EDITOR'S MESSAGE
Winter Wonderland

RESEARCH ARTICLE
The Use of Occipital Nerve Blocks & Trigger Point Injections in Headaches with Occipital Tenderness

REVIEW ARTICLES
Osteopathic Considerations in the Infections of the Respiratory Tract
Knee Pain in Adults with an Osteopathic Component
Not a Peep: Delirium in the Geriatric Patient

BRIEF REPORT
Underlying Appendicitis Leading to Chorioamnionitis in Preterm Rupture of Membranes

CLINICAL IMAGES
Bilateral Painless Eye Lesions

PATIENT EDUCATION HANDOUT
Respiratory Infections
OFFICIAL CALL • 2017 CONGRESS OF DELEGATES
OF THE AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

You are hereby notified that the ACOFP Congress of Delegates will convene on March 15-16, 2017 at the Gaylord Palms Resort & Convention Center in Kissimmee, Florida.

Credentialing of Delegates and Alternate Delegates will take place on the afternoon of Wednesday, March 15 before the start of Session I, and Session II which will convene on the morning of Thursday, March 16.

Each ACOFP Affiliate State Society shall certify the names of its Delegates and Alternate Delegates to the ACOFP Executive Director by February 1, 2017.

Any reports, resolutions, or other business for this meeting should be submitted by February 13, 2017 to Annie DeVries at annied@acofp.org so that they can be posted on the ACOFP website to allow Delegates to review in advance.

Elizabeth A. Palmarozzi, DO, FACOFP
Speaker of the Congress of Delegates
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<tr>
<th>EXAMS</th>
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<tr>
<td>Family Medicine / OMT Certification / OCC</td>
<td>AOA OMED Conference</td>
<td>April 1, 2017</td>
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<tr>
<td>Performance Evaluation Only</td>
<td>Philadelphia, PA</td>
<td>Late fee through June 1</td>
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<td>Family Medicine / OMT Certification / OCC</td>
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If you have questions, please call 847.640.8477 or email aobfp@aobfp.org.
2017 CALL FOR PAPERS

Osteopathic Family Physician is the ACOFP's official peer-reviewed journal. The bi-monthly publication features original research, clinical images and articles about preventive medicine, managed care, osteopathic principles and practices, pain management, public health, medical education and practice management.

INSTRUCTIONS FOR AUTHORS

Reserve a review article topic today by emailing ACOFP Managing Editor, Belinda Bombei at belindab@acofp.org. Please provide your name and the review title you would like to reserve. Once you reserve a review article topic, you will receive an email confirmation from ACOFP. This will initiate a three-month deadline for submission. If the paper is not received within three months, the system will release the review article topic for other authors to reserve.

Articles submitted for publication must be original in nature and may not be published in any other periodical. Materials for publication should be of clinical or didactic interest to osteopathic family physicians. Any reference to statistics and/or studies must be footnoted. Material by another author must be in quotations and receive appropriate attribution.

ACOFP reserves the right to edit all submissions. Visit ofpjournal.com to view author guidelines, policies, and manuscript checklist.

CLINICAL IMAGES

We are seeking clinical images from the wards that covers essential concepts or subject matter to the primary care physician. Please provide a brief synopsis of how the case presented along with 1-4 questions and approximately 1 page of education with reference to the image and questions.

RESEARCH PAPERS

We are seeking original clinical or applied research papers. Original contributions include controlled trials, observational studies, diagnostic test studies, cost-effectiveness studies, and survey-based studies. The OFP will accept basic scientific research only if the work has clear clinical applications. For randomized controlled trials, study flow diagrams must be submitted. For all other types of original contributions, flow diagrams are encouraged. Original contributions should be 3000 words with no more than 50 references and 5 tables or figures. OFP requires you to submit a 250-word abstract, along with four to six keywords.

The content should include the following:

Abstract | Introduction | Methods | Results | Discussion | Conclusions | Acknowledgements

REVIEW ARTICLE TOPICS:

» Advances in Skin Care Diagnosis & Treatment

» Newborn Disorders & Nutritional Guidance

» Direct Primary Care: Emerging Practice Alternative

» Skin and Soft Tissue Infections: It's More than Just MSRA

» Patient Engagement (Help define the science of engaged research, provide tangible examples of the impact of engaged research, or answer a question or controversy related to patient engagement.)
EDITOR’S MESSAGE

Winter Wonderland
Amy J. Keenum, DO, PharmD

FROM THE PRESIDENT’S DESK

Payment Readiness - Part III: Quality Reporting/Improvement & Resource Use
Larry W. Anderson, DO, FACOFP dist.

RESEARCH ARTICLE

The Use of Occipital Nerve Blocks & Trigger Point Injections in Headaches with Occipital Tenderness
Samuel Madore, DO; Mitchell K. Ross, MD; Amber Hayden, DO

REVIEW ARTICLES

Osteopathic Considerations in the Infections of the Respiratory Tract
Sheldon Yao, DO; Nardine Mikhail, OMS III; George Koutsouras, OMS III; Allison Coombs, OMS III; Michael J. Terzella, DO

Knee Pain in Adults with an Osteopathic Component
Rohan Datta, OMS III; Lyudmila Burina, OMS III; Filippo Romanelli, OMS III; Theodore B Flaum, DO, FACOFP

Not a Peep: Delirium in the Geriatric Patient
Ronna New, DO, FACOFP

BRIEF REPORT

Underlying Appendicitis Leading to Chorioamnionitis in Preterm Rupture of Membranes
Jennifer Gibbs, DO; Firas Bridges, MD; John J. Vullo, DO; Anthony Sampino, DO

CLINICAL IMAGES

Bilateral Painless Eye Lesions
Craig Bober, DO

CALENDAR OF EVENTS

2017 Calendar of Events

PATIENT EDUCATION HANDOUT

Respiratory Infections
OSTEOPATHIC FAMILY PHYSICIAN SPECIALTY PEER REVIEWERS

Dana Baigrie, DO
Clinical Images

Jeffrey Benseler, DO
Radiology

Shagun Bindlish, MD
Diabetes and Endocrinology

John Bissett, DO
Clinical Images

Warren Bodine, DO
Sports Medicine & Family Medicine

Grace Brannan, PhD
Statistics/Design

Natasha Bray, DO
Ethics

Rob Danoff, DO
Emergency Medicine, Preventive

Robin Devine, DO
Statistics/Design

Brian Downs, DO
HIV, Wound Care

G. Scott Drew
Dermatology

Dennis Eckles, DO
Diabetes, Rural Medicine

Gail Feinberg, DO, FACOFP
Academic

Robert Grubb, DO
Sports Medicine

Rose Hall, DO
Family Medicine

Nadia Hasan, DO
Clinical Images

Richard Januchowski, DO
Rural/Underserved

Ronald P. Januchowski, DO
Military & Rural/Underserved

Holly Kanavy, DO
Dermatology

Amy Keenum, DO, PharmD
Healthy Literacy, International & Patient Education

Uzma Khan, DO
Family Medicine

Sarah Mitchell, DO
Family Medicine

Wadsworth Murad, DO
Psychiatry

Merideth Norris, DO, FACOFP
Addiction

Michael O’Connell, DO
Pain, Rehabilitation, Musculoskeletal, Neurology, & Sports Medicine

Prabhat Pokhrel, MD, MS, PhD, FAAFP
Pharmacology, Cardiology, Nephrology, Pulmonology

Joseph Reyes, DO
Pain Management

Bernadette Riley, DO
Medical Education, Academic, Simulation Medicine, Physician Leadership, Health Policy

Mark Rogers, DO, MA, CAQSM, FAAFP
Family Medicine, Sports Medicine, OMM, Medical Ethics

Lawrence Sawicki, DO
Clinical Images

Jay Shubrook, Jr., DO, FACOFP
Endocrinology

Leslie Siewwen, MD
Community Medicine

Daryn Sleuwen, MD
Pulmonary

Lindsay Tijaattas-Saleski, DO
Clinical Images, Emergency Medicine

Michael Watkins, DO
OB/GYN & Women's Health

Stuart Williams, DO
OMM

Barbara Wolf, DO
Psychology

William Woolery, DO, PhD, FACOFP
Geriatrics

Julian Vega, DO
Clinical Images

Peter Zajac, DO, FACOFP
Patient Education

2017 STUDENT PEER REVIEW & WRITING INTERNS

Vaidehi Ambai
Philadelphia College of Osteopathic Medicine

Kristen Constantine, MPH
Lake Erie College of Osteopathic Medicine

McKenzie Denton
University of Pikeville –Kentucky
College of Osteopathic Medicine

Ashton Dixon
University of Pikeville –Kentucky
College of Osteopathic Medicine

Nicole Findlay
Texas College of Osteopathic Medicine

Matthew Hadfield
Liberty University College of Osteopathic Medicine

Robert Malinak
University of Pikeville –Kentucky
College of Osteopathic Medicine

Sujith Modugular
University of Pikeville –Kentucky
College of Osteopathic Medicine

Benjamin Oldach
Ohio University College of Osteopathic Medicine

Thomas Thacker
University of Pikeville –Kentucky
College of Osteopathic Medicine

Jordan Wong
University of Pikeville –Kentucky
College of Osteopathic Medicine

INSTRUCTIONS FOR AUTHORS:

Articles submitted for publication must be original in nature and may not be published in any other periodical. Materials for publication should be of clinical or didactic interest to osteopathic family physicians. Any reference to statistics and/or studies must be footnoted. Material by another author must be in quotations and receive approipropriate attribution. ACOFP reserves the right to edit all submissions. To submit a manuscript or to access additional submission guidelines visit mc04.manuscriptcentral.com/ofp.

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NOW SEEKING
CLINICAL IMAGES

This section showcases clinical images from the wards that cover essential concepts or subject matter to the primary care physician.

Each installment of “Clinical Images” comprises 1 or 2 medical images along with a brief synopsis of how the case presented along with 1-4 questions and approximately 1 page of education with reference to the image and questions.

Submissions should be submitted online at ofpjornal.com via our Scholar One publication process.
The holidays are over and winter has dug in. Snow for some and colder temperatures for others throughout the United States. Respiratory illnesses abound.

So appropriately, our lead article this month is Osteopathic Consideration in the Infections of the Respiratory Tract. It emphasizes the use of osteopathic manual medicine but mentions risk analysis tools and references antibiotic articles. For additional consideration, it reviews the models of osteopathy including biomechanical considerations, the respiratory-circulatory model, and the metabolic-energy model. Neurological and behavioral considerations are also discussed as are other approaches to consider among the five osteopathic models when treating respiratory infection.

Another osteopathic focused article this issue is Knee Pain in Adults with an Osteopathic Component. The article reviews the structure, function, diagnosis, and treatment of knee pain in adults along with reviewing the anatomy, injury risk factors, and osteopathic structural exam management. Other topics highlighted are who needs imaging, immediate treatment, drugs for pain relief and osteopathic manual therapy and or physical therapy. It is well organized and worth a read.

With our growing geriatric population, Not a Peep: Delirium in the Geriatric Patient is a timely research paper. Most of the research takes place in the hospital but most of the delirium likely does not take place there. Delirium can be loud with the patient screaming or quiet or a combination of both. Drugs are the first suspects when seeking a cause but infection, environmental factors, cognitive impairment and lack of sleep are other factors. Finding the underlying cause is key.

Occipital nerve and trigger point injections are discussed in Use of Occipital Nerve Blocks. The article that states most of the patients were helped. While the article gives the reader a clear description of how to do the injections, the assessment tool was not clearly outlined. The reader is left to assume the physician asked the patient at various times after the injections if they were helped but this is not clear.

Underlying Appendicitis Leading to Chorioamnionitis in Preterm Rupture of Membranes is a brief report of a complex patient case that included acute appendicitis, chorioamnionitis and preterm premature rupture of membranes (PPROM.)

We continue our clinical image category with Bilateral Painless Eye Lesions, which includes images and an in depth discussion.

