kinase (CK) in 1960. These enzymes rise slowly following injury and are not specific to cardiac tissue, reducing their diagnostic usefulness. In 1975, an isoenzyme of CK, CK-MB, was recognized as an acceptable and more specific test for the diagnosis of MI (Woods, Froelicher, & Motzer, 2000).

Initially, CK-MB enzyme activity was measured to determine myocardial damage. At present, CK-MB mass assay is the preferred method of testing (Alpert et al., 2000). A large body of evidence supported the use of CK-MB as a marker because of its high sensitivity and specificity for myocardial injury. Sensitivity refers to the degree to which the test can detect the presence of a condition and the potential of the test to rule out false negatives. Specificity is the ability of a test to rule out false positives and the ability of the test to be positive only for those cases where the condition actually exists (Murphy & Berding, 1999).

CK-MB has demonstrated sensitivity of 97% and specificity of 90%

at 12 to 48 hours following onset of symptoms (Wu & Lane, 1995). However, there are disadvantages to using CK-MB. Early intervention is impeded, as blood concentrations do not increase until four to six hours following the onset of symptoms, making CK-MB unreliable in ruling out MI until 10 to 12 hours following onset of symptoms (Puleo et al. 1994). Moreover, skeletal muscle also contains CK-MB resulting in a false positive diagnosis of MI, particularly when there is concurrent skeletal and myocardial damage. Because CK-MB is thought to be released only in situations of irreversible necrosis, it is only diagnostic for MI and fails to detect myocardial ischemia (Wu, 1999). Thus, it cannot be used as a means of risk stratification to direct future treatment decisions (Christenson & Duh, 1999).

In summary, low specificity of AST, LD, and CK led to the study and development of other cardiac markers (Wu, 1999). The development of new laboratory tests allowed for the measurement of enzyme concentrations, or mass assays, as well as non-enzyme protein markers (Wu, 1999). Recent research now indicates that other cardiac markers such as myoglobin, CK-MB isoforms, and cardiac troponins I and T can contribute to early and accurate diagnosis of MI as well as aid in risk stratification and evaluation of thrombolytic and reperfusion therapies (see Table 1).

Biomarkers for Early Diagnosis of MI

Myoglobin

Myoglobin, a low-molecular weight oxygen-binding protein found in cardiac as well as skeletal muscle, is responsible for oxygen storage. It is made up of one heme molecule and one iron molecule attached to a protein chain. Myoglobin gives muscles their characteristic red color and is released into circulation only after muscle damage (Woods et al., 2000). Normal blood levels for myoglobin vary, as cutoff points are highly method dependent. Upper limits for normal reference levels range from 70 to 150 mg/L (Bhayana & Henderson, 1995; Montague & Kircher, 1995; Ng et al., 2001).

Table 1: Overview of Selected Cardiac Biomarkers for Acute Coronary Syndromes

Biomarker	Onset	Peak	Duration	Strengths	Limitations
CK-MB	4-6 hrs	24 hrs	8-72 hrs	inexpensive	increased in renal failure, skeletal muscle injuries and disorders
CK-MB isoforms	1-4 hrs	4-6 hrs	1-2 days	high sensitivity early marker	low specificity testing is expensive
Myoglobin	2-4 hrs	8-12 hrs	12-30 hrs	high sensitivity early marker monitors MI extension & recurrence & reperfusion therapy	increased in neuromuscular disorders, renal failure, IM injections, strenuous exercise, other muscle disorders low specificity
Troponin I	3 hrs	14-18 hrs	5-7 days	high specificity late diagnosis risk stratification monitors reperfusion therapy	may mask reinfarction occurring in 5-7 days
Troponin T	3-5 hrs	72 hrs	>21 days	high sensitivity risk stratification monitors reperfusion therapy	lower specificity than Troponin I increased in renal failure and certain musculoskeletal disorders patent restrictions

Because of its small size and high tissue concentration, myoglobin is released rapidly into circulation following muscle damage (Christenson & Duh, 1999). Following MI, myoglobin levels increase in 2 to 4 hours, peak in 8 to 12 hours, and return to normal anywhere from 12 to 30 hours (Woods et al., 2000). With sensitivity rates reported as high as 100% in 2 hours, myoglobin can be considered an excellent early cardiac marker (Montague & Kirchner, 1995). Unfortunately, myoglobin can be abnormally increased in renal failure, in neuromuscular and other disorders affecting muscle function, and following intramuscular injections and strenuous exercise (Plebani & Zaninotto, 1998) resulting in specificity values as low as 46% (Zaninotto, Altinier, Lachin, Celegon, & Plebani, 1999). Because of this relatively low specificity, myoglobin levels are primarily used to rule out MI or to direct further testing (Braunwald et al., 2000).

CK-MB Isoforms

CK-MB can be further broken down through electrophoresis into isoforms known as CK-MB1 and CK-MB2. CK-MB2 increases significantly within one hour after the onset of chest pain associated with myocardial tissue damage making it an excellent early indicator of MI. This isoform (CK-MB2) peaks within 4-6 hours and returns to normal in 1-2 days (Siomko, 2000). However, laboratory testing requires special expertise and equipment. Experience to date has been predominantly in dedicated research centers (Braunwald et al., 2000).

Troponins

Troponin is a protein complex, found in striated muscle cells that regulates the interaction between actin and myosin molecules. There are three known isotopes: troponin T, troponin I, and troponin C. Unlike troponin C, troponins I and T found in cardiac muscle can be differentiated from troponin I and T found in skeletal muscle (Alonsozana & Christenson, 1996; Fromm & Roberts, 2001; Murphy & Berding, 1999).

Troponins I and T are not normally present in the blood of healthy individuals. There are discrepancies in reference values, as well as in sensitivity and specificity measures for these markers due to variation in equipment and testing methods, institutional policies, and interpretation of research findings (Apple & Wu, 2001; Murphy & Berding, 1999). Normal ranges have been reported as 0 to 2 ng/ml (0-0.2 mg/L) for troponin I and 0 to 3.1 ng/ml (0-0.3 mg/L) for troponin T (Woods et al., 2000). Troponin I levels begin to increase 3 hours after MI, peak at 14 to 18 hours, and remain elevated for 5 to 7 days. Troponin T increases within 3 to 5 hours peaks at 72 hours and remains elevated for up to 21 days (Murphy & Berding, 1999).

