Scholarship for Evidence-Based Practice

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Dan Stuart
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Margaret Vugrin
AGENDA: NRSG 3208, Integrated Learning I  
Place: 1C125A  
Monday, Jan. 29, 2018  
Time: 10:00 - 12:00  
Approx: 81 students  
Contact: Priscilla Stansell, MSN, RN-Bc

<table>
<thead>
<tr>
<th>Time</th>
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<tr>
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<td>Introduction - Priscilla Stansell, Peggy Edwards</td>
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<td>10 min</td>
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Hover on School/Program, select Nursing
PowerPoint for Resources for NRSG 3208
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Click Citation Tools
Click for link to *Basics of APA Style – Video Tutorial*
Click for link to Basics of APA Style – Video Tutorial
Basics of APA Style Tutorial

In this tutorial you will learn how to apply some basic rules of APA Style.

For in-depth guidance on style and for comprehensive information on publishing in the social and behavioral sciences, we urge you to buy the *Publication Manual of the American Psychological Association, Sixth Edition*.

Where various elements of APA Style are described, chapter and section numbers are given to tell you where more details can be found in the *Publication Manual*.

Begin the tutorial
Identifying Citations

A citation is the:

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- title of the article
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Enter heart disease

Suggested Subject Headings

click Search
Click the underlined term **Heart Diseases**
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Select the subheading *Familial And Genetic*
Click Advanced Search
Refining Results
Select English Language and Peer Reviewed
At the bottom of the screen, click Search.
Change date to 2012 in the left-hand box to narrow to articles published in the last 5 years.

Press Return
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Results have narrowed to 11 articles.
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Mechanotransduction and Metabolism in Cardiomyocyte Microdomains

Francesco S. Pasqualini,1,2 Alexander P. Nesmith,1 Renita E. Horton,1,3 Sean P. Sheehy,1 and Kevin Kit Parker1

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2Institute for Regenerative Medicine (IRM), Wyss Translational Center, University and ETH Zurich, Zurich, Switzerland
3James Worth Bagley College of Engineering and College of Agriculture and Life Sciences, Mississippi State University, Starkville, MS, USA

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Efficient contractions of the left ventricle are ensured by the continuous transfer of adenosine triphosphate (ATP) from energy production sites, the mitochondria, to energy utilization sites, such as ionic pumps and the force-generating sarcomeres. To minimize the impact of intracellular ATP trafficking, sarcomeres and mitochondria are closely packed together and in proximity with other ultrastructures involved in excitation-contraction coupling, such as t-tubules and sarcoplasmic reticulum junctions. This complex microdomain has been referred to as the intracellular energetic unit. Here, we review the literature in support of the notion that cardiac homeostasis and disease are emergent properties of the hierarchical organization of these units. Specifically, we will focus on pathological alterations of this microdomain that result in cardiac diseases through energy imbalance and posttranslational modifications of the cytoskeletal proteins involved in mechanosensing and transduction.

1. Introduction

The dynamic range and durability of the heart are enabled by the hierarchical organization of sarcomeres, the organ’s force-generating units [1]. Sarcomeres are highly ordered arrays of molecular motors developed and maintained through a finely regulated mechanotransductive mechanism, sarcomericogenesis [2]. A second mechanism, myofibrillogenesis, ensures that sarcomeres serially register along actin bundles, creating the parallel arrays of myofilaments responsible for the cardiomyocyte striated appearance [3]. Through dedicated cell-cell junctions, myofilaments register across multiple cardiomyocytes creating myocardial sheets that wrap themselves around the cardiac chambers ensuring efficient pumping of blood in the circulation [4, 5].

While the anisotropic organization of the contractile cytoskeleton ensures efficient organ-level contraction, large quantities of Ca2+ ions and adenosine-triphosphate (ATP) molecules are required at the subcellular level for sarcomere contraction. In fact, cardiomyocytes feature a network of dedicated calcium storages, known as sarcoplasmic reticulum, as well as a large number of mitochondria, the organelles responsible for synthesizing ATP molecules from a range of available energy substrates [6]. Specifically, while glycolytic mechanisms are sufficient to meet the organ’s ATP requirements during development, an increased energy demand coupled with an abundance of energy dense fatty acids promotes a shift towards oxidative metabolism as the organism matures. Fatty acid oxidation requires a complex set of enzymes that cluster into mitochondria to effectively participate in the tricarboxylic acid cycle [7, 8].