Keep warm.
Extract
Extract patient outcomes data, tests, well-care visits, vaccines, etc. from your EMR

Actionable Reporting
Actionable reporting on 20 categories of care; over 200 total measures

Avoid Penalties
Avoid penalties by reporting data to CMS to meet quality reporting requirements (PQRS)

Improve Quality of Care
Improve quality of care by viewing your patients’ data vs. CMS benchmarks

Identify Patients
Identify patients who have missed appointments, are due for annual wellness visits, or need to have tests done

Segment Patients
Segment patients by age, disease, testing, etc. to view and act on those at highest risk

Enhance Workflow
Use Quality Markers 7.0 to enhance workflow and pre-plan for patient visits

1A subset of Quality Markers measures qualify for PQRS and QCDR reporting.
2Provider is responsible to register with CMS as necessary and to have available the necessary data points for reporting requirements.
FROM THE PRESIDENT’S DESK

Payment Readiness, Part III: Quality Reporting/Improvement & Resource Use

Larry W. Anderson, DO, FACOFP dist.
2016 - 2017 ACOFP President

QUALITY PAYMENT PROGRAM (QPP)

Centers for Medicare and Medicaid Services (CMS) released its 2017 Final Rule on the new Quality Payment Program (QPP) on October 14, 2016. With the final rule, CMS eased some of the requirements for Quality Reporting and Resource Use. This was in response to many organizations, including ACOFP, insisting that solo and small practices would be disadvantaged by the original proposed rule.

If you do not meet the threshold for Medicare patients, you are exempt from the CMS Quality Payment Program. If you are not in a CMS certified Advance Payment Model (APM) (see #4, right) you will be in the CMS Merit-Based Incentive Payment System (MIPS). The remainder of this article will be about the requirements for the MIPS program for calendar year 2017.

In 2017, Quality will account for 60% of an Eligible Professional’s/Group’s Composite Performance Score (CPS). Resource Use will account for 0% of the CPS (for 2017 only). Resource use will still be reported to CMS via normally administered claims. No additional steps are required. The data from EP will be analyzed and used as a “benchmark” for 2018 Resource Use comparison. Review the four CMS categories in Table 1 below, which will comprise the 2017 Composite Performance Score.

PICK YOUR PACE

For the calendar year of 2017, CMS is using a “Pick Your Pace” approach to Quality Reporting. There are four ways you can avoid a non-reporting penalty, and potentially gain incentives of plus 4%.

1. Report quality on at least one individual or PCP measure for any period of time. This documents to CMS that you have the ability to correctly report quality for your practice or group. By doing this, you will avoid a non-reporting penalty, but will not be eligible for an incentive payment.


4. If you are in an Advanced Alternative Payment Model (APM), you automatically qualify for a 5% incentive payment for 2017. These risk-sharing models include: Medicare Shared Savings Program (MSSP) Tracks 1 and 2; CPC+ Model (this is a demonstration project by CMS, and will reopen to new participants soon), Next Gen ACO, Pioneer ACO, Chronic Kidney Care Model, and Oncology Care Model. The PCMH model is not currently qualified by CMS this year, but a new model will be launched by NCQA in March 2017. This model should meet CMS requirements for an APM.

If you choose not to report at all, you will receive a maximum penalty of negative 4% which will be deducted from your Medicare Part B payments.

Table 1: The Four CMS Categories Used to Determine an EP’s Composite Performance Score

<table>
<thead>
<tr>
<th>Measurements</th>
<th>2017 - Percentage of CMS Composite Performance Score</th>
<th>Possible Point Score</th>
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<tr>
<td>Quality Reporting</td>
<td>60%</td>
<td>70 points</td>
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<tr>
<td>Resource Use</td>
<td>0% for 2017 - Benchmark Year</td>
<td>No points for 2017</td>
</tr>
<tr>
<td>Advancing Care Information (ACI)</td>
<td>25%</td>
<td>100 points</td>
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<tr>
<td>Previously Meaningful Use</td>
<td></td>
<td></td>
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<tr>
<td>Clinical Practice Improvement Activities</td>
<td>15%</td>
<td>40 points</td>
</tr>
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</table>

From the American College of Osteopathic Family Physicians.
REPORTING & AVOIDING PENALTIES

The first step to reporting your quality is to select at least one individual or Primary Care measure to report on. You can make your selection within your EMR system (contact your EMR vendor to learn how). If you do not have an EMR system, you still can report using your Medicare claims. Record the Quality Data Codes (QDC) for reimbursement on the Medicare Claim Forms. Follow the guidance in the 2016 Physician Quality Reporting System (PQRS): Claims-Based Coding and Reporting Principles. Contact Debbie Sarason† for a copy of the document.

In closing, a number of members have contacted me with news that they received a letter from CMS at the close of 2016. The letter stated that they were subject to a negative 2% payment penalty on Medicare Part B payments for 2017, which was due to not reporting to CMS in 2015. This will be an annual occurrence, with increasing penalties, if you choose not to follow the these guidelines.

Leverage the 2017 Payment Ready Toolkit and the information that is provided in the weekly President’s Newsletter to avoid the penalty for 2017 (impact seen in 2019). Subscribe to ACOFP Quality Markers 7.0™ to fine tune your ability to identify and intervene in the treatment of those patients who are pulling your quality score down. Seamlessly report your measures through Quality Markers’ CMS approved QCDR registry to insure your measures reach CMS in the right format and on time. The reporting fee is included in the annual subscription price. Go to acofpqualitymarkers.org for more information and a subscription form.

Larry W. Anderson, DO, FACOFP dist.
ACOFP President

REFERENCES:

*For those EP’s who see less than 100 Medicare Part B patients, or receive less than $30,000.00 in revenue from these patients, these EP’s are exempt due to "low volume threshold" from the CMS Quality Payment Program requirements.

†Debbie Sarason
ACOFP Manager of Practice Enhancement & Quality Reporting
debbies@acofp.org | 847-952-5523

RESOURCES

If you need assistance in selecting an EMR system which is best suited for your practice, helpful advice is available at no charge from Software Advice, www.softwareadvice.com. (See category “Electronic Medical Records). They can help you select an EMR from over 300 vendors in 10-15 minutes. Ph. (844) 686-5616.

More information is located at www.acofp.org under “Practice Enhancement.” View the 2017 Payment Ready Toolkit at www.acofp.org/PaymentReadyToolkit to find out more information and instructions on all CMS payment requirements.
The Use of Occipital Nerve Blocks & Trigger Point Injections in Headaches with Occipital Tenderness

Samuel Madore, DO,1 Mitchell K. Ross, MD,2 & Amber Hayden, DO3

1Maine-Dartmouth Family Medicine Residency, August, Maine
2Central Maine Medical Center, Auburn, Maine
3New Hanover Regional Medical Center, Wilmington, North Carolina

INTRODUCTION

Despite the many advances in pharmacotherapy and our understanding of the biologic mechanisms involved, headaches (HA) still remain a difficult condition to treat in many. Patients desire treatments that offer near complete pain relief with minimal side effects, however current medications on the market do not offer that for many patients. When pharmacotherapy and lifestyle changes fail, physicians have turned to needle based therapies. Occipital nerve blocks (ONB) and trigger point injections (TPI) are examples of these therapies and have been shown to provide significant relief to patients suffering from difficult to treat headaches.1,2

RELEVANT ANATOMY

The nerves implicated in occipital neuralgia and often the occipital tenderness of migraines are the greater, lesser, and rarely the third occipital nerve. The greater occipital nerves (GON) receive sensory input from a large part of the posterior scalp bilaterally and innervates the semispinalis capitis. This nerve originates from the dorsal rami of C2, travels superiorly towards the occiput while passing through the belly of the semispinalis capitis muscle and becomes subcutaneous after passing through the aponeurosis of the trapezius (Image 1). Multiple anomalies of the nerves course have been noted. A cadaveric study found that in 16.7% of subjects the GON passed through the trapezius muscle and in 6.7% of subjects it pierced the inferior oblique muscle.3 Clearly, there are multiple areas within the course of the GON and its cited variations that leave it vulnerable to irritation, compression, and entrapment resulting in head pain.

The lesser occipital nerve (LON) originates from C2 -3 and innervates the skin of the posterior auricular and lateral neck regions. It ascends the scalp subcutaneously after passing around the posterior border of the sternocleidomastoid muscles. A cadaveric study has demonstrated that the LON actually pierces the sternocleidomastoid, instead of wrapping underneath, in 13% of cadavers.4 This variation potentially leaves the nerve susceptible to irritation from spasm or overloading of the muscle that can occur in a forward head posture.5

The third occipital nerve arises off the C3 and provides sensory innervation of the posterior neck and scalp. It is not commonly treated with nerve blocks for headaches.4

Keywords:
Occipital Nerve Block
Trigger Point Injection
Occipital Neuralgia
Migraine
Neurology
Procedural Medicine

Introduction: Occipital nerve blocks and trigger point injections are often used to treat headaches of various etiologies. The extent and duration of benefit from these injections reported in the literature varies widely. In one community neurology clinic, patients who receive these therapies often report reduced pain and improved quality of life lasting two to three months after treatment.

Methods: A retrospective chart review of patients who received occipital nerve blocks in a single neurologists office during the dates of January to July 2014 was performed.

Results: Seventy-one patients were treated in the study. Eighteen were treated with occipital nerve blocks alone while fifty-three received nerve blocks and trigger point injections. Overall, both groups had a median length of benefit of 8 weeks and 91% of patients received benefit. The group who received occipital nerve blocks with trigger points injections had an average increase in benefit of less than one week compared to nerve block only.

Conclusions: The effectiveness and low side effect profile of occipital nerve blocks make it a useful therapy in patients with difficult to control headaches. In this study, the addition of trigger point injections did not lead to a significant increase in length of benefit over occipital nerve blocks alone. The inclusion criteria of occipital tenderness may be responsible for the higher response rate of these nerve blocks compared to prior studies.
RELEVANT RESEARCH

On review of the literature, there is little data demonstrating the long-term effects of ONB on occipital neuralgia. One study of ten people who received ONB containing bupivacaine and steroids for occipital neuralgia showed 40% received complete HA relief for one week or less, 40% for two to four weeks and 20% for ten to sixteen weeks.6

Another study involved five hundred patients with idiopathic headaches, of which 48% of these headaches were reportedly due to irritation of the GON. Two groups of patients, those with migraines and those with occipital neuralgia, received lidocaine and methylprednisolone injections into the GON region. Both groups showed similar results; roughly 88% of patients in each group became headache free for a mean of 32 days.7

In migraines, ONB has shown varying results as both an abortive and prophylactic treatment. Studies vary in their selection criteria, doses and types of injected solutions, and endpoints. The percentage of migraine sufferers who receive benefit ranges, in most studies, between 45%-85%.1,8 While there is a wide range in the percentage of patients that receive benefit as well as the length of such benefit, the research shows the ONB is effective in reducing pain in the majority of migraine sufferers. One study involved patients with migraines who were having 15 headache days per month that were relatively treatment refractory. These patients received injections containing local anesthetic and methylprednisolone to the greater occipital nerve on the affected side. Of the fifty-four patients, twenty-six (48%) received complete or partial relief lasting a mean of nine and sixty one days, respectively. The authors found that tenderness of the GON was significantly associated with a positive response and that this may be useful in selecting out which patients are more likely to benefit.9

ONB can be a diagnostic tool to determine if the patient has occipital neuralgia and is often used to treat migraine and other types of headaches.1,10 Cervicogenic, cluster, post concussive, hemicrania continua, and migraine headaches have been shown to improve with ONB while tension-type headaches and medication rebound headaches do not have sufficient evidence to support its widespread use.1,11,12,15 The author often uses ONB to treat patients who suffer from migraine if they have occipital tenderness and standard treatments have failed. Occipital tenderness is a common symptom among many forms of headaches. Migraines and occipital neuralgia can cause patients to have pain in the neck, shoulders, occiput, and retro-orbitally in addition to nausea, vision impairment and dizziness. It has been suggested that irritated occipital nerves could be a trigger for migraines due to the convergence of C2 nerves and the trigeminal system.14 While there is little data on the prevalence of occipital neuralgia, it is suggested by some researchers that there is considerable overlap between it and the diagnosis of migraine.14

In addition to anesthetizing the occipital nerves, injecting and deactivating trigger points (TrP) is a useful technique. TrP are focal, hypertonic areas of skeletal muscle that are tender to palpation and can cause radiation of pain to distant sites or have a twitch when grasped. They have been shown in multiple studies to be increased in numbers or severity in patients with migraines and tension type headaches.1,2,15 A study showed that 93% of patients with migraines had cervical or cephalic trigger points compared to 29% of headache free controls. Palpation of those points actually caused a migraine in 30% of patients.16

Treatment of those points using anesthetic injection has been shown to reduce the frequency and severity of multiple headache types.9,17 When a TrP is palpatied, often times the patient will feel pain or an odd sensation in some of the areas that they feel their headaches. Common TrP in headache syndromes are found in the trapezius, temporalis, sternocleidomastoid, semispinalis cervicis, and splenius cervicis muscles.9,15

In a community neurology clinic that often sees patients in consultation for headaches, both treatments are commonly used if the patient still has a significantly reduced quality of life after medication trials. Patients who receive these injections often claim they wish they had been offered the procedure long ago. The purpose of this study was to determine the percentage of patients who received benefit, categorize the length of this benefit, and to evaluate if the addition of trigger point injections led to improved responses.