Specificity rates for both troponin I and T are above 92% at 2 hours after the onset of symptoms (Tucker, et al., 1997; Zaninotto et al., 1999; Zimmerman et al., 1999). Sensitivity increases as time after onset of symptoms increases, ranging from 16 % at 2 hours to 96 % at 18 hours (Zimmerman et al., 1999). Because of its excellent specificity and persistent elevation, troponin I is very useful in the late diagnosis of MI (Fromm & Roberts, 2001; Kost, Kirk & Omand, 1998). Troponin I has the ability to detect microscopic zones of myocardial necrosis (Alpert et al., 2000).

Troponin T has a reported sensitivity of 87 % for MI 10 hours following the onset of symptoms (Wu & Lane, 1995). Initially, the specificity of troponin T was reported as less than that of troponin I due to its increase in renal disease and certain musculoskeletal disorders (Wu, Apple, & Warshaw, 1999). However, recent advances in laboratory technology have increased the specificity of troponin T (Fromm & Roberts, 2001). At present, one manufacturer holds patent restrictions on assays for troponin T, making troponin I the more practical laboratory test (Wu, 1999).

Clinical Significance of New Cardiac Biomarkers

Because it is the earliest biomarker to be released, has a high sensitivity, and testing can yield rapid results (turn around time of less than one hour), myoglobin is the best early indicator of MI. The ability of this test to rule out MI allows non-cardiac patients to be triaged and treated appropriately earlier than CK-MB. Recent developments in CK-MB isoform assays indicate that this biomarker is also an excellent early indicator of MI. Both of these markers are useful in ruling out MI but a more definitive test is necessary to confirm the diagnosis (Fromm & Roberts, 2000). These early markers are not useful for individuals who present at the emergency department well after the onset of symptoms (Apple & Wu, 2001). Research is now indicating that troponins I and T are equal to or superior to CK-MB in sensitivity and specificity for the diagnosis of MI (Christenson & Duh, 1999; Jaffe et al., 2000; Wu, 1999). The National Academy of Clinical Biochemistry has recommended that troponins replace CK-MB and become the new standard for diagnosis of MI (Apple & Wu, 2001; Wu et al., 1999).

These new biomarkers present other benefits in addition to their role in the initial diagnosis of MI. Myoglobin has been shown to monitor extension or recurrence of infarction (Plebani & Zaninotto, 1998). In contrast, troponins are not useful as they have a longer diagnostic window that may mask the diagnosis of reinfarction (Lee & Goldman, 2000).

The cardiac troponins can indicate minor cardiac damage that may aid in risk stratification. In the GUSTO IIA and TIMI IIIB studies, unstable angina patients with troponin levels that were elevated but below the level indicative of MI had significantly higher mortality rates than those with normal levels (Lindahl et al., 2000; Murphy and Berding, 1999; Zimmerman et al., 1999). Randomized drug trials with glycoprotein IIb/IIIa inhibitors and low molecular weight heparins have shown that the risk of cardiac events can be reduced for unstable angina patients with elevated troponin T levels (Braunwald et al., 2000; Wu, 1999). In the setting of myocardial ischemia, it is now possible to define minimal size infarcts as well as large infarcts using troponins (Alpert et al., 2000).

Measurements of myoglobin and troponin levels have also been shown effective, non-invasive means of evaluating coronary artery reperfusion after interventional therapies such as thrombolytics and angioplasty (Cross, 1998; Murphy & Berding, 1999; Plebani & Zaninotto, 1998). Once patency is established following MI, a bolus amount of enzymes and proteins are released into circulation. This is known as the "washout phenomenon" and is evident in blood samples drawn 1-2 hours post procedure (Wu et al., 1999). Contrary to those findings, Alpert and colleagues (2000) maintain that an increase in biomarkers after angioplasty is indicative of cell death and should be labeled MI.

Research is ongoing into protocols for frequency of blood collection and appropriate decision limits. Blood should be obtained for testing on hospital admission, within 2-4 hours and 6-9 hours. A blood sample is warranted at 12-24 hours if clinical suspicion remains high but markers are negative (Alpert et al., 2000; Jaffe et al., 2000). More studies are needed to determine the usefulness of cardiac biomarkers in the diagnosis of perioperative MI as no biomarker is capable of distinguishing damage due to acute infarction from damage from the procedure (Apple & Wu, 2001; Fromm & Roberts, 2001). Troponin I is normal or minimally affected by cardioversion (Allan et al., 1997).

Cost Effectiveness

The costs incurred by the institution for performing the testing varies. Several researchers identified that the most expensive tests of the five cardiac markers were Troponin T and CK-MB isoforms because of significant labour costs associated with performing the tests (Kost et al., 1998). The relative costs of the other three markers per test were CK-MB mass < myoglobin < Troponin I. In that institution, CK-MB mass and myoglobin were used in the chest pain evaluation unit (Kost et al.).

Decreased lengths of stay in hospital as well as avoiding unnecessary admissions to, or wrongful discharge from, hospital are other indicators used to determine costs. For example, the issue of rapid access and early triaging on CK-MB isoforms saves billions of dollars in the United States based on avoidance of hospital admissions because it provides the earliest diagnosis (Puleo et al., 1999). However the test is expensive and may not be practical for smaller institutions in Canada. Another study showed shorter hospital stay and lower charges were associated with rapid testing in the emergency room (Gomez, Anderson, Karagounis, Muhlestein, & Moores, 1996). One study observed that mean length of stay decreased by 0.7 days but laboratory costs were higher when CK-MB testing was performed three times daily or on demand (Wu & Clive, 1997). A decrease in length of stay in a Coronary Care Unit was found in another study that used serial enzymes to confirm diagnosis (Collinson, Ramhamadamy & Stubbs, 1993). Further research is required to determine if serial testing affects overall mortality.