To ensure rapid and efficient transfer of ATP molecules, mitochondria in cardiomyocytes localize in close proximity with sarcomeres, the sarcolemmal invaginations known as t-tubules, and the sarcoplasmic reticulum. This creates a functional microdomain, termed the intracellular energetic unit.
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- Care Plans (4)
- CEUs (173)
- Books (5,313)
Tour of Material Types
Quick Lesson
Heart Failure: an Overview

Description/Etiology
Heart failure (HF; formerly called congestive heart failure [CHF]) is a progressive clinical syndrome in which the heart fails to pump a sufficient supply of blood to the body due to a functional or structural cardiac disorder. (Note: The term congestive heart failure applies only to decompensated HF with fluid retention.) HF can be classified as left-sided or right-sided, acute or chronic, systolic or diastolic. In systolic HF (also referred to as HF with reduced ejection fraction [HFrEF; defined by an ejection fraction [EF] ≤ 40%]), the left ventricle (LV) is usually dilated and exhibits electrical and mechanical dyssynchrony (i.e., disorganized depolarization that is not followed by a coordinated ventricular contraction) and reduced contractility. In diastolic HF (also referred to as HF with preserved EF [HFP EF; defined by an EF ≥ 50%]), the LV wall increases its thickness and becomes stiff, which reduces the capacity of the LV to relax and fill with an adequate amount of blood prior to each contraction. HF is the endpoint of progression of several types of cardiovascular disease (CVD) when the disorder impairs the systolic or diastolic function. Over time, prolonged activation of neurohumoral compensatory mechanisms causes additional cardiac work and accelerates cardiac decompensation. Initially, only the left side of the heart might be affected, but eventually, the right side of the heart fails as well. Although it can be treated with drugs or heart transplantation, HF is often fatal.

No single test exists for diagnosing HF; the diagnosis is usually based on clinical presentation, patient history, and the results of laboratory and imaging studies, including echocardiogram. The American College of Cardiology and the American Heart Association divide HF into four distinct stages:

- Stage A: no identifiable structural heart disorder or symptoms, but high risk of developing HF
- Stage B: structural heart disorder without symptoms of HF
- Stage C: past or current symptoms of HF associated with structural heart disease
- Stage D: refractory HF requiring advanced support

The New York Heart Association classifies HF into four functional classes based on the response to physical activity. In functional class I, the patient is asymptomatic or tolerates normal activity levels; in functional class II, symptoms occur with ordinary activities; in functional class III, symptoms appear with light activities; and in functional class IV, symptoms occur at rest.

Treatment of HF depends on the stage, type, and etiology of the disease. Underlying conditions, including hypertension (HTN), diabetes mellitus (DM), metabolic syndrome, atherosclerosis, and coronary artery disease (CAD), should be treated or controlled. Lifestyle modifications to reduce the impact of known risk factors (for details, see Risk Factors, below), cardiac resynchronization therapy, and pharmacologic treatment, are the current therapeutic options for patients with HF. In severe refractory cases, implantation of mechanical supportive devices (e.g., left ventricular assist device [LVAD]) or cardiac transplantation are therapeutic options. Medications can include angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), a sinoatrial node modulator, an angiotensin receptor-neprilysin inhibitor (ARNI), beta blockers, diuretics, and inotropic agents. Implantable cardiac defibrillators (ICDs) can be used to prevent arrhythmias and sudden cardiac death.
Evidence-Based Care Sheets
Coronary Artery Disease: Modifiable Risk Factors

What We Know

- Coronary artery disease (CAD) is a potentially life-threatening condition characterized by pathologic changes in the walls of the coronary arteries (i.e., arteries that supply blood to the heart muscle), usually due to a buildup of atherosclerotic plaque, that results in coronary blood flow obstruction. Patients with CAD can be asymptomatic or experience angina pectoris (i.e., chest pain; commonly called angina) or acute myocardial infarction (AMI). Angina is the most common symptom of CAD. \(^{(5)}\) (For information on angina, see Quick Lesson About ... Angina Pectoris)
- CAD is the leading cause of disability and death in developed countries. \(^{(1,2)}\)
- Cardiac risk factors are conditions or behaviors that increase the risk of developing CAD. Although everyone is at risk for CAD to some degree, certain factors substantially increase risk. In general, the greater the number and severity of individual risk factors, the greater the risk of developing CAD. \(^{(1,3,5,7)}\)
- Risk factors for CAD are classified according to whether they can be treated or controlled; categories include modifiable, nonmodifiable, and novel (also called contributory or nontraditional) risk factors. \(^{(5,7)}\) (For information on nonmodifiable and novel risk factors, see Evidence-Based Care Sheet: Coronary Artery Disease: Nonmodifiable Risk Factors; and Evidence-Based Care Sheet: Coronary Artery Disease: Novel (Nontraditional) Cardiac Risk Factors)
- Modifiable risk factors for CAD include smoking, hypertension, dyslipidemia, diabetes mellitus (DM), overweight/obesity, physical inactivity, and certain psychosocial and dietary factors. \(^{(1,2,3,4,5,6,8,9,10,11,12,13)}\)