METHODS

A retrospective chart review studied patients who received occipital nerve blocks and trigger point injections from the date of January 2014 to July 2014 in a single community neurology office. Inclusion criteria for this study included being diagnosed with occipital neuralgia or migraine by the examining neurologist, tenderness of the occipital region on exam, and ONB performed during that visit. Exclusion criteria were incomplete follow up note or lack of follow up within 3 months after the procedure. Outcomes were measured at follow up visits or by phone. Timing of initial follow up varied among patients but was between 8 and 12 weeks after the procedures. If patients had continued benefit on the first follow up, their data was recorded for a total of 6 months post procedure.
if seen again in the clinic. On each follow up, patients were asked about any side effects from the procedures and asked to give a percentage value of the reduction in severity and frequency of headaches compared to pre-treatment.

The authors defined benefit as patient reported reduction in frequency or severity of headaches by at least 50%. Fifty-nine of the procedures were performed by a single neurologist and the remaining by two medical students under the direct supervision of this neurologist. Prior to these therapies, patients had already undergone medication trials and lifestyle modifications and were not asked to make any additional changes during the time of the study. Data was analyzed by determining the median and mean length of benefit.

THE PROCEDURES

Patients were placed in a seated position with the clinician standing behind them and landmarks were palpated. The injection of the GON occurs 1/3 the way along a diagonal line from the occipital protuberance towards the mastoid process. The injection site for the lesser occipital nerve block is 2/3 the way towards the mastoid process on the same line. The injected solution is 4ml of 0.5% bupivacaine plus 80mg of methylprednisolone suspension and 0.5–7.5 cc of this is injected into each landmark using a 30 gauge needle. All patients received greater occipital nerve blocks bilaterally and received lesser occipital nerve blocks if tender over the corresponding area. No patients received blockade of the third occipital nerve.

The clinician then examined for trigger points in the upper trapezius and cervical musculature bilaterally and injected them with .5–7.5 cc of the remaining solution. The number of trigger points injected was not recorded. Detailed information on trigger points and their treatment have been published.18

RESULTS

Overall, patients in both groups experienced a median benefit of 8 weeks and 91% of all subjects obtained benefit (see Table 1).

Of the ONB/TPI group, forty-eight of fifty-three patients (90.5%) received benefit with a mean of 9.1 weeks. Of the ONB only group, seventeen of eighteen patients (94%) received benefit with a mean of 8.6 weeks.

Thirteen patients who arrived to the clinic with a headache claimed resolution of it prior to leaving office. From this retrospective chart review it is not known truly how many had headache on arrival.

DISCUSSION

The benefits obtained in this study for patients with migraine headaches or occipital neuralgia appears to be greater than some studies have reported.19 Patients who receive these injections claim to have an overall improvement in quality of life as well as require less abortive medicines and ER visits. Some patients say this is the first time they have had a headache free week in years. One forty-two year old female left the clinic almost headache free after she reported fifty-three straight days of head pain.

Occipital nerve blocks and trigger point injections deserve a spot in the armamentarium of the clinician to treat headaches. The benefit of the procedures in this study are the ease of administration, low rate of adverse events, low cost and high availability of the materials. Each patient with a headache should be evaluated for occipital tenderness and cervical/upper thoracic trigger points. If occipital tenderness is present, ONB could be offered to patients. Other therapies for occipital neuralgia and migraine include pharmacotherapy, Botox injections, nerve stimulators, and lifestyle modifications. If ONB is used as a diagnostic tool for occipital neuralgia, an alternative diagnosis should be sought for if there is successful anesthetization of the GON, as evidenced by decreased sensation of its sensory distribution, yet the patients’ headache is not improved.

If trigger points are found on exam, it is paramount that they be considered in the treatment of the patient. In addition to injection therapy for trigger points, treatment should include educating the patient on stretching and proper posture. Osteopathic manipulation should be considered to address the patient with musculoskeletal complaints and headaches or with trigger points felt to be contributing to their headaches.20

Studies have shown that one of the main problems in chronic headaches is central sensitization due to prolonged afferent signals from myofascial tissues.16,21,22,23 A model of headache pain suggests that trigger points located in muscles innervated by cervical roots 1-3 or by trigeminal nerves are responsible for potentially excessive afferent input into the trigeminal system which may lead to central sensitization.15

By reducing the sum total of noxious afferent stimuli coming from the myofascial system innervated by cervical nerves, TPI can be used to desensitize or at least help prevent further sensitization of pain receptors and the CNS.

In addition, the ONB functions to reduce noxious afferent input into the central nervous system. An inflamed or injured occipital nerve will bombard the spinal cord and CNS with afferent stimuli,
which can cause occipital neuralgia symptoms. Those same stimuli could also influence sensitization or pain referral patterns of the trigeminocervical complex, often associated with migraines.\textsuperscript{25}

The contraindications for the use of ONB are few, but include skull surgery compromising the occiput, Arnold Chiari Malformation, skull deformity and allergies to local anesthetic. The adverse effects of ONB have been minimal in the author’s clinical experience. Some patients have reported head soreness that resolves spontaneously 1 hour to 3 days after the procedure. The author no longer uses powdered steroids in the solution after a patient, prior to this study, developed prolonged occipital muscle ache that was thought to be due to precipitated steroid crystals. Adverse events recorded in this study consisted of two patients, both with a prior history of pre-syncpe, who developed vasovagal type dizziness that resolved within two minutes after lying supine.

If a patient is experiencing a headache at the time of the injection, the patient may describe a “head-rush” sensation in which they feel a coolness or warmth wash over their skull with some associated lightheadedness, which consistently resolves in a minute or two. As with the use of all local anesthetic injections, there is a risk of arrhythmias with intra-arterial compromise, but with appropriate draw-back technique this risk is minimized. One study noted a case of iatrogenic Cushing syndrome after the administration of 480mg triamcinolone via six bilateral GONB over a period of three months.\textsuperscript{26} Currently, the author performs these procedures no more frequently than every three months.

Limitations of this study include the inclusion of two types of headaches, a small number of patients, and the retrospective chart review of patient reported benefit is likely to cause some degree of recall bias.

Future areas of research should attempt to elucidate the best anesthetic solution and if it should contain steroids. To date there is no evidence that including steroids increases the ONB effectiveness.\textsuperscript{27} In addition, the inclusion of placebo controls in future studies would help further validate this therapy.

In this study, patients who received less than one week of benefit seemed to have more mixed headache types and were more likely to have been using more abortive medications prior to injection.

A prospective study at the authors’ clinic is planned that may better characterize the therapeutic response of these procedures, quantify the reduction in headache medication use, and determine if repeated injections changes the nature of the patients headaches.

CONCLUSIONS

In this retrospective chart review of seventy-one patients who were treated for migraines or occipital neuralgia and found to have occipital tenderness on exam, 91% of patients received benefit with a mean length of 9 weeks using occipital nerve blocks. The median benefit obtained was 8 weeks for both groups. Those who received trigger point injections, in addition to nerve blocks, had an average increased length of benefit of less than one week compared to ONB alone and it is not felt to be significant.

The response rate of 91% is higher than some other studies have reported for migraines and occipital neuralgia.\textsuperscript{19} It is likely higher in this study due to the inclusion requirement of occipital tenderness.

The patients in this study had all been referred to a neurologist for difficult to treat headaches and many suffered for years before finding any treatment that provided significant benefit without bothersome side effects.

Given how effective occipital nerve blocks appear to be for some headaches with occipital tenderness, further studies are warranted to confirm this retrospective chart review. Both ONB and TPI should be consistently included in the training of physicians who treat headaches. They are easy and safe to perform office procedures that can significantly reduce headache frequency and severity in the majority of patients who experience treatment refractory migraines and occipital neuralgia.

ACKNOWLEDGEMENTS

Frank Willard PhD, of The University of New England College of Osteopathic Medicine, for the use of anatomical dissection of the occipital nerves in Image 1.

CONFLICT OF INTEREST STATEMENT

The Authors declares that there are no conflict of interest.

IRB approval: Approval given by Central Maine Medical Center.

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Osteopathic Considerations in the Infections of the Respiratory Tract

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Respiratory Tract
Antibiotic Use
Disease Prevention & Wellness
Osteopathic Manipulative Medicine
Community Acquired Pneumonia

Respiratory tract infections are a common reason for office visits in primary care settings. Respiratory tract infections can often be managed in an outpatient setting, however hospitalization may be necessary in some more emergent and life threatening cases. A thorough history and physical will often help guide physicians on the proper course and setting for management. Furthermore, a thorough osteopathic assessment will guide the physician in diagnosing and treating somatic dysfunctions caused by respiratory infection. Osteopathic manipulative treatment can aid in recovery by providing relief of symptoms, and restoring proper structure and function of the respiratory system.

INTRODUCTION

Acute respiratory infections (ARI) are currently the most common reason for seeking ambulatory care.1 Additionally ARI’s are the leading cause of seeking medical treatment in returning travelers.2 Because the realm of ARI’s is so broad, it is important to be able to correctly differentiate between cases that can be adequately treated in an outpatient setting, and those that will require hospitalization. Accounting for such a high number of office visits, it is important for osteopathic family physicians to be knowledgeable and confident in their approach to a patient with an (ARI). Understanding the interplay between the various components of the respiratory system, and the effect somatic dysfunctions have on function is central to the proper management of a patient with an ARI.

STRUCTURAL & FUNCTIONAL CONSIDERATIONS OF THE RESPIRATORY TRACT

The respiratory system is composed of the oropharynx, conducting airways, lungs, muscles of respiration, and the chest wall.3 The distinction between upper and lower respiratory infections is an anatomical one. The nose, mouth, pharynx and larynx comprise the upper airway, which is also connected to the middle ear via the Eustachian tube.3 Infections in these areas are considered upper respiratory infections. Lower respiratory infections can potentially include infections that extend from the bronchus to the alveoli.

The upper respiratory tract humidifies inspired air, and offers protective measures against entering microorganisms.3,4 Inspiration brings exogenous microorganisms, dust, gases, and smoke into the lungs.3 Because of this, the respiratory tract has to have a system of filtration for removal of harmful inspired material. Cilia and mucous entrap entering microorganisms, while tonsils and adenoids provide immunologic defense against biologically active material.3 Smaller particles that escape to the trachea and bronchial airways get trapped in the mucus which is ultimately removed by mucociliary transport to the pharynx and mechanical expulsion via coughing and sneezing.5 In the lower respiratory tract, alveolar macrophages engulf and destroy inhaled microorganisms and particles.5 Somatic dysfunctions disrupting structural and functional relationships of the face and thoracic cage can therefore impede host defenses against infection.