An effective turn around time to rule-in or rule-out MI will enhance patient treatment. Patients with unstable angina would be triaged early and would not receive thrombolytic procedures that are expensive and may be harmful (Fromm & Roberts, 2001). Alternatively, patients with MI would receive prompt and appropriate treatments.

It is recognized in our institution that testing cardiac markers according to the new cardiac algorithm will likely increase cost over the previous method (Besso-Waterman, 2000). However, the new method is diagnostically more beneficial and will likely decrease overall hospital costs for management of chest pain patients (Dr. E. Randell, personal communication, July 14, 2000). Further research is needed to investigate overall savings to the institution.

Nursing Implications

Rapid interpretation of cardiac biomarker results facilitates early treatment. The appearance of certain biomarkers is indicative of minor myocardial injury and cardiac necrosis (Adams & Miracle, 1998). For patients with MI, time to treatment is crucial in salvaging cardiac muscle. Nurses in cardiac settings or in emergency rooms were familiar with previous markers used over the last decades. The research on these new biomarkers is ongoing and reference values for diagnosis of infarction are being altered. Staff education departments need to be aware of the changes to include them in education sessions for practising nurses.

Cardiac biomarkers can be used for the diagnosis of ACS and for risk stratification. Clinical guidelines are required for timing of specimen collection, specific patient populations, and decision limits (Christenson & Duh, 1999). For some, serial testing is required at various times. Nurses need knowledge of these results and testing times in order to inform patients of the implications of the various results. This will enable patients to be informed of their treatment options and any follow-up that is required.

Knowledge is necessary if patients are to receive appropriate, timely, and cost effective care. Nurses must become aware of the advantages and disadvantages of the various markers. The sensitivity of myoglobin for diagnosis of MI is decreased in 12-24 hours. Thus, patient history of chest pain onset and pattern is important for interpreting results.

Point of care or bedside testing for cardiac markers is a relatively new procedure in which nurses play an important role. Bedside tests for cardiac specific troponins are highly sensitive for early detection of myocardial cell injury in ACS (Hamm et al., 1997). Research has shown that bedside multimarker testing provided better risk stratification for mortality than traditional laboratory testing (Newby et al., 2001). Nurses may be responsible for obtaining blood samples, performing tests, and interpreting results at the bedside.

Education is necessary to ensure accurate and proper interpretation of results. Nurses are also responsible for evidence-based practice. Current literature reviews are necessary to determine the advantages and disadvantages of the various markers.

Case Studies

Case study 1.

A 49-year old male was seen in the ER with a 4-hour history of chest pain. On admission, ECG showed ST elevation in II, III, and AVR. Although he had a previous CABG in 1992, he continued to smoke two packs of cigarettes per day, had hyperlipidemia, increased blood glucose, and was obese. On presentation, his troponin I was 0 (N – 0.0-0.4 mg/L) and myoglobin was 82 (N - 0-115 mg/L). Two hours following, his myoglobin was 292 mg/L. Further testing of troponin at 6 hours revealed a level of 48 mg/L. This case illustrated the importance of serial testing of biomarkers to rule in MI.

Case study 2.

A 48-year old female was seen in the ER with a history of chest pain for 3 days. She had quit smoking 9 years previous. Her history included hypertension and hyperlipidemia. ECG showed some t-wave changes in V 1-3. Her admission troponin was 0 mg/L and myoglobin was 40 mg/L. Her myoglobin at 2 hours was 33 mg/L. Troponin rose at 6 hours to 0.4 mg/L but returned to normal at 12 hours (0.1 mg/L). She went on to have a cardiac catherization which was normal. This case study demonstrated the need for serial markers to rule out MI.

In summary, diagnosis of MI in a timely and efficient manner is enhanced with proper use of cardiac biomarkers. Costs to the health care system can be reduced and patient care improved with prompt treatment and early intervention for cardiac patients. New advances in the development and testing of markers will further improve patient care. The usefulness of these new biomarkers in risk stratification cannot be overemphasized. In patients with unstable angina, prevention of MI is cost effective for the patients as well as for society. Nurses need to keep updated on these latest developments in order to provide evidence-based practice.

Further research is necessary to determine decision limits for MI in patients undergoing surgery. Similarly, protocols are necessary in assessment of reperfusion following thrombolytics and angioplasty using cardiac biomarkers.

For clinical usefulness, the WHO definition for diagnosis of MI as a triad needs to be altered. The use of biochemical markers that are not enzymes in the diagnosis of MI needs to be included.

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Mediastinal Chest Sump Tubes

Following Cardiac Surgery: An Unconventional Method

D. Rebeyka, R.N., M.S.N., CCN(C)
S. Roschkov, R.N., B.Sc.N., M.N. (c)

The practice of using conventional mediastinal chest tubes (MCTs) connected to a closed collection device is commonplace in cardiovascular surgery care settings. The MCT collection device requires a closed system to maintain a negative intrathoracic pressure with the goal of preventing inadvertent trapping of air and blood within the mediastinal space. Despite the proposed integrity of the closed system, there is no guarantee that the suction's negative pressure will prevent cardiac tamponade. In addition, it has been postulated that the required negative intrathoracic suction may potentiate mediastinal tissue damage. This clinical paper will describe the use of multi-lumen MCTs open to atmosphere following surgical repair for congenital heart defects. It is postulated that open MCTs may potentially reduce the risks of cardiac tamponade and mediastinal tissue damage. Through case presentation, the mechanics of open MCTs, nursing care, and possible complications will be delineated.

Key words: mediastinal chest tubes

Contact Information:
G. Urquhart, R.N., M.N., CCN(C)
(780) 407-7464 (work), (780) 407-3321 (fax), gurquhar@cha.ab.ca
5H1.05, 8440 - 112 Street, Edmonton, Alberta T6G 2B7

The insertion of drainage tubes into the mediastinal and/or pleural cavities is an essential standard of care following cardiac surgery. The tubes provide a conduit for drainage and are typically connected to a closed collection system with suction to maintain negative pressure (Gordon, Norton, Guerra, & Perdue, 1997). Traditionally, cardiovascular nurses have developed skills and expertise in caring for patients with the closed chest tube (CT) drainage system. However, despite a patent closed system, there is no guarantee that the suction's negative pressure will prevent occlusion of the CTs or cardiac tamponade. In addition, limited research suggests that pleural pressure may impair left ventricular function (Magder, Lichenstein, & Adelman, 1983; Stahly & Terch, 1977).