- Tobacco smoking is among the most important modifiable CAD risk factors. \(^{(1,3,5,14)}\)
  - The influence of smoking on the development of CAD is complex and multifactorial; smoking most likely results in endothelial dysfunction and a hypercoagulable state. \(^{(3)}\)
  - Cigarette smoking is associated with a 2- to 3-fold increase in risk of CAD-related death. \(^{(6)}\)
  - Cigarette smoking-related risk tends to drop dramatically with smoking cessation such that the incidence of CAD in individuals who recently quit smoking is similar to the incidence in those who have not smoked for 2 years. \(^{(3)}\)
- Hypertension (e.g., elevated systolic and/or diastolic blood pressure) accelerates the development of atherosclerotic vascular disease and progression to CAD and is associated with an increased incidence of CAD-related complications. \(^{(1,3,5,14)}\)
  - Long-term hypertension is associated with increased blood vessel stiffness, which causes vessel wall injury and inflammation, accelerating and aggravating the progression of atherosclerosis and the onset of CAD. \(^{(5)}\)
- Dyslipidemia (i.e., higher levels of LDL cholesterol or triglycerides, lower levels of HDL cholesterol, and higher total cholesterol/HDL ratios) is a well-established risk factor for CAD. \(^{(1,3,5,14)}\)
  - Persons who have a high level of total blood cholesterol (> 170 mg/dL), triglycerides (> 150 mg/dL), or LDL cholesterol (> 160 mg/dL), or a low level of HDL cholesterol (< 40 mg/dL) are at increased risk of developing CAD.
Care Plans
### CARE PLAN
Heart Rate, Increased

**Related to:** Check all that apply
- Cardiac disorder
- Trauma/Shock (early stage)
- Stress
- Pulmonary disorder
- Metabolic disorder
- Hypovolemia
- Infection
- Electrolyte imbalance
- Medication side effects
- Other ___________

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<th>Date/Select</th>
<th>Outcome</th>
<th>Select</th>
<th>Interventions</th>
<th>Status: Achieved/Progressing/Not Met (comment for negative variances)</th>
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<td></td>
<td>Patient will regain and maintain normal heart rate</td>
<td>Assess vital signs, heart sounds (noting S3 &amp; S4) and lung sounds (noting adventitious sounds)</td>
<td>Monitor hemodynamic indicators (CVP, RAP, PAP, PWP, SVO2, SVR, MAP) and EKG as ordered; maintain hemodynamic parameters, as prescribed by physician</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Monitor I&amp;O, weight</td>
<td>Administer IV fluids as ordered; maintain optimal fluid balance</td>
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What questions do you have?
EBP Literature from PubMed’s Clinical Queries
**PubMed Database**

- Biomedical & life sciences journal literature - @6,000 journals

- International scope

- Developed and maintained by the:
  
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Evidence-Based Practice is guided by thoughtful integration of the best available scientific knowledge with clinical expertise.
PubMed’s Clinical Queries tool that searches for evidence-based literature
Clinical Queries Searchable by:

- **Diagnosis**
- **Therapy**
- **Etiology** (cause)
- **Prognosis** (prediction of probable outcome)
Clinical Queries Searchable by:

- Diagnosis
- Therapy
- Etiology (cause)
- Prognosis (prediction of probable outcome)
PubMed’s Clinical Queries

uses pre-formulated search strategies with a keyword search

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<th>Sensitive/Specific</th>
<th>PubMed Equivalent</th>
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<td>sensitive/broad</td>
<td>99%/70%</td>
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Enter asthma

Click Search
Results for Therapy and Broad Scope
Click on the Therapy drop-down menu.
Change from Broad to Narrow Scope
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The effectiveness of clinical pharmacist's intervention in improving asthma care in children and adolescents: Randomized controlled study in Jordan.

Almomani BA1, Mayyas RK2, Ekteish FA3, Ayoub AM2, Ababneh MA2, Alzoubi SA4.