EPIDEMIOLOGY

Infections of the upper and lower respiratory tract affect all individuals, but the probability of severe disease is observed in a bimodal distribution, as the young and the elderly are at greatest risk. In the United States, respiratory infections are currently the leading infectious cause of hospitalization and death among adults, and are the overall leading cause of hospitalization in children.6,7 Acute respiratory infections are also one of the leading causes of death in children under 5 years of age.8,9 Risk factors that result in more severe illness include being male, inhalation of pollutants, malnutrition, and extremes of age.8 Upper respiratory tract infections, which are summarized in Table 1 (page 18), contribute to disability and days lost from school or work.9 In 2016, just twelve
**TABLE 1:**
Upper Respiratory Infections

<table>
<thead>
<tr>
<th>Disease</th>
<th>Etiology</th>
<th>Common Symptoms</th>
<th>Common Physical Examination Findings</th>
<th>Considerations</th>
<th>Common Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pharyngitis&lt;sup&gt;32, 34, 35&lt;/sup&gt;</td>
<td>Viral &amp; Bacterial (GAS)</td>
<td>Fever (&gt;38°C), Sore Throat, Myalgia Headache</td>
<td>Cervical LAD, Pharyngeal Erythema, Exudates</td>
<td>Respiratory Distress, Poor Feeding, Resistant to antibiotic therapy</td>
<td>Antimicrobial therapy if high bacterial suspicion</td>
</tr>
<tr>
<td>Allergic Rhinitis&lt;sup&gt;36, 37&lt;/sup&gt;</td>
<td>Viral</td>
<td>&gt;2 sx: Sneezing, Nasal pruritus, Rhinorrhe, Congestion &gt; 1 hour for most days</td>
<td>Infamed Nasal turbinates, Associated with sinusitis, asthma, OM &amp; conjunctivitis</td>
<td>Rule out non-allergic causes including drug induced, &amp; inflammatory disorders, etc.</td>
<td>Nasal decongestants Intranasal steroids</td>
</tr>
<tr>
<td>Acute Sinusitis&lt;sup&gt;38, 39, 40&lt;/sup&gt;</td>
<td>Viral with possible secondary bacterial</td>
<td>Nasal obstruction &amp; nasal secretions &lt; 10 days</td>
<td>Sinus swelling Rhinorrhea</td>
<td>IN THE NEWBORN: Poor feeding &amp; focal signs of sinus involvement</td>
<td>IN NEWBORNS: Antibacterial therapy covers S. aureus, GAS &amp; GBS</td>
</tr>
<tr>
<td>Rhinosinusitis&lt;sup&gt;41, 42&lt;/sup&gt;</td>
<td>Viral with possible secondary bacterial</td>
<td>ACUTE: &gt; 3 times/year, with &gt; 2 sx: mucopurulent (not clear) drainage. Nasal obstruction, Facial Pain, &amp; Anosmia</td>
<td>ACUTE &amp; CHRONIC: Purulent nasal discharge, CHRONIC: With or without nasal polyps seen on rhinoscopic exam or sinus CT scan.</td>
<td>Associated with asthma, GERD, OM, immunodeficiencies, defects in mucociliary clearance (CF or PCD)</td>
<td>CRS: Antibiotics are controversial, with potential use of a 10-14 day course with or without oral steroids.</td>
</tr>
<tr>
<td>Epiglottitis&lt;sup&gt;43, 44, 45&lt;/sup&gt;</td>
<td>H. influenza, Streptococcus spp., Virall</td>
<td>Fever (&gt;38°C), sore throat, hoarseness, dyspnea, inspiratory stridor, with a “hot potato” or muffled voice</td>
<td>Unique posture of the head &amp; neck. Gross appearance of the pharynx may appear normal</td>
<td>Posture &amp; Stridor, Unstable vital signs &amp; distress, ADULTS: stridor not as frequently seen</td>
<td>BSA &amp; steroids; Emergency intervention when necessary</td>
</tr>
<tr>
<td>Laryngitis&lt;sup&gt;46, 47&lt;/sup&gt;</td>
<td>Irritants Viral</td>
<td>Hoarseness &amp; Aphony ~3 - 4 days duration</td>
<td>Benign examination</td>
<td>If URT, consider alternative diagnosis</td>
<td>Voice Rest</td>
</tr>
<tr>
<td>Croup&lt;sup&gt;48, 49, 50&lt;/sup&gt;</td>
<td>Viral (MC Parainfluenza) with possible secondary bacterial</td>
<td>PRODROME: URT sx 12 - 48 hours before “barking” cough with inspiratory stridor &amp; hoarseness</td>
<td>RADIOGRAPH: AP neck film with “steeple” or “hourglass” sign Westley Score</td>
<td>Rapid course, Drooling &amp; High fever may be present</td>
<td>Conservative Management; Emergent intervention when necessary</td>
</tr>
<tr>
<td>Otitis Media</td>
<td>S. pneumonia, H. influenzae, M. catarrhalis</td>
<td>&lt; 3 years old are most susceptible: Fever, otalgia &amp; impaired hearing</td>
<td>Fluid accumulation in the middle ear &amp; erythema of the TM</td>
<td>Unvaccinated children Signs of pharyngeal irritation Recurrent &amp; persistent episodes</td>
<td>Antimicrobial Therapy</td>
</tr>
</tbody>
</table>

Table 2: Lower Respiratory Infections

<table>
<thead>
<tr>
<th>Disease</th>
<th>Etiology</th>
<th>Common Symptoms</th>
<th>Common Physical Examination Findings</th>
<th>Considerations</th>
<th>Common Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Bronchitis²¹,²²</td>
<td>Viral (Influenza &amp; RSV) Bacteria (Streptococcus spp, Atypical Bacteria)</td>
<td>Cough +/- sputum 1-3 weeks</td>
<td>Upper &amp; Lower Respiratory signs without crackles</td>
<td>Hospitalizations Comorbidities Vomiting &amp; &gt; 4 weeks duration: Consider B.pertussis</td>
<td>Cough suppressants, nasal decongestants, expectorants, beta agonists, antihistamines, &amp; Abx therapy.</td>
</tr>
<tr>
<td>Bronchiolitis⁵³,⁵⁴,⁵⁵,⁵⁶,⁵⁷,⁵⁸,⁵⁹,⁶⁰,⁶¹</td>
<td>Viral (MC is RSV)</td>
<td>&lt; 2 yrs old, MC within 1st year Wheezing, Fever, Cough, Rhinorrhea</td>
<td>Decreased lung sounds with crackles Dyspnea Chest retractions</td>
<td>Prematurity, Lower cord blood antibody titers to RSV, lower SES, smoke exposure.</td>
<td>Conservative management Consider Abx if bacterial superinfection suspected</td>
</tr>
<tr>
<td>Pneumonia⁶,⁷,¹²,⁶²,⁶³,⁶⁴,⁶⁵,⁶⁶,⁶⁷,⁶⁸,⁶⁹,⁷⁰,⁷¹,⁷²,⁷³</td>
<td>BACTERIA: S. pneumonia, S. aureus, H.Influenzae VIRAL (CHILDREN): RSV, Parainfluenza, Influenza VIRAL (ADULTS): Influenza &amp; RSV</td>
<td>Fever &amp; Chills, Pleuritic chest pain, Productive cough with purulent sputum</td>
<td>Leukopenia Tachypnea Tachycardia Crackles Signs of consolidation Sputum: thick &amp; purulent, possibly rust colored</td>
<td>Older age; Unvaccinated; Comorbidities</td>
<td>Beta-lactam plus a macrolide or fluoroquinolone therapy</td>
</tr>
</tbody>
</table>

**ABBREVIATIONS:** RSV: Respiratory Syncytial Virus, SES: Socioeconomic Status, Abx: antibiotics, MC: most common

weeks into the year, influenza-like illness had already accounted for 2.9% of visits reported through the U.S Outpatient Influenza-like Illness Surveillance Network.¹⁰

In adults, community-acquired lower respiratory tract infections are an important cause of acute illness.¹¹ Lower respiratory tract infections, which include bronchitis, bronchiolitis, and pneumonia, are summarized in Table 2.⁴ Pneumonia is an important contributor to mortality worldwide, and together with influenza, constitutes one of the leading causes of death in the United States.¹² In children, the most common lower respiratory infections are pneumonia and bronchitis; however, in children less than two years of age, bronchiolitis predominates.⁵

**ASSESSMENT & MANAGEMENT OF ACUTE RESPIRATORY DISEASE**

The key to proper diagnosis and treatment of respiratory disease depends on a thorough history and physical examination. Key diagnostic history and physical exam findings are presented in Tables 1 and 2. Several important considerations can be used to differentiate between patients who can be managed conservatively, and those who need emergent care. For example, in cases of upper respiratory infections that present with respiratory compromise, rapid disease progression, and symptoms of dyspnea, tachypnea, tachycardia, stridor, and drooling, hospitalization must be considered. Epiglottitis has the greatest potential of the upper respiratory infections to yield the need for airway intervention.

Proper assessment of whether a patient with community acquired pneumonia (CAP) requires hospitalization or can be managed in an outpatient setting, can be done using the Pneumonia Severity Index, which assesses severity of illness and associated mortality risk within 30 days, and the CURB-65 scores.¹³ Some red flags that may warrant further investigation into whether a patient should be hospitalized or treated in an outpatient setting for CAP include altered mental status, temperature ≤35°C or ≥40°C, coexisting illnesses, respiratory rate of 30 breaths per minute or greater, systolic blood pressure < 90 mmHg or diastolic blood pressure <60 mmHg, and patient age.¹³ Determining whether a patient will be managed in the hospital or outpatient setting for CAP will also determine the proper antibiotic regimen to be used.¹³
RESPIRATORY INFECTIONS & PROPER ANTIBIOTIC USE

Judicious antibiotic use should be a consideration when assessing treatment options for respiratory illness. Physicians often prescribe antibiotics during most visits for ARI’s, even when most upper respiratory tract infections are viral in nature.\textsuperscript{14,15} Fifty percent of all antibiotics prescribed for adults and 75% of all antibiotics prescribed for children are for the treatment of respiratory infections.\textsuperscript{1} Antibiotic overuse may lead to resistance, increased costs, and increased adverse effects; thus, it is important to differentiate between bacterial and viral etiologies.\textsuperscript{15} For example rhinosinusitis, which is commonly seen in outpatient settings, can lead to over-prescription of antibiotics if care is not taken to differentiate between bacterial and viral causes.\textsuperscript{15} Bacterial rhinosinusitis should not be suspected until symptoms have lasted for 10 days or greater with worsening symptoms after initial improvement. Furthermore, purulent nasal discharge, maxillary tooth or facial pain, unilateral maxillary sinus tenderness, and initial improvement followed by worsening symptoms often indicate a bacterial etiology. Even cases of rhinosinusitis caused by bacterial etiology can be managed with watchful waiting if they are mild, and if proper follow up can be ensured.\textsuperscript{15} In lower respiratory infections like CAP, the decision to treat with empiric antibiotic therapy should be based on the most likely pathogen involved, risk factors for antimicrobial resistance, clinical trials proving efficacy, and medical comorbidities that can influence the likelihood of a specific pathogen. Because antibiotics are not always indicated, OMT may fill a possible gap in treatment options in patients seeking treatment, and possibly in children.

INTEGRATION OF OSTEOPATHIC ASSESSMENT

Respiratory infections often manifest with cranial, cervical, and upper thoracic dysfunctions.\textsuperscript{4} These somatic dysfunctions contribute to many of the symptoms that accompany upper respiratory infections and necessitate a thorough osteopathic structural exam in order to complete a comprehensive patient assessment.\textsuperscript{4,6} Furthermore, by assessing and treating associated somatic dysfunctions, recovery can be achieved more efficiently.

INTRODUCTION TO THE MODELS OF OSTEOPATHY

When addressing a patient with a respiratory illness, one should consider the models of osteopathy and what treatment approach specifically addresses each model. The five models are the Biomechanical model, the Respiratory-Circulatory model, the Metabolic-Energy model, the Neurological model, and the Behavioral model.\textsuperscript{16} As described below, these models represent a conceptual thought process in which a physician may utilize OMT.