Recently, an alternative CT drainage system composed of a mediastinal chest sump tube (MCST) open to atmospheric air has evolved as the preferred method of CT therapy for adult and pediatric patients post congenital heart surgery. The MCST has not been reported in any other patient populations. No studies have been conducted on the efficacy of the open system in the congenital heart surgical patients. However, several theoretical advantages have been proposed and no negative sequale have been reported (Dr. I. Rebeyka, personnel communication, December 1999).

The purpose of this paper is to describe the use of the Axiom filter® MCST system in the immediate postoperative period following adult congenital cardiac surgery. Discussion will include the mechanics of open versus closed MCSTs and the required alterations of the collection device. This will be followed by an overview of nursing care, possible complications, and future research recommendations.

Principles of Mediastinal Chest Tubes

Conventional Mediastinal Chest Tubes

Chest tubes are essential post cardiac surgery to avoid the accumulation of fluid, including blood, or air around the heart, which may result in impairment of ventricular filling leading to cardiac tamponade (Gross, 1993). In addition, excessive collection of fluid and air may cause a pneumothorax or hemothorax, which can compromise ventilation and impair gas exchange (Puntillo, 1996). Typically, a conventional CT is single-lumen and is connected to a disposable collection system, such as the Pleur-Evac® device. The entire system is closed to atmospheric air to ensure there is no inadvertent entrapment of air in the mediastinal or pleural space (Table 1).

Important Considerations for Mediastinal Chest Sump Tubes

Recently, a multi-lumen MCST has been used in the adult congenital cardiac surgical patient to enable removal of air and fluid following cardiac surgery. The MCSTs may be double or triple lumen, and the specific catheter employed is based

Table 1: Comparison of Mediastinal Chest Tubes: Conventional versus Sump

Characteristics	Conventional CT	Chest Sump Tubes
Varying catheter size	✔	✔
Use of disposable drainage system (e.g. Pleur-Evac®)	✔	✔
Use of wall suction	✔	✔
Single lumen	✔	✔
Multi lumen		✔
Open system		✔
Closed system	✔	✔

Note: CT = chest tubes; ✔ = yes; e.g. = for example

on physician preference. This clinical paper will focus on the triple lumen MCST (Figure 1); however, the principles for the double lumen MCST are identical (Table 1). Similar to the conventional CT, the MCST is attached to a disposable system, such as the Pleur-Evac" device.

The triple lumen MCST has one central lumen and two side lumens (Figure 1, see page 22). The central lumen drains fluid, including blood and air from the mediastinal space, into a disposable collection system. The central lumen is closed to atmospheric air while the side lumens may be closed or opened to atmospheric air.

Air Entrapment. When the side lumens are closed, the MCST works like a conventional CT. Typically, the MCST side lumens are open to atmospheric air for the initial six to eight hours (hrs) after cardiac surgery. When the MCST side lumens are open, air is entrained into the mediastinal space. The entrainment of air is designed to lessen the impact of the negative suction on the tissue immediately surrounding the MCST. The air travels up the side lumens and is drawn into the central lumen of the MCST and removed from the mediastinal space. This design prevents the accumulation of air in the thoracic cavity (Dr. I. Rebyeka, personal communication, December 1999). Air that is continuously being removed via the central lumen is believed to decrease the risk of occlusion of the central lumen with blood. Therefore, it is postulated that the risk of cardiac tamponade is lessened (Dr. I. Rebeyka, personal communication, December 1999).

Mechanical Ventilation. During normal inspiration intrathoracic pressure decreases as the diaphragm flattens, which allows air to be entrained into the lungs (Gross, 1993). If the side lumens of the MCST are open to atmospheric air during normal inspiration, entrainment and entrapment of air within the chest cavity may occur. Therefore, the patient requires positive pressure mechanical ventilation when the MCST side lumens are open. Positive pressure ventilation is postulated to decrease the risk of air entrapment, since intrathoracic pressure is positive during inspiration (Dr. I. Rebeyka, personal communication, December 1999). Synchronized intermittent mandatory ventilation (SIMV) is one such mode in which intrathoracic pressure increases during ventilator induced inspiration.

Application of Collection Device. When the MCST side lumens are open to atmospheric air, the Pleur-Evac" system requires minor alterations to ensure air is adequately circulated through the three lumens of the MCST. As well, there must be no accumulation of air within the chest cavity. Similar to the conventional CT system, the suction control chamber is filled to -20 centimeters of water (cmH_20) and is attached to wall suction (Gross, 1993). With the conventional CT system, suction control chamber ports are required to ensure there is no excessive pressure build up within the collection device (Gross, 1993). However, with the open MCST system, the company supplied control caps are removed and the ports are sealed with occlusive tape. Unlike the conventional CT system, where suction is set so there is gentle bubbling within the suction control chamber (Gross, 1993), the open MCST device requires wall suction to be set at maximum. The use of occlusive tape and maximum wall suction is employed to ensure there is entrainment of air up the side lumens, as well as the immediate removal of air and fluid via the MCST central lumen (Dr. I. Rebeyka, personal communication, December 1999). Adequate circulation of air through the MCST is evident when the following three criteria are met: (1) an audible hissing sound via the MCST side lumens, (2) presence of an air leak in the water seal chamber, and (3) flattened and compressed latex tubing of the

Pleur-Evac" (Dr. I. Rebeyka, personal communication, December 1999).

Nursing Care

Nursing care and associated CT management differs depending on whether the MCST side lumens are open or closed to atmospheric air. When the MCST side lumens are closed, no air is being entrained into the chest cavity; therefore, nursing care for the patient is identical when a conventional CT system is used. The MCST are removed when the side lumens are closed; therefore, the procedure is identical to the conventional CT system. Since the focus of this paper is the open MCST system, conventional CT nursing care will be not reviewed.