Objective: To evaluate the effectiveness of clinical pharmacist's intervention on achieving better asthma control, quality of life and other clinical parameters.

Methods: A prospective randomized controlled study in north Jordan was conducted. Pediatric patients with asthma (aged 7-18 years old) were included and randomly allocated into two groups, intervention and control. Both groups were interviewed at the first visit and followed up twice by phone (at 3 and 6 months). Education was provided to patients and their caregivers in the intervention group only.

Results: Of 206 eligible patients recruited and randomized to our study, 178 patients completed the study (48.3% intervention versus 51.7% control). There were no significant differences in all baseline data between the two groups. We identified significant differences in the improvement of asthma control ($p<0.001$) and consequently pediatric and caregiver quality of life ($p<0.001$) between both groups at the end of study. Significant differences were also detected in clinical parameters ($p<0.05$).

Conclusion: Implementation of clinical pharmacy service can positively influence asthma control, pediatric and caregiver's quality of life, and other clinical parameters.

Practice implications: To maintain a good asthma status, education of pediatric patients and their caregivers should be part of routine assessment during clinic visit.

Keywords: Asthma; Asthma control; Clinical pharmacist intervention; Patient education; Pediatric patients; Quality of life

PMID: 27839892 DOI: 10.1016/j.pec.2016.11.002
The effectiveness of clinical pharmacist’s intervention in improving asthma care in children and adolescents: Randomized controlled study in Jordan

Basima A. Alnowami, Rawan K. Mayyas, Faisal Abu Ekteish, Abeer M. Ayoub, Mera A. Ababneh, Sirin A. Alzoubi

Research paper

Abstract

Objective
To evaluate the effectiveness of clinical pharmacist’s intervention on achieving better asthma control, quality of life and other clinical parameters.

Highlights

• Clinical pharmacist’s intervention can improve asthma care in children.
• Education of patients and their caregivers should be part of routine assessment during clinic visit.
• Collaboration between physician and pharmacist should be encouraged.
The effectiveness of clinical pharmacist’s intervention in improving asthma care in children and adolescents: Randomized controlled study in Jordan

Basima A. Almomania,⁎, Rawan K. Mayyasb, Faisal Abu Ekteishc, Abeer M. Ayoubb, Mera A. Ababnehb, Sirin A. Alzoubi

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Asthma control
Pediatric patients
Clinical pharmacist intervention
Patient education
Quality of life

ABSTRACT

Objectives: To evaluate the effectiveness of clinical pharmacist’s intervention on achieving better asthma control, quality of life and other clinical parameters.
Methods: A prospective randomized controlled study in north Jordan was conducted. Pediatric patients with asthma (aged 7–18 years old) were included and randomly allocated into two groups, intervention and control. Both groups were interviewed at the first visit and followed up twice by phone (at 3 and 6 months). Education was provided to patients and their caregivers in the intervention group only.
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Conclusion: Implementation of clinical pharmacy service can positively influence asthma control, pediatric and caregiver’s quality of life, and other clinical parameters.
Practice implications: To maintain a good asthma status, education of pediatric patients and their caregivers should be part of routine assessment during clinic visit.

1. Introduction

Asthma is associated with an incredible economic and health care burden for both patients and society [1]. Poor asthma control and exacerbations increase the risk of patients’ mortality and morbidity [2]. Improper handling of inhaler devices is among the most common causes of uncontrolled asthma and is associated with frequent emergency department visits [3]. Lack of adherence to medications is another important factor and it has been associated with severe asthma exacerbation [4]. Other factors affecting asthma control include the availability of different types of inhaler devices and insufficient time spent by physician to teach and examine patients inhalation technique [5].

In Jordanian hospitals, there is a strong coordination between physicians, nurses and pharmacists to provide the best management plan. Physicians are responsible for treating asthma patients and teaching them the proper use of inhaler devices. Nurses provide assistance to physicians and pharmacists dispense the prescribed medications. However, hospitals are overcrowded and outpatient clinics workload prevents physicians from providing appropriate education for their patients. Clinical pharmacists can play a significant role in offering better asthma control in clinical practice [6,7]. Additionally, they can improve patients’ knowledge about disease and medications [8] and encourage patients on self-management and monitoring the early symptoms of asthma exacerbation [6]. Furthermore, clinical pharmacist can assist in improving adherence to asthma guidelines [9,10], employing the correct inhalation technique [11] and improving other humanistic outcomes such as asthma patients’ quality of life [12].

Only few studies have assessed the role of clinical pharmacist in children and adolescents with asthma [7,12,13]. Importantly, these
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