Furthermore, as these modalities are applied on an individual patient basis, the osteopathic treatment plan should vary accordingly. For example, the quantity of OMT sessions needed to treat various illnesses is dependent on both the patient and the course of the disease. Acute conditions often require fewer treatment sessions, while chronic conditions require more OMT sessions.\textsuperscript{17} Table 3 (pages 21 and 23) summarizes osteopathic manipulative treatments by region that can be useful in the treatment of a patient with an ARI.

BIOMECHANICAL CONSIDERATIONS

When performing an osteopathic structural exam, it is important to give special attention to the cervical, thoracic, and lumbar spines, clavicles, ribcage, thoracic inlet, and diaphragm. Respiratory infections are often coupled with coughing or labored breathing, resulting in the recruitment of accessory muscles of inspiration including the sternocleidomastoid, scalenes, levator scapulae, pectoralis minor, and upper trapezius.\textsuperscript{18} Such increased respiratory effort overwhels the capacity of the thoracic diaphragm causing somatic dysfunctions. Treating the first rib helps to relax the anterior scalene, enhancing the respiratory motion of the upper thoracic rib cage. Improving clavicle motion through techniques such as balanced ligamentous tension and muscle energy may help restore optimal respiratory motion, since it serves as an insertion point for many muscles involved in respiratory activity. In addition, optimizing movement of the diaphragm to return it to a non-hypertonic, freely mobile state is appropriate for a patient in respiratory discomfort. Treatment of the origins and insertions of the diaphragm may be considered. The diaphragm crura insert on the lumbar spine at the level of L1 to L3 and, if they are hypertonic, can be treated at the associated vertebral levels to help relax and encourage normal thoracic diaphragm motion.\textsuperscript{19} The upper segment of the thoracic rib cage, specifically ribs 1-4, should be treated to encourage proper range of motion to enable proper respiratory mechanics. The intercostal muscles can spasm and fatigue with labored breathing. Treatment with OMT may help to decrease spasms and improve rib cage mobility.

For patients presenting with complaints localized to the head and neck, such as sinusitis and otitis media, special attention should be paid to the cranium and cervical spine. Somatic dysfunctions of the head should be assessed and treated with cranial osteopathic manipulative medicine(COMM). Anatomically, the upper respiratory tract includes structures in areas enclosed by the sphenoid, basiocciput, temporal, and frontal bones.\textsuperscript{4} Therefore, dysfunctions of the cranial base and facial bones can affect the upper respiratory tract.\textsuperscript{4} More specifically, dysfunctions affecting the vagus nerve can affect parasympathetic tone and influence pharyngeal motor activity.\textsuperscript{4} Retro-orbital and retro-auricular pain may be produced by anterior atlas dysfunctions in patients with sinusitis or congestive symptoms.\textsuperscript{4} In these patients, consider frontal and maxillary lifts, and a nasion spread in order to facilitate the movement of facial bones. This will facilitate removal of secretions from the maxillary, frontal, and ethmoid sinuses. The Galbreath technique can be used to help with auricular pain secondary to middle ear congestion by mechanically decompressing the auditory canal.\textsuperscript{4}
### TABLE 3:
Osteopathic Manipulative Techniques (OMT) to address respiratory disease organized by body region

<table>
<thead>
<tr>
<th>Techniques</th>
<th>Potential Treatment Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Head</strong></td>
<td></td>
</tr>
<tr>
<td>Balanced membranous tension</td>
<td>• Treats cranial strain patterns</td>
</tr>
<tr>
<td></td>
<td>• Decreases dural strain complications</td>
</tr>
<tr>
<td>Sinus drainage technique</td>
<td>• Facilitates movement of facial bones</td>
</tr>
<tr>
<td>Cranial bone lifts &amp; effleurage</td>
<td>• Improves sinus drainage</td>
</tr>
<tr>
<td></td>
<td>• Decreases facial pain</td>
</tr>
<tr>
<td>Galbreath technique (Mandibular lift)</td>
<td>• Improves Eustachian tube drainage</td>
</tr>
<tr>
<td></td>
<td>• Decompresses the auditory canal</td>
</tr>
<tr>
<td></td>
<td>• Decreases auricular pain</td>
</tr>
<tr>
<td>Sphenopalatine ganglia inhibition</td>
<td>• Normalizes parasympathetic tone to nasal mucosa and sinuses</td>
</tr>
<tr>
<td></td>
<td>• Regulates blood flow to nasal conchae and encourages thinner mucosal secretions</td>
</tr>
<tr>
<td></td>
<td>• Decreases headache and facial discomfort</td>
</tr>
<tr>
<td>Venous sinus drainage</td>
<td>• Improves venous and lymphatic flow of the brain</td>
</tr>
<tr>
<td></td>
<td>• Decreases dural strain</td>
</tr>
<tr>
<td></td>
<td>• Decreases headache</td>
</tr>
<tr>
<td>Occipito-atlantal decompression</td>
<td>• Decreases muscle spasms and restores upper cervical mobility</td>
</tr>
<tr>
<td>Suboccipital release</td>
<td>• Frees the passage of the vagus nerve, normalizing parasympathetic tone</td>
</tr>
<tr>
<td><strong>Neck &amp; Cervical Spine</strong></td>
<td></td>
</tr>
<tr>
<td>Soft tissue &amp; myofascial techniques addressing secondary muscles of inspiration</td>
<td>• Relaxes the Sternocleidomastoid and scalene muscles to aid in the drainage of the superficial and deep cervical lymph nodes</td>
</tr>
<tr>
<td></td>
<td>• Allows improved respiration by relaxing the attachments to the manubrium and clavicle</td>
</tr>
<tr>
<td>Direct techniques (MET, articulatory, HVLA)</td>
<td>• Improves somatic dysfunctions in the cervical spine allowing increased range of motion of the neck</td>
</tr>
<tr>
<td>to cervical spine dysfunctions</td>
<td>• Regulates neural influence over the trigeminal nucleus&lt;sup&gt;4&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>• Treatment of C3-C5 can affect diaphragmatic innervation</td>
</tr>
<tr>
<td></td>
<td>• Removes mediastinal fascial restrictions freeing the pathway of the vagus nerves</td>
</tr>
<tr>
<td>Indirect techniques (CS,FPR,BLT) to cervical spine dysfunctions</td>
<td>• CS alleviates acute tenderpoints</td>
</tr>
<tr>
<td></td>
<td>• Treatment of upper cervical region balances autonomies</td>
</tr>
<tr>
<td></td>
<td>• Indirect techniques can have the same treatment effects as direct techniques and may be an alternative to direct techniques or used when direct techniques are not tolerated by the patient.</td>
</tr>
<tr>
<td><strong>Lumbar</strong></td>
<td></td>
</tr>
<tr>
<td>Direct/indirect treatment of the lumbar spine (FPR or BLT of L2-L3)</td>
<td>• Addresses restrictions at diaphragmatic attachments on the lumbar spine at L1-L3&lt;sup&gt;19&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>• Encourages respiratory diaphragm motion</td>
</tr>
<tr>
<td></td>
<td>• Improves lymphatic drainage (cisterna chyli lies anterior to the lumbar spine)</td>
</tr>
<tr>
<td>Direct/indirect treatment of the psoas and QL</td>
<td>• Addresses myofascial restrictions at the attachments of the diaphragm</td>
</tr>
<tr>
<td></td>
<td>• Psoas restrictions affect the upper lumbar spine and cross into the pelvis and lower extremities</td>
</tr>
<tr>
<td><strong>Pelvis &amp; Sacrum</strong></td>
<td></td>
</tr>
<tr>
<td>Direct/indirect treatments of the sacrum and pelvis (sacral rock, pelvic SD treatment with MET)</td>
<td>• Improves biomechanical restrictions of the sacrum to allow for proper motion of the spine with respiration</td>
</tr>
<tr>
<td></td>
<td>• Improves motion of the pelvic diaphragm, allowing for the descent of the abdominal diaphragm with inhalation</td>
</tr>
<tr>
<td></td>
<td>• Balances autonomic innervation affecting overall autonomic tone</td>
</tr>
<tr>
<td>Direct/indirect treatment of the hip and lower extremity SD</td>
<td>• Improves head, neck, and thoracic cage mobility due to the musculoskeletal attachments of 17 muscles from the scapula to the regions mentioned</td>
</tr>
<tr>
<td></td>
<td>• Improves upper extremity restrictions that can decrease thoracic lymph drainage</td>
</tr>
<tr>
<td>Direct/indirect treatment of the hip and lower extremity SD</td>
<td>• Improves diaphragmatic motion through the psoas and additional musculoskeletal attachments from the LE into the pelvis and sacrum</td>
</tr>
</tbody>
</table>

**ABBREVIATIONS:** CS: Counterstrain, FPR: Facilitated Positional Release, MET: Muscle Energy Technique, SD: Somatic Dysfunction, QL: Quadratus lumborum, BLT: Balanced Ligamentous Tension, HVLA: High Velocity Low Amplitude
breathing. Specifically, somatic dysfunctions in the ribs, clavicles, and upper thoracic spine contribute to decreased lymphatic drainage through the thoracic inlet. Proper thoracic cage compliance is vital to proper lymphatic drainage. Rib, thoracic inlet, thoracic spine, and thoraco-abdominal somatic dysfunctions prevent full excursion during respiration and therefore negatively impact the change in intrathoracic pressure which in turn, can decrease lymph drainage. Additionally, the thoracic diaphragm serves as a lymphatic pump, as well as an aid for circulation of blood return to the heart. These dysfunctions may be the cause or the effect of poor diaphragmatic movement, which proves vital in the normal changes in intrathoracic pressure. Venous and lymphatic drainage from the head and neck are also disrupted by these dysfunctions, leading to decreased clearance of microorganisms.

Optimizing thoracic movement and respiratory effort has many implications for improving the diseased state. Treatment of the ill patient suffering from respiratory infection favors addressing thoracic or upper lumbar dysfunctions to assist diaphragm attachments, to optimize the diaphragm’s ability to act as a pump for lymphatic fluid and circulation. Restricted fascia in the neck and upper thorax should be treated to help aid in improved circulation to and from areas of infection. Opening of the thoracic inlet with myofascial techniques facilitates lymph drainage via the thoracic and the right lymphatic ducts. Once the thoracic inlet is opened and relaxed, lymphatic fluid can freely return to the venous circulation subsequently decongesting the body. Draining the cervical and neck lymph chains toward the inlet and lymphatic pumps are particularly useful during acute illness to mobilize lymphocytes and to facilitate lymphatic return and decongestion. Efficacy of medical therapy may be decreased in the presence of tissue congestion, as this impedes the ability of leukocytes and medications to reach their target tissue. Improving circulation therefore enhances drug delivery, should antibiotics be required, and also facilitates the immune system functionality by increasing the ability of white blood cells to reach infected areas.

**NEUROLOGICAL CONSIDERATIONS**

Viscerosomatic reflexes manifested as tissue texture changes and tenderness on palpation in the upper cervical paravertebral soft tissues can support the physician’s diagnosis of an upper respiratory tract infection, as well as indicate areas that need treatment. Viscerosomatic reflexes often seen in respiratory illness can be found generally in the upper thoracic region for sympathetic reflexes and in the upper cervical region for parasympathetic reflexes. Furthermore, somatovisceral reflexes like Chapman’s points can affect the autonomic nervous system, upsetting the balance that exists between the sympathetic and parasympathetic nervous system. OMT can normalize this balance, reducing the duration and intensity of symptoms and enhancing the efficacy of other therapies. Chapman’s reflex points which are listed in Table 4 were first defined by Frank Chapman, D.O as gangliform contractions which are tissue texture abnormalities that correspond to visceral dysfunction, and can be treated by rotatory motion.

Somatic dysfunctions in the upper thoracic spine causing facilitation of those spinal segments can increase the activity of the sympathetic nervous system. An increase in sympathetic tone leads to an increase in airway epithelial hyperplasia which leads to increased goblet cells and increased luminal secretions. In order to normalize sympathetic tone in patients with respiratory infections, consider treating the sympathetic chain between T1-T7. Rib raising is a technique that targets the rib angles in order to inhibit and normalize the sympathetic ganglia that lie paravertebrally. Rib raising can be used with great success, even in patients who are severely ill.

An increase in parasympathetic tone leads to relative bronchial constriction. Head complaints involving nasal sinuses and laceration can be treated with COMM as is seen in Table 3 (page 22). In order to normalize parasympathetic tone, target C2, C3, and the mediastinal fascia to free the pathway of the vagus nerves as they pass to the thorax. Finally, the upper respiratory tract delivers somatosensory input to the central nervous system through the trigeminal nerve. Thus, muscles innervated by the trigeminal nerve may be subject to viscerosomatic reflexes. Furthermore, because parasympathetic innervation to the upper respiratory tract from the facial nerve reaches its final destination through nerves in the distribution of the trigeminal nerve, treating these areas can correct the effects of parasympathetic hyperactivity. Trigeminal nerve stimulation using OMT directed to the cervical and thoracic regions can reduce nasal congestion and increase secretions since it carries sympathetic and parasympathetic postganglionic fibers to the upper respiratory tract.

**BEHAVIORAL MODEL CONSIDERATIONS**

Quality of life and psychological health are often altered in patients with respiratory infections, thus, the osteopathic family physician is highly encouraged to consider the patient’s psychological and behavioral well-being. Infants and children experience prolonged symptoms of the common cold compared to adults. The degree of socioeconomic impact is therefore quite large. It is reported that a significant amount of time is missed from school by children, and from work by parents caring for sick children. The severity and chronicity of respiratory infections correlates with impairments in well-being, quality of life, and sleep. As upper respiratory infections and chronic diseases such as asthma and Chronic Obstructive Pulmonary Disease (COPD) are correlated, these comorbid conditions correspond with an increase in anxiety and depression. Although direct evidence is lacking on the effect OMT has on the quality of life of patients with respiratory infections, OMT has been shown to improve several parameters of pulmonary function and exercise capacity.

**EVIDENCE FOR USING OSTEOPATHIC MANIPULATIVE TREATMENT IN RESPIRATORY INFECTIONS**

There have been several studies showing the efficacy of OMT in treating respiratory illness. OMT has been shown to accelerate the recovery of preoperative values of forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) of postoperative patients with atelectasis. OMT has also been shown to aid in the recovery from pneumonia by enhancing the functioning of the immune system, and maximizing the effects of antibiotics. In addition, OMT has been associated with decreased hospital-stay duration, decreased use of intravenous antibiotics, and decreased incidence of respiratory failure or death in elderly patients hospitalized with pneumonia.
TABLE 3 (CON’T):
Osteopathic Manipulative Techniques (OMT) to address respiratory disease organized by body region

<table>
<thead>
<tr>
<th>Techniques</th>
<th>Potential Treatment Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thorax</td>
<td></td>
</tr>
</tbody>
</table>
| Techniques to address first rib SD (Still’s, FPR, MET, articulatory) | • Enhances respiratory motion of upper thoracic rib cage  
- Relaxes anterior and middle scalene accessory muscles of respiration  
- Removes restrictions at thoracic inlet |
| Thoracic inlet release | • Removes myofascial restrictions in the region of terminal lymphatic drainage<sup>19</sup>  
- Improves upper rib motion |
| Lymphatic pumps | • Augments lymphatic drainage of lungs<sup>19</sup>  
- Increases rib mobility (side thoracic pump) |
| Respiratory diaphragm doming / release | • Optimizes thoracic movement and respiratory effort  
- Restores proper diaphragmatic tone and structure  
- Facilitates lymphatic pump action of the diaphragm  
- Aids in return of circulation to the heart |
| Direct/indirect treatments of the thoracic spine and rib cage | • Improves somatic dysfunctions allowing increased thoracic cage excursion and improves range of motion of spine and ribs  
- Balances sympathetic innervation to the head, neck, and lungs  
- Improves lymphatic drainage by allowing for improved pressure gradient changes with respiration |
| Rib raising | • Targets the rib heads between T1-T4, where the sympathetic chain lies in order to inhibit and normalize the paravertebral sympathetic ganglia |
| Direct/indirect treatments of the intercostal muscles and ribs (CS and MET) | • Optimizes thoracic cage movement by relaxing intercostal muscles  
- Encourages lymph flow  
- Improves diaphragmatic motion by addressing diaphragm attachments (anterior costal margin to ribs 11 & 12 posteriorly) |

**ABBREVIATIONS:**  

TABLE 4:
Pertinent Chapman’s Reflex Points for Respiratory Infections<sup>4,74</sup>

<table>
<thead>
<tr>
<th>Structured Affected</th>
<th>Anterior Points</th>
<th>Posterior Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neck</td>
<td>Neck of the humerus</td>
<td>C3-C7 transverse processes</td>
</tr>
<tr>
<td>Tongue</td>
<td>Anterior surface of the 2&lt;sup&gt;nd&lt;/sup&gt; costal cartilage at the junction of the sternum</td>
<td>C1 transverse processes</td>
</tr>
<tr>
<td>Tonsils</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; Intercostal space, close to the sternum</td>
<td>C1 transverse processes</td>
</tr>
<tr>
<td>Nasal Sinuses</td>
<td>1&lt;sup&gt;st&lt;/sup&gt; rib medial to the junction with clavicle</td>
<td>C2 transverse process</td>
</tr>
<tr>
<td>Pharynx</td>
<td>Surface of the 1&lt;sup&gt;st&lt;/sup&gt; rib near the sternal notch</td>
<td></td>
</tr>
<tr>
<td>Sinuses</td>
<td>7-9 cm lateral to the sternum on the upper edge of the second rib</td>
<td>C2, midway between the spinous process and transverse process</td>
</tr>
<tr>
<td>Larynx</td>
<td>Anterior surface of 2&lt;sup&gt;nd&lt;/sup&gt; rib, at the costochondral junction</td>
<td></td>
</tr>
<tr>
<td>Bronchi</td>
<td>2&lt;sup&gt;nd&lt;/sup&gt; intercostal space</td>
<td>T2 midway between the spinous process and transverse process</td>
</tr>
<tr>
<td>Upper Lung</td>
<td>3&lt;sup&gt;rd&lt;/sup&gt; intercostal space</td>
<td>T3 midway between the spinous process and transverse process</td>
</tr>
<tr>
<td>Lower Lung</td>
<td>4&lt;sup&gt;th&lt;/sup&gt; intercostal space</td>
<td>T4 midway between the spinous process and transverse process</td>
</tr>
</tbody>
</table>
CONCLUSION

Acute respiratory illnesses commonly present to the osteopathic family physician. A thorough evaluation and treatment integrating osteopathic manipulative medicine can be effective in the care of patients with respiratory infections. Considering the models of osteopathic care and utilizing an osteopathic approach targeting each of the 5 models can provide comprehensive treatment to a patient with a respiratory infection. Doing so can aid in the return of proper respiratory functioning, helping to restore patients back to optimal health.

REFERENCES:


Knee Pain in Adults with an Osteopathic Component

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Keywords: Knee Pain, Osteoarthritis, Osteopathic Manipulative Medicine (OMM), Primary Care, Ottawa Knee Rules

INTRODUCTION & EPIDEMIOLOGY

Knee pain is among the most commonly cited reasons for outpatient doctor visits, accounting for over 1.9 million visits annually.1 The aging population of the United States and the obesity epidemic have contributed to a nearly twofold increase in the incidence of symptomatic knee conditions over the past decade. Today, over one-half of adults in the U.S. can expect to experience clinically significant knee pain within their lifetime and over 25% are currently affected.2,3 While age, overuse, and trauma are the most common etiologies, rheumatologic, infectious, vascular, and referred causes also contribute to the clinical picture.1 Osteoarthritis of the knee results in more than $28 billion dollars in annual health care costs and is among the top 5 leading causes of disability in the United States.4,5

The incidence of knee pain is increasing due to the rising prevalence of obesity, sedentary lifestyles, and aging baby boomer population in the United States. Both acute and chronic knee conditions can result in the increased utilization of pain medications and a decreased quality of life. A multimodal approach to knee pain management can thus greatly benefit the patient population and decrease the burden of knee conditions on the healthcare system. This article presents the epidemiology, clinically relevant anatomy, physiology and major risk factors associated with common knee pain conditions. An overview of etiologies is presented in terms of major clinical presentation, diagnostic testing, and treatments. Practical guidelines for an osteopathic approach to the examination and diagnosis of knee pain are then discussed, with a focus on the osteopathic structural exam and the use of special tests to discern and localize soft tissue injury. A novel diagnostic algorithm summarizing a step-by-step approach to a patient with knee pain is also presented. This method integrates the physical exam, special tests, lab work, and imaging to formulate an evidence-based protocol for formulating a knee pain diagnosis. Finally, the article presents management strategies for common causes of knee pain including conservative, pharmacologic, manipulative, and alternative/complementary treatments. Evidence-based recommendations for manipulation efficacy are reviewed from meta-analysis data, randomized controlled trials, and a case report. The article also provides a description of commonly used manipulation techniques and their indications with respect to the anatomic location of knee pain and its underlying etiology.

BIOMECHANICS

In order to discern the etiology of knee pain and injury, it is important to understand the normal anatomy of the knee. The knee joint is a complicated articulation and the largest joint of the body with a normal range of motion (ROM) of 0 degrees extension, 140 degrees flexion, and 8 to 12 degrees rotation.6,7 The knee joint is enclosed within a synovial capsule and functions as a complex hinge joint with three articulations: the medial and lateral femorotibial articulations, and the patellofemoral articulation.6,8 The knee also has 6 degrees of motion which contribute to its instability and should be considered during evaluation: (1) flexion/extension, (2) internal/external rotation, (3) varus/valgus, (4) anterior/posterior translation, (5) medial/lateral translation, and (6) compression/distraction.6,8 There are variants of these motions that can be considered normal or abnormal depending on the patient. Genu valgus for example is a posture where the feet are spread apart but the knees are close together. This is more commonly found as normal in women, but can be abnormal based on the joint pathology causing this. Genu varus is the opposite and is when the feet are close together but the knees are far apart. This is rarely normal and is...
sometimes correlated with rickets. It is also important to understand that knee joint stability is reliant on foot biomechanics, which can absorb mechanical stress from ground contact and can impact postural alignment at the knee joint. Therefore, patients with flat feet (pes planus) or who have a high arch (pes cavus), are more likely to get knee pain and medial tibiofemoral cartilage damage. Knee joint stability is conferred mainly by the soft tissues of the capsule: ligaments, tendons, and menisci. The ligaments confer static stability to the knee joint, while the muscles and tendons provide dynamic stability during motion. Furthermore, the knee can be divided into four compartments: anterior, posterior, medial, and lateral. This classification has both anatomical and clinical implications.

### RELEVANT ANATOMY

The medial aspect of the knee is the most commonly injured compartment in knee pain. It contains the medial collateral ligament (MCL), which is the most commonly injured ligament in the knee, the medial meniscus, and the medial patellofemoral ligament (MPFL) ligament. The muscles of the compartment are the semimembranosus, sartorius, gracilis, and semitendinosus. The latter three form a conjoined insertion onto the anteromedial tibia, which is commonly implicated clinically in pes anserinus tendonitis and bursitis. The MCL is the primary resistor to valgus strain, and is commonly injured by lateral blows to the knee. The MPFL is the primary stabilizer of lateral patellar motion and is often involved in patellar dislocations, which are more common in females due to an increased Q-angle. The Q-angle is a measurement of the angle between the quadriceps muscle and the patella tendon. A high Q-angle on physical exam means that the patella has abnormal movement over the front of the knee joint, which overtime can lead trauma to the posterior cartilage of the patella. Finally, the medial compartment contains three bursae: the medial gastrocnemius bursa, the anserine bursa, and the semimembranosus bursa. If injured, the bursae can swell and produce localized tenderness on physical exam.

The anterior aspect of the knee is the second most common region involved in knee pain. The anterior compartment contains the patellofemoral articulation, composed of the quadriceps tendon, the patella, the patellar tendon, and additional patella-stabilizing ligaments. These are individually involved in conditions like tendinitis, Osgood Schlatter, and Sinding-Larsen-Johansson syndrome. Tendinitis is the inflammation of a tendon and can be either patellar or quadriceps in this case. Osgood Schlatter and Sinding-Larsen-Johansson are both conditions that affect teens during growth, but involve inflammation of different attachments points of the patella tendon. All of these ligaments and tendons are collectively involved in patellofemoral syndrome. In addition, this compartment contains the anterior cruciate ligament (ACL), the intermeniscal ligament, and the bursae. The ACL is the main stabilizer to anterior translation of the tibia. It’s commonly associated with non-contact pivoting injuries. This is often seen with athletes who compete in sports like soccer that involve sudden deceleration, landing and pivoting maneuvers. The anterior compartment also contains five bursae: pretibial, suprapatellar, subcutaneous, deep infrapatellar, and prepatellar. The prepatellar bursa is the most common bursa involved in injury of the knee.

The posterior compartment is comprised of the posterior cruciate ligament (PCL), meniscofemoral ligament, and the oblique popliteal ligament. In terms of muscles it is made up of the popliteus, gastrocnemius, and plantaris muscles. The PCL is the primary resistor to posterior translation of the tibia and is among the least injured ligaments of the knee. Most posterior compartment pain is not associated with direct structural injuries, but with effusions present within the knee. An effusion in the back of the knee is often aggravated by flexion and can result in the posterior displacement of fluid and the formation of a baker’s cyst. Posterior or popliteal pain can also result from extra articular causes such as deep vein thrombosis (DVT) and popliteal artery aneurysms.

The lateral compartment of the knee is less commonly implicated in knee pain and contains the lateral collateral ligament (LCL), lateral meniscus, popliteofibular ligament (PFL), and arcuate ligament. The muscles of the lateral compartment include the iliotibial band (ITB) and biceps femoris. Pain along the lateral joint line is most often associated with lateral meniscal or LCL injuries, while pain localized over the lateral femoral condyle is characteristic of ITB syndrome. The lateral compartment also contains three bursae: the lateral gastrocnemius bursa, fibular bursa, and fibulopopliteal bursa.

### RISK FACTORS

The risk factors for knee pain vary by etiology, but can generally be divided into modifiable and non-modifiable. Major modifiable risk factors are excess body mass, joint injury (trauma, sports, intense exercise), muscle weakness, structural malalignment, and occupation. Non-modifiable risk factors include gender, age, race, and genetic predisposition. Addressing modifiable risk factors via weight loss, bracing, strengthening exercises, and activity modification is often the initial treatment goal in non-traumatic knee pain presentations.

It is important to understand the most common presentations and etiologies of knee pain in the primary care setting in order to successfully arrive at a diagnosis using the minimum amount of resources. Table 9 lists the clinical presentations and treatment strategies for the majority of knee pain etiologies encountered by the primary care practitioner.

### OSTEOPATHIC STRUCTURAL EXAM/CLINICAL APPROACH

The osteopathic approach to treating a patient with knee pain incorporates osteopathic manipulative treatment (OMT) into a comprehensive treatment plan that may include medication, rehabilitative exercises, nutrition, surgical procedures, and lifestyle counseling. Through proper education on health promotion and disease prevention, osteopathic medicine emphasizes the overall wellness of its patients. The added benefit of hands on manipulation allows osteopathic physicians to address the shift in homeostasis that can occur in any pathology. This allows them to accelerate the healing process through natural means and develop a more therapeutic relationship with their patients.

Knee pain is a common reason for both outpatient and emergency room visits depending on its severity. Since there is a wide differential for knee pain, osteopathic physicians use a combination of a detailed history and osteopathic structural exam to ascertain potential causes and treatments to alleviate pain. When taking a history of a patient with knee pain, it’s important to focus on its origin, duration, and possible connection to trauma or other high-
Conservative management should be initiated in the majority of cases of knee pain presenting in the primary care setting. The level of clinical suspicion for a fracture can be assessed using the Ottawa Knee Rules and confirmed with plain film x-rays. If a patient meets at least one criterion and is positive for a fracture on X-ray, they should be referred to an orthopedic or sports medicine specialist. However, if the x-ray is negative or a patient does not meet the criteria, special tests should be performed to rule out ligamentous and meniscal injury. This is where a thorough physical exam is the most important, as it determines if a physician should refer their patient for an MRI or follow up with conservative treatment. The most common cause of acute knee pain, which should be considered if imaging and special tests are negative, is a sprain or strain. For older patients with chronic knee pain, a physician should consider osteoarthritis high on the differential.

PHARMACOLOGIC MANAGEMENT

Appropriate pharmacologic management is critical for acute ligamentous injuries and chronic degenerative conditions such as osteoarthritis. For short-term pain relief in patients with acute knee injuries, non-selective, non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen or naproxen (first-line) and tramadol (second-line) may be used. Long-term pain management for osteoarthritis may begin with acetaminophen and progress to selective NSAIDs, such as celecoxib, as the disease advances. Topical NSAIDs are advantageous for chronic use due to higher selectivity and less GI side effects when compared to oral NSAID regimens. Topical Capsaicin, a naturally derived compound from chili peppers relieves pain by reduced sensitivity and analgesia. Other natural remedies that can be used for knee pain include turmeric, ginger tea, and epsom salt soak. Corticosteroid injections provide effective temporary relief in moderate to severe degenerative disease and are most effective when local inflammation is present as indicated by erythema or synovial effusion. Opiates may be used in chronic pain refractory to all other types of therapy. Narcotic medications should always be used at the minimum effective dose, in conjunction with acetaminophen or NSAIDs. Transdermal patches may be preferable for patients who take numerous medication or have esophageal irritation. It is important to be aware of the side effects and drug interactions of opioid medications. The most serious side effect is respiratory depression, especially pronounced if opioid use is concurrent with benzodiazepines or ethanol.

NON-PHARMACOLOGIC MANAGEMENT

Combinations of manual therapy (OMT and PT) with supervised exercise have been shown to decrease pain and improve functioning in patients suffering from a variety of chronic knee pain conditions. The most common conditions for which nonpharmacologic management is used are osteoarthritis (OA) and patellofemoral pain syndrome (PFPS). A study by Deyle showed that a combination of manual therapy applied to the lumbar spine, ankle, and pelvis yielded a significant functional benefit in patients with OA of the knee as well as delayed the need for surgery. The strengthening of the quadriceps muscle was shown to improve joint stability and significantly decrease pain. Studies have also revealed that there is some gluteal muscle strength weakness in those with patellofemoral pain syndrome, and hence gluteal strengthening can be an effective treatment. Pinto found that exercise therapy and manual therapy were more cost effective when compared to pharmacological therapy for OA of the knee.
<table>
<thead>
<tr>
<th>Etiologies, Diagnosis, and Treatment of Knee Pain</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical Presentation</strong></td>
</tr>
<tr>
<td><strong>Patellar Dislocation</strong></td>
</tr>
<tr>
<td><strong>MCL / LCL</strong></td>
</tr>
<tr>
<td><strong>ACL / PCL</strong></td>
</tr>
<tr>
<td><strong>Meniscal Injuries</strong></td>
</tr>
<tr>
<td><strong>Osteochondral Lesions / Osteochondritis Dessicans</strong></td>
</tr>
<tr>
<td><strong>Popliteus Tendinitis</strong></td>
</tr>
<tr>
<td><strong>Patellar Tendinitis</strong></td>
</tr>
<tr>
<td><strong>Iliotibial Band Syndrome</strong></td>
</tr>
<tr>
<td><strong>Patellofemoral Syndrome</strong></td>
</tr>
<tr>
<td><strong>Bursitis (Pes anserinus pain syndrome (PAPS))</strong></td>
</tr>
<tr>
<td><strong>Bursitis (Pre-Patellar) Housemaid’s Knee</strong></td>
</tr>
<tr>
<td><strong>Synovial Effusion</strong></td>
</tr>
<tr>
<td><strong>Osteoarthritis</strong></td>
</tr>
<tr>
<td><strong>Rheumatoid Arthritis</strong></td>
</tr>
<tr>
<td><strong>Gout</strong></td>
</tr>
<tr>
<td><strong>Pseudogout</strong></td>
</tr>
</tbody>
</table>

RICE: rest, ice, compression, and elevation; PRICE: physical therapy, rest, ice, compression, and elevation; PRICE OMM: PRICE with the additional application of OMM; PT: Physical Therapy; OT: Occupational Therapy.
FIGURE 1:
Diagnostic Algorithm for Knee Pain in the Primary Care Setting
This algorithm lists a step by step approach of how to diagnose and treat/refer a patient with knee pain as a primary care physician. While there are always rare etiologies, this covers the most common causes and how they can be ascertained based on the history.

Considering treatment options for chronic anterior knee pain (patellofemoral pain syndrome), Collins31 presented a meta-analysis reviewing twenty-seven studies investigating the effects of multimodal physiotherapy, manual therapy, exercise, tape, foot orthoses, electrotherapy, and acupuncture. Evidence from the meta-analyses strongly supported the use of multimodal physiotherapy while evidence from individual studies such as Bratingham,32 suggested only moderate clinical benefit of exercise, patella taping, foot orthoses, and acupuncture when compared to placebo.

Numerous studies have investigated OMT effectiveness in the treatment of knee pain over the last decade. Perlman33 found statistically and clinically significant decreases in pain after application of soft tissue (myofascial) and high velocity, low amplitude (HVLA) techniques in patients with knee OA.28 For patellofemoral pain syndrome, articulatory and myofascial techniques were found to significantly reduce pain, increase step test scores, and increase range of motion in a study by Van Den Dolder.28,34 Suter35 reports significant decreases in PFPS pain scores after treatment with HVLA combined with patellar mobilization, tape, exercise, and stretch.

OSTEOPATHIC APPROACH TO KNEE PAIN TREATMENT
In approaching the management of non-traumatic knee conditions, it is critical to conduct a careful exam of the knee, hip, foot and ankle joints and identify restrictions in ROM, tender points, and somatic dysfunctions (SD’s). To evaluate and treat the osteopathic findings, the common principles of each technique should be applied to the area of dysfunction and treated according to the anatomic region of the knee in which the SD is found. Table 3 (page 33 & 35) lists the common treatments as they apply to the patient with knee pain based on their associated clinical findings and diagnoses.6,34 The best studied conditions with proven OMT efficacy are osteoarthritis, patellofemoral pain syndrome and post-surgical care.6,34

KNEE CONDITIONS COMMONLY TREATED WITH OMT OSTEOARTHRITIS OF THE KNEE
The goals of non-pharmacologic treatment of knee OA are to control pain, improve function, and increase the patient’s ability to complete activities of daily living. OMT for OA consists of HVLA, muscle energy, articulation, and myofascial release.33,34 These techniques aim to improve arthritic pain, promote healing, and increase mobility. A study by Deyle29 demonstrated that OMT combined with standard medical care is more effective for OA treatment than standard medical care alone. Furthermore, the authors found that the combination of manual physical therapy and supervised exercise yielded functional benefits for patients with OA in the knee and delayed the need for surgical interventions.
### TABLE 1 (CONT.):

Etiologies, Diagnosis, and Treatment of Knee Pain

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Clinical Presentation</th>
<th>Diagnosis</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Infectious</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septic Arthritis</td>
<td>Febrile. One, painful, swollen joint with limited ROM</td>
<td>Radiographs. Assess neurovascular integrity</td>
<td>Non-displaced Fracture: Casting</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Displaced Fracture: Surgery</td>
</tr>
<tr>
<td>Viral Arthritis</td>
<td>Acute onset, symmetric polyarticular joint involvement, short duration, rash</td>
<td>Patella apprehension test. Radiographs</td>
<td>Patellar reduction with casting</td>
</tr>
<tr>
<td>Lyme Disease</td>
<td>Erythema migrans (early stage), nerve and cardiac symptoms (later stage), monoarthritis (late in disease)</td>
<td>Serological testing. Arthrocentesis if joint effusion</td>
<td>Antibiotics</td>
</tr>
<tr>
<td><strong>Referred</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extrinsic Pain</td>
<td>Non-localized knee pain with concurrent thigh / calf pain</td>
<td>Lumbar, sacroiliac, hip, knee and ankle exam</td>
<td>Address underlying case of pain</td>
</tr>
<tr>
<td>(myotomal, dermatomal, sclerotomal)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Vascular</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Popliteal artery aneurysm</td>
<td>Claudication. Fullness or pain behind knee if large</td>
<td>Duplex Ultrasound</td>
<td>Symptomatic or &gt; 2.0 cm-thrombolytic therapy, surgical repair</td>
</tr>
<tr>
<td>Deep Vein Thrombosis (DVT)</td>
<td>Swelling, pain, erythema</td>
<td>Compression ultrasonography</td>
<td>Anticoagulant therapy, thrombolitics, IVC filter</td>
</tr>
<tr>
<td>Hemarthrosis</td>
<td>Usually caused by trauma (ACL tear, fracture), immediate swelling within 2 to 4 hours</td>
<td>Joint aspiration if diagnosis unknown</td>
<td>RICE, analgesics, and arthrocentesis</td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tumor Osteochondroma</td>
<td>Painless bump near joints; pain with activity; numbness/tingling</td>
<td>Bony growth on X-ray; MRI/CT to confirm</td>
<td>Observation; Excision if symptomatic</td>
</tr>
<tr>
<td>Popliteal (Baker’s) Cyst</td>
<td>Fluid-filled mass within popliteal fossa</td>
<td>Medial popliteal mass prominent with full extension</td>
<td>Fluid drainage: PT; Medications: corticosteroids</td>
</tr>
<tr>
<td>Plica</td>
<td>Anterior-medial knee pain; snapping with flexion/extension</td>
<td>Inelastic, band-like structure on palpation; redundant folds in CT on MRI</td>
<td>Stretching/strengthening; steroid injections; refractory: arthroscopic band resection</td>
</tr>
</tbody>
</table>

*RICE: rest, ice, compression, and elevation; PRICE: physical therapy, rest, ice, compression, and elevation; PRICE OMM: PRICE with the additional application of OMM; PT: Physical Therapy; OT: Occupational Therapy.*

---

**PATELLOFEMORAL PAIN SYNDROME**

Patellofemoral pain syndrome is a common, chronic overuse condition presenting with anterior knee pain (Table 1). Nonsurgical modalities are the primary treatment method. Collins conducted a systematic review and meta-analysis on the short- and long-term efficacy of non-surgical interventions for PFPS. Interventions studied were modal physiotherapy, manual therapy, exercise, tape, foot orthoses, electrotherapy, acupuncture, and pharmacotherapy. The results of the study showed favorable effects for multimodal physiotherapy compared to other nonsurgical interventions.

---

**POST-SURGICAL CARE**

To optimize a patient’s return to normal function after surgery, OMT can address preoperative musculoskeletal findings as well as somatic dysfunctions that develop during rehabilitation. An anterior cruciate ligament (ACL) tear is one of the most common and debilitating knee injuries. A JAOA Case report by Gugel presents a 27-year-old patient who was actively treated with OMT after undergoing ACL reconstruction. OMT was used to address specific somatic dysfunctions in the patient’s neck, thoracic, and lumbar/sacrum/pelvic areas. The patient was able to return to his athletic activities without restrictions 6 months following the reconstruction.
### TABLE 2:
Special Tests for Diagnosis of Knee Pain

<table>
<thead>
<tr>
<th>Test</th>
<th>Method or Appearance</th>
<th>Pictures</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Varus-Valgus Stress Test</td>
<td>Abduction/adduction motion to the proximal tibia with knee extended and flexed</td>
<td></td>
<td>Laxity at 30 degrees = Injury to the MCL (valgus) or LCL (varus)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Laxity at 0 degrees = Injury to the MCL/LCL and PCL</td>
</tr>
<tr>
<td>Lachman Test (most sensitive)</td>
<td>30 degrees of flexion, one hand on tibia and other on thigh, articulate tibia anteriorly</td>
<td></td>
<td>Positive test = anterior translation of the tibia on the femur = ACL injury</td>
</tr>
<tr>
<td>Pivot Shift Test</td>
<td>Knee in extension. Internally rotate tibia and place valgus stress on knee</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior Drawer Test</td>
<td>90 degrees of flexion. Translate tibia anteriorly</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterior Drawer Test</td>
<td>90 degrees of flexion. Translate tibia posteriorly</td>
<td></td>
<td>Positive test = posterior translation of the tibia = PCL injury</td>
</tr>
<tr>
<td>McMurray’s Test</td>
<td>Monitor joint line, flex knee, internally rotate tibia and apply a varus stress while extending the knee, or externally rotate tibia and apply a valgus stress while extending the knee</td>
<td></td>
<td>Palpable click or pop and pain = medial or lateral meniscal injury</td>
</tr>
<tr>
<td>Apley’s Compression Test</td>
<td>90 degrees of flexion, press on heel down while internally/externally rotating foot</td>
<td></td>
<td>Joint pain = medial or lateral meniscal injury</td>
</tr>
<tr>
<td>External Rotation - Recurvatum Test</td>
<td>Lift patient’s leg by great toe while stabilizing distal thigh, 10 degrees of flexion, release calf to allow full extension</td>
<td></td>
<td>Knee hyperextended and tibia externally rotated = injury to the posterolateral corner (PCL) - fibular collateral ligament, arcuate ligament and the popliteus</td>
</tr>
<tr>
<td>Knee Joint Effusion Test (Bounce Home Test)</td>
<td>Knee extended, push patella inferiorly, tell patient to contract quadriceps muscles</td>
<td></td>
<td>Knee unable to fully extend = abnormal amount of joint fluid</td>
</tr>
<tr>
<td>Patellofemoral Grind Test</td>
<td>Knee extended, push patella inferiorly, tell patient to contract quadriceps muscles</td>
<td></td>
<td>Increased patellar motion, pain or crepitus = Deterioration of the cartilage under the patella (possibly) patellar chondromalacia)</td>
</tr>
<tr>
<td>Thessaly Test</td>
<td>Patient on one leg, holding onto examiners hands for balance, patient flexes knees to 20 degrees and rotates femur on tibia medially and laterally while maintaining flexion</td>
<td></td>
<td>Medial or lateral joint line discomfort, or a sense of locking or catching of the knee = meniscus tear</td>
</tr>
</tbody>
</table>
### TABLE 3:
OMT Treatments of Knee Pain

<table>
<thead>
<tr>
<th>Technique</th>
<th>Region of Treatment</th>
<th>Clinical Findings</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Muscle Energy:</strong></td>
<td></td>
<td>Foot inversion, foot adduction, tibial rotation</td>
<td>Symptoms of compression of peroneal nerve</td>
</tr>
<tr>
<td></td>
<td>Posterior Fibular Head</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anterior Fibular Head</td>
<td></td>
<td>Lateral Knee Pain</td>
</tr>
<tr>
<td></td>
<td>Tibiofemoral joint:</td>
<td>Internal rotation of femur, external rotation of tibia (due to relaxation of popliteus)</td>
<td>OA, RA, Baker’s Cyst</td>
</tr>
<tr>
<td>Knee Extension/Flexion, Internal / External Rotation Somatic Dysfunction</td>
<td></td>
<td>External rotation of femur, internal rotation of tibia (due to contraction of popliteus)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hip: anterior / posterior rotation, superior / inferior shear, inflare / outflare somatic dysfunction</td>
<td>Flexion / Extension Abduction / Adduction</td>
<td>Extrinsic causes / Referred Pain (see Figure 2)</td>
</tr>
<tr>
<td>Lumbar Spine</td>
<td>Type I SD</td>
<td></td>
<td>Neutral Group Curve</td>
</tr>
<tr>
<td></td>
<td>Type II SD</td>
<td></td>
<td>Non-neutral Group Curve</td>
</tr>
<tr>
<td><strong>Counterstrain:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Anterior Patella</td>
<td>T.P - Patellar tendon</td>
<td>Patellofemoral pain syndrome</td>
</tr>
<tr>
<td></td>
<td>Medial/Lateral Patella</td>
<td>T.P - Medial or lateral patellar surface</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Posterior Knee</td>
<td>T.P - Medial or Lateral ACL T.P - Center of Popliteal Fossa T.P - Lower popliteal fossa</td>
<td>ACL/PCL injury; Gastrocnemius sprain; Gastrocnemius (Baker’s cyst); DVT</td>
</tr>
<tr>
<td></td>
<td>Medial Knee</td>
<td>T.P - Medial Joint Line T.P - Medial hamstring muscle, distal attachment</td>
<td>Medial Meniscus injury, OA, pes anserine bursitis, medial plica syndrome, medial collateral ligament sprain, medial meniscal tear</td>
</tr>
<tr>
<td></td>
<td>Lateral Knee</td>
<td>T.P - Lateral joint line T.P - Lateral hamstring, distal attachment, near fibular head</td>
<td>Lateral meniscus injury, lateral compartment OA, lateral collateral ligament sprain, lateral meniscal injury, iliobibial band tendinitis</td>
</tr>
<tr>
<td><strong>FPR:</strong> Articulation is placed into freedoms. Compression is applied to shorten involved muscle. Joint is moved in direction of muscle being treated and hold until release</td>
<td>Tibiofemoral joint</td>
<td>Point tenderness at and medial to midpoint of knee joint</td>
<td>OA, pes anserine bursitis medial plica syndrome, medial collateral ligament sprain, medial meniscal tear</td>
</tr>
<tr>
<td><strong>HVLA:</strong> Restricted joint placed into restrictive barrier(s). A small to moderate amount of force is applied to the joint in a way that moves it through its barriers</td>
<td>Anterior / Posterior Fibular Head</td>
<td>Lateral Knee Pain; if Posterior Fibular Head symptoms of peroneal nerve compression</td>
<td>Lateral Compartment OA, lateral collateral ligament sprain, lateral meniscal tear, iliobibial band tendinitis</td>
</tr>
</tbody>
</table>

*FPR: Fascilitated Positional Release; HVLA- High Velocity Low Amplitude, T.P.- Tender point*
DISCUSSION/CONCLUSION

With knee pain accounting for almost a third of primary care visits, osteopathic family physicians play an important role in improving their patient’s overall quality of life. While the differential for patients presenting with knee pain is extensive, it is important for the family physician to combine their knowledge of knee anatomy, the common etiologies of knee pain, a detailed history, and a complete osteopathic structural exam to come up with an appropriate diagnosis and treatment plan. Osteopathic physicians hence provide a new approach to the management of these patients through incorporating osteopathic principles into their diagnosis and treatment. Manipulation has been shown to significantly reduce pain and improve functionality in patients with a wide range of knee pain etiologies. Future studies must be conducted to establish an OMT protocol that can be used and identify other etiologies of knee pain for which OMT is effective. However, the progress that has been made over the years is remarkable as it is and represents how OMT should be used as a standard of care for patients with knee pain.

REFERENCES:


35. Perlman A. Massage Therapy for Osteoarthritis of the Knee. Archives of Internal Medicine. 2006;166(22):2533.
### TABLE 3 (CONT): OMT Treatments of Knee Pain

<table>
<thead>
<tr>
<th>Technique</th>
<th>Region of Treatment</th>
<th>Clinical Findings</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Articulatory Technique:</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Joint is repeatedly taken through physiologic range of motion in all possible planes</td>
<td>Tibiofemoral joint</td>
<td>Decreased ROM in Flexion / Extension or Internal / External Rotation</td>
<td>OA</td>
</tr>
<tr>
<td><strong>Myofascial/Soft Tissue (Popliteal Spread):</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior and lateral force is applied to popliteal fossa to engage fascial barriers</td>
<td>Popliteal Fossa</td>
<td>Decreased lymphatic drainage proximal to tibiofemoral joint</td>
<td>Lymphedema (i.e- post-op); popliteal cyst; non-inflammatory effusion</td>
</tr>
</tbody>
</table>


Delirium is a common acute geriatric syndrome with a fluctuating course that is characterized by inattention and cognitive changes that may not be attributed to dementia. Older patients, those with comorbidities or history of psychiatric illness as well as those with cognitive disorders or geriatric