Nursing care for the open MCST includes three components: (a) preparation, (b) ensuring patency

Table 2: Nursing Care Plan for Open Mediastinal Chest Sump Tubes

Goal	Nursing Care
Preparation of MCST system	☺ Ensure correct set-up of Pleur-Evac® **except** for: (1) supplied suction control chamber caps removed, (2) occlusive tape applied to suction ports, & (3) wall suction set at maximum. ☺ Maximum suction present via: (1) audible hissing sound from MCSTs side lumens, (2) air leak present in water seal chamber, (3) Pleur-Evac® latex tubing flat & compressed & (4) vigorous bubbling present in suction control chamber. ☺ Patient receiving positive pressure ventilation.
Patency & safety of MCST system	☺ Side lumens remain open initial 6 to 8 hrs after cardiac surgery. ☺ Assess a minimum of every hr or with any alteration in CT loss, patient position, or hemodynamic & respiratory status by: (1) suction control chamber filled to minimum of -20 cmH_2O, (2) suction ports occluded with occlusive tape, (3) wall suction set at maximum, (4) vigorous bubbling in suction control chamber, (5) Pleur-Evac® latex tubing flat & compressed, (6) air leak present, & (7) audible hissing sound from MCSTs side lumens. ☺ Ensure no occlusion to MCSTs side ports (e.g., covered by linen). There will be no circulation of air through the MCSTs with the risk of occlusion of the MCSTs central lumen & mediastinal tissue damage. ☹ Ensure MCSTs central lumen and Pleur-Evac® latex tubing not occluded or kinked, as air movement through the central lumen will ↓ or cease, therefore, ↑ risk of air entrapment. ☹ Ensure vigorous bubbling in suction control chamber. If there is no bubbling, air may be entrained up side lumen, yet not removed via central lumen. ☹ Irrigate each MCST side lumens with 10-ml. Sterile NS prn for non-patency. The NS will not remain within the chest cavity and will be removed via the central lumen into the Pleur-Evac® collection chamber. The movement of NS will irrigate the central lumen. ☹ Patient must not be removed from positive pressure ventilation while the MCST side lumens remain open. ☹ Chest assessment (heart & breath sounds) may be difficult due to the hissing sound. Short-term disconnection is acceptable when performing an assessment to obliterate hissing sound. ☹ Pneumothorax may not be evident by typical decreased air entry because of difficult chest assessment. Observe for ↑ ventilator pressures & ↓ oxygen levels.
Conversion to conventional CT	✋ Cover MCSTs side lumens with occlusive tape. 💣 Immediately remove occlusive tape from suction control ports. Re-cap the suction ports with caps provided with the Pleur-Evac® device. 💣 Decrease wall suction immediately until **gentle bubbling** is present in suction control chamber to prevent mediastinal tissue from being entrained into the central lumen. This final step has converted the MCST from an open system to the conventional closed system.

Note: MCST's = mediastinal chest sump tube; hr = hour; cmH_2O = centimeters of water; CT = chest tube; e.g. = for example; ↓ = decreased; ↑ = increased; ml. = milliliter; NS = normal saline.

and safety, and (c) eventual conversion to a conventional CT system. The goals of nursing care are to ensure the central and side lumens are patent and that there is no accidental accumulation of air or blood within the chest cavity. A nursing care plan specific for a patient with an open MCST system, utilizing the wet suction Pleur-Evac" drainage system, is outlined (Table 2, see page 19).

Case Study

A 17-year-old female, Ms. N.J., who had previous surgical repair for Tetralogy of Fallot underwent surgical replacement of her pulmonary valve for severe pulmonic regurgitation. Ms. N.J. required resection of her right ventricular outflow tract to enable accurate fitting of the bioprosthetic valve and a right atria patch due to inadvertent opening of the right atrium with the sternal saw. Femoral-femoral cardiopulmonary bypass was employed over a period of 67 minutes (mins). There was no requirement for cross clamping of the aorta. Two Axiom filter" MCST tubes were inserted prior to closure of the sternum. During the surgical procedure, Ms. N.J. received four units of packed red blood cells and two units of 25 percent (%) albumin.

Ms. N.J. was admitted to the cardiovascular intensive care unit (CVICU) with the MCSTs side lumens open to atmospheric air. The system was functioning properly, as the anticipated air leak, audible hissing sound from the MCSTs side lumens, and compression of the latex tubing of the Pleur-Evac" system were present. The MCSTs required irrigation with 10 milliliters of normal saline 20 mins after admission because of a decrease in the audible hissing sound from the side ports of the MCSTs. No further irrigation was required.

Upon admission to the CVICU, Ms. N.J. was placed on positive pressure ventilation, as required to prevent entrapment of air with within the thoracic cavity. The ventilator settings were as follows: (a) SIMV at a rate of 8, (b) positive end-expiratory pressure (PEEP) of 5 cmH_20, (c) pressure support (PS) of 3 cmH_20, and (d) fraction of inspired oxygen (FiO_2) at 70 %. The patient remained on positive pressure ventilation for 10 hrs and 40 mins, which was slightly longer than the anticipated 6 to 8 hours. During this period, the FiO_2 was decreased to 30 %, although the original settings for PEEP, PS, and SIMV were not altered. All arterial blood gas levels were acceptable.

During the initial 6 hrs after surgery, Ms. N.J. received Propofol (Diprivan) ranging 1 - 5 milligrams (mgs) per kilogram per hour to maintain a modified Ramsey scale of 3 - 5. As well, during the initial 9 hrs and 30 mins, Ms. N.J. received Morphine Sulphate (Morphine) 2 mgs via the intravenous (IV) route 5 times for a total of 10 mgs.

The bedside nurses, to ensure the system was patent, performed hourly assessments of the MCSTs. Assessment included the suction port (occluded with occlusive tape, vigorous bubbling present), wall suction remained at maximum, tubing flat and compressed, and an air leak evident via an audible hissing sound from the MCST side lumens. Nine hours and 15 mins after admission to the CVICU, the physician was contacted regarding inadvertent occlusion of the MCSTs due to decreased CT drainage. Upon assessment, the physician determined the MCSTs were functioning well because of an audible hissing sound via the side lumens, an air leak was present, and there was complete compression of the latex tubing. Does this mean that the hissing and the leak are related or not?

At 10 hrs and 40 mins after admission, the MCSTs side lumens were covered with occlusive tape, while the occlusive tape from the suction control ports were removed. Immediately following, the ports were occluded with the company-supplied caps. All of the steps were required to convert the open MCST device to the conventional CT system. The steps are required to prevent the inadvertent build-up of pressure within the thoracic cavity, which could lead to mediastinal tissue damage or the risk of entrapment of mediastinal tissue into the central lumen of the MCSTs. Following conversion of the open MCST device to the conventional CT system, Ms N.J. was placed on PS at 5 cmH_20 with a FiO_2 of 30%. Blood gas levels were acceptable 160 mins after the MCSTs side lumens were covered with occlusive tape. The patient

Table 3: Determinants of Mediastinal Bleeding

Value	# of Tests	Time Frame	Range	Mean
PT INR	4	19 hr	1.20 - 1.60	1.33
HCT (liter/liter)	6	72 hr	0.31 - 0.38	0.33
Ion Ca^{++} (mmol/liter)	5	24 hr	1.15 - 1.23	1.18
CT loss (ml/hr)	16	16 hr	0 - 30	5.81

Note: # = number; PT INR = prothrombin time international normalized ratio; hr = hour;
HCT = hematocrit; Ion Ca^{++} = serum ionized calcium; mmol = millimoll; CT = chest tube; ml = milliliter.

was extubated and placed on nasal prongs at 5 liters per minute. The MCSTs were removed 288 mins following extubation. Shortly thereafter, Ms. N.J. was transferred to the postoperative cardiac surgical unit.

Minimal MCST blood loss was noted (Table 3) and blood products were required only in the operating room. The remainder of Ms. N.J. postoperative course was uncomplicated, and she was discharged home on postoperative day 5.

Conclusion

Implications for Practice, Education, and Research

This article has addressed the application and management of the MCST system through the use of a case study. With enhanced technical sophistication of the MCST system, there is a need for nurses to formulate accurate assessments and appropriate interventions based on physiological principles that differ greatly from the conventional CT system.

Delivery of optimal nursing care hinges on a thorough understanding of the open MCST design and the relationship to intrathoracic pressure changes during mechanical ventilation. Other important concepts include the prevention and early detection of complications through the maintenance of CT patency and eventual conversion to the conventional CT system. Moreover, it should be noted that comfort levels reported by nurses caring for these patients vary greatly based on the exposure to the sump system. Therefore, ongoing education is the key to attaining and maintaining competency.

As information regarding the MCST system remains in its infancy, numerous opportunities exist to expand the body of scientific knowledge. Additional research is needed to validate the theoretical advantages of decreased tissue damage and cardiac tamponade proposed with the use of the open system. Comparison between the open and conventional closed system in area of hemostasis, hemodynamic instability, perceived pain, safety, and efficiency warrants investigation. Presently, the MCST is used exclusively in the congenital surgery population. It is hoped that the knowledge gained through future studies will help establish practice standards that optimize care for all cardiovascular surgery patients.

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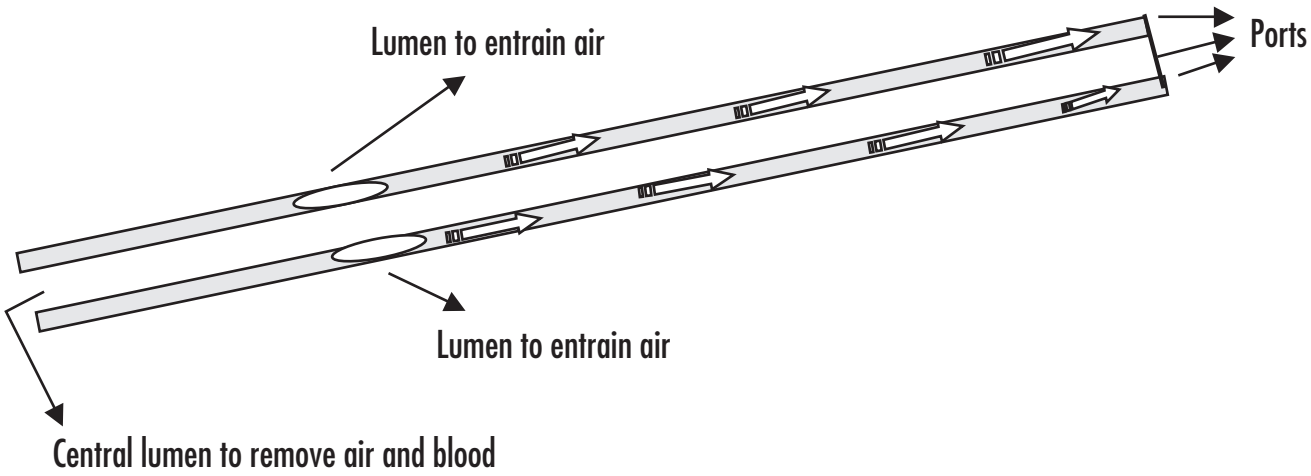
Figure 1: Axiom Mediastinal Sump Chest Tube



(Figured used with permission of Axiom Medical, Incorporated, 2000).

My Abstract was Accepted! Now What?

Cathy Williams, BA[1] and Kathryn King, RN, PhD[2]

Some time ago, a Research Rounds column was dedicated to writing abstracts (King, 1998). Since then, a number of people have commented positively about its utility. It is now time for the sequel. This column will be dedicated to following the steps from abstract acceptance through to the presentation.

The conference planners usually notify authors that their abstract has been accepted some time before the conference date. In this correspondence, information is often included regarding the nature (oral/poster), time allotment, as well as the date and time of the presentation. At minimum, the authors are informed about when they can expect this information to be forthcoming. Although the conference date may seem far in the future, it is important that the authors begin preparing themselves EARLY for the conference presentation.

Oral Format

Content.

Generally, the content of your abstract should be the basis upon which your presentation is developed. The audience has come to hear you speak because they were attracted by your interesting title and abstract in the conference guide (Martin, 2000). It is helpful to begin by identifying the intent of your presentation; that is, presenting a brief outline or overview of what you intend to cover. Most attendees will appreciate having a sense of where you are going in your presentation and what they can expect to hear from you (Noreiko, 1995). If presenting research, an introduction/background to the topic, research questions, methodology (design, data collection methods, data analyses), findings, and conclusions should be the major components of the presentation—just as they were in the abstract. If presenting a clinical or theory-based paper, an introduction to the issue or clinical problem should be followed by identification of how the issue or clinical problem was addressed and identification of appropriate conclusions. The amount of detail you choose to include should be based on the needs or interests of your audience and how much time you have to speak.

Script?

When beginning to make public presentations, some people think it is important to use a script. Others will disagree. The trick is to find out what works best for you. A script has certain advantages in that the presentation will be well prepared and planned. If you are a bit anxious or new to presentation work, a script ensures that you will not struggle for words as the format is well organized. If you do choose to use a script, make sure it is easy to read. Use large font and double space your notes. It may also be helpful to highlight important points that you don't want to miss (Fondiller, 1994) and to mark clearly points when you wish to change your slides or overheads. A drawback with notes is that a person can rely too heavily on them and appear to be 'just reading off the paper.' By comparison, the unscripted version does not mean 'winging it.' The presenter can still make notes about key points illustrated in the visual aids (e.g., slides, overheads, PowerPoint™ presentation). Brief notes should be placed on a print copy of any slides/overheads you are using. Given this choice, it is important to be sure that your notes are comprehensive and that they include all critical points.

Whether you choose to use a script or your slides as prompts, PRACTICE. It is one thing to 'know' what you are going to say; it is another to get the words out. You want a polished presentation—one that is clear and crisp, one in which you avoid mumbling, speaking rapidly, or using jargon, and one that is within the allotted time (Howe, 1994).

Visual Aids.

Your conference organizer will identify what aids will be available for your presentation. Advances in computer technology

have made it very easy to create successful presentations in overhead and slide formats as well as PowerPoint™ format.

There are several factors to keep in mind when designing overheads, slides, and PowerPoint™ presentations. To begin, Evans (2000) suggests choosing a font that is easy to read; Times New Roman, Bookman Old Style, Century Schoolbook, and Arial are recommended. To assure that the font size is easily read from a distance, Evans suggests limiting the amount of text on each slide to no more than five lines (excluding the title); a 40-point font for the title and a 32-point font for the text will usually work. To accomplish this, place only the main points you wish to make about the content on a slide. Also, you will want the audience to listen to your interesting content and not to be busy reading what is projected on the screen.

Your choice of colors in the presentation will have an impact on how your presentation is received. Evans (2000) suggests that colours can connote meaning. For example, red and yellow can bring emphasis to words and are considered 'warm'; dark reds and browns may be linked to masculinity; gold, silver, and black can be associated with wealth; blue and green are considered 'cooling' colours. Depending on your topic, you may want to select a combination of colors that corresponds to the theme of your presentation.

Speakers often use laser pointers to highlight certain aspects of their non-animated presentations. Laser pointers can be very effective and useful tools. However, they can be very distracting if used inappropriately (e.g., when used too often or by a nervous and shaky presenter). If you wish to use a laser pointer, it is helpful to practice!

An important tip to remember is to bring a copy of your PowerPoint™ presentation in another medium (e.g., overheads). It is not uncommon for technological glitches to occur; likewise, plans can change at the last minute for the conference organizers and digital projectors can fail. Overhead projectors are generally available as standbys. This practice could save your valuable presentation.

When using computer-aided projection (LCD), remember that just because certain "fun" features are available through a presentation software package, they should not be necessarily be used in your presentation. In fact, the use of animated text and sound effects can be very distracting and can actually make your presentation difficult to follow (Evans, 2000). Use animation and sound only in those situations where they will enhance the learning experience of the audience. An example of effective animation is the use of bullets that build with the click of a button. This technique allows the viewer to focus on the single concept you are explaining and that accompanies the text.

There are a few more things that you may wish to consider before you get to the podium to speak:

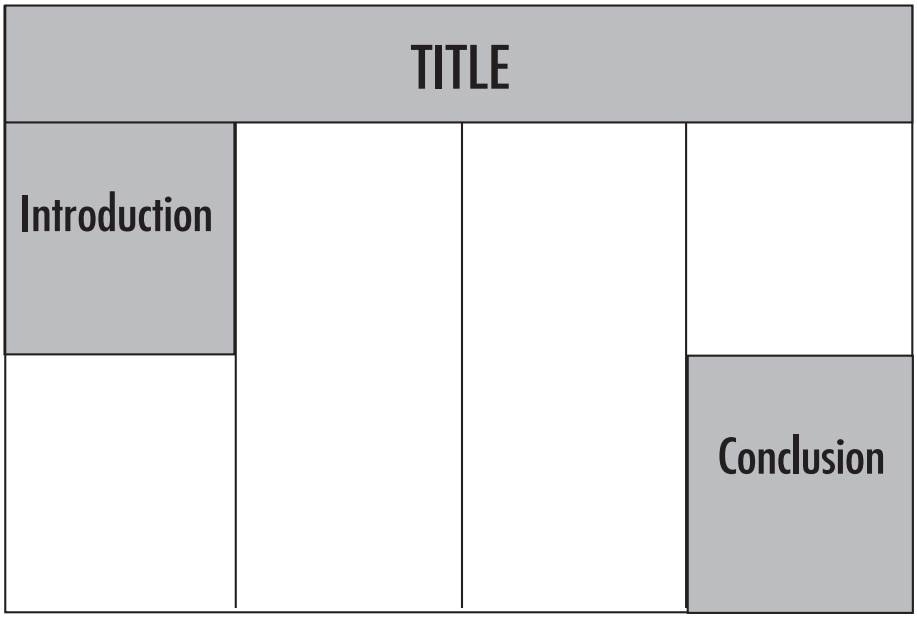
- Remember that body language says a lot about how confident and prepared you are. Try to relax before the presentation and appear enthusiastic in front of the audience (Martin, 2000). Good preparation and practice will help with this.
- Try to introduce a little humour into your talk. This can be in the form of a story or analogy. Humour has a way of making the entire audience relax and identify with you as the speaker (Fallon Smith, 2000).
- When you are at the podium, wear a watch to remind yourself how much time you have available (Fondiller, 1994). Do not let yourself speak over the allotted time. Doing so is disrespectful to your audience and the speakers who follow.
- Try to maintain eye contact with the conference attendees (Robinson-Wolf & Donnelley, 1993). Doing so assures the audience that you are engaged with them.
- When concluding a presentation, make sure you are prepared for questions, and take the opportunity to meet with audience members after your talk. This is an excellent chance to associate with other people who are interested in your topic (Fondiller, 1994).

Poster Format

It is important that your poster be as successful as possible in getting across the main purpose of your study, project, or topic. As with an oral presentation, the elements of the abstract that drew conference participants' attention must be reflected in your poster. The conference organizers will inform you of the size allotment for posters (the size is often 4' x 6'). These dimensions will assist in making decisions regarding how to lay out your information. (See Poster Format layout on page 25)

Radel (1996) recommends the poster layout shown below. Note that the methods and findings (or 'content') occupy the most and central space. The flow of the material should be from top to bottom of three to four columns

Poster Format Layout



of material. You don't need to spend a lot of money to have an effective poster (Moneyham, Ura, Ellwood, Bruno, 1996). An effective poster presentation may be created with PowerPoint™ computer software and colour copying. Thereafter, single panels/slides can be mounted on foam core board or construction paper. If you have access to PowerPoint™ and special printing services, a one-piece poster can be created to the desired size.

Be sure to use a variety of elements (e.g., photos, figures, text) to 'tell the story' (SANHRRU, 2001). Remember that posters are visual presentations. There should be a balance between graphics and supportive text. Note that the visitor to a poster should understand the story or message of the poster in less than five minutes (Biancuzzo 1994a). Thus, it is imperative that only the most important points be included on the poster. When determining poster content, Biancuzzo (1994a) suggests that feedback from colleagues regarding content and formatting is imperative. A fresh viewpoint will assist you in editing ruthlessly. The editing stage is much like practising an oral presentation. The essential question is to keep in mind is this: "Does the poster make sense of the material and flow well?". A poster should also contain a good balance of content and empty space to enable differentiation of poster segments. Some recommend up to 40-50% of empty space (Carter & Nilsson, 2000; Woosley, 1989).

Font style and size are also important elements because the poster needs to be easily read from a distance. As indicated earlier, there are some fonts that have particular 'readability.' The title of a poster can be up to 96-point font whereas the text can be as small as 24- to 36-point font (SANHRRU, 2001). Colour is also a consideration. In a brightly lit hall, muted colours will be more appealing to the eye than will bright, fluorescent colours (Woolsley, 1989). Occasionally, conference organizers will provide setup supplies (e.g., Velcro™, tacks). However, you are wise to bring these with you.

Some poster presenters bring a brochure or one-page outline that captures the main points of the poster presentation. These aids can serve to leave a lasting impression of the main message you are conveying in your poster (Moneyham et al., 1996). If you have business cards, it helpful to have them available to distribute to individuals who have similar interests (Biancuzzo, 1994b).

Some additional points to think about if you are doing a poster presentation are as follows:

- Arrive early to have sufficient time to get your poster set up and organized (Biancuzzo, 1994b; Rempusheski, 1990)
- Anticipate questions that may be posed to you. Ask your peers to assist you with this. Practising will enhance your personal presentation of the material (Biancuzzo, 1994b).
- Actively encourage conversation and discussions. This will also help you create contacts that may be helpful to you later (Troth Lippman & Stonkas Ponton, 1989).

Last Words

Never place your presentation materials in checked baggage.

Remember, you are going to the conference to learn and to meet colleagues who share similar interests. Have fun!

About the Authors

Cathy Williams is a 3rd year student in the Faculty of Nursing, University of Calgary. This paper is based on a class project for Dr. Kathleen Oberle's course in Nursing Research.

Kathryn King is an Associate Professor in the Faculty of Nursing, University of Calgary and the National Research Chairperson for CCCN.

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Permission from the copyright holder for any previously published material (i.e. excerpts, tables and illustrations) that is appearing in the manuscript.

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Follow the APA guidelines for grammar, punctuation, usage (capitalization, numerals, seriation), unbiased language, references and citations. Two exceptions from APA are these: spelling should be current Canadian usage where applicable; abstract may be expressed in a maximum of 150 words.

Tables, graphs, illustrations: Prepare in accordance with Chapter 3 of the APA Manual. Each table, figure or illustration should be submitted on a separate sheet and numbered as it appears in the article (i.e. Figure 1, etc.),
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Four to five keywords from the CINAHL. Subject Heading list should appear on the title page.

Acknowledgements

Sources of funding for the research that resulted in this manuscript should appear in the acknowledgement section of the paper.

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Les disquettes ne sont pas nécessaire avec cette première soumission originale.

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Texte: Tous les manuscrits doivent être conforme au troisième chapitre de la quatrième édition du American Psychological Associations's Publication Manual (APA). Tous les articles doivent obéir à l'APA pour la grammaire, la ponctuation, l'usage (capitalisation, numérotation, catégorisation), le langage nonbiaisé, les références et les citations.

Tableaux, figures, illustration : Doivent être conforme au troisième chapitre du manuel APA. Tout tableau, figure ou illustration doit apparaître sur une page séparée et numéroté d'après son apparition dans l'article (i.e. Figure 1, etc.). Les illustrations doivent être générées par ordinateur ou dessinées de façon professionnelle. Des duplicats de photos doivent apparaître sous forme de copie dans la soumission du manuscrit, et non en montage.

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Indiquer sur la page titre à cinq mots clés de l'article de la banque de donnée CINAHL.

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L'information sur l'aide financière obtenue devrait apparaître dans la section des remerciements.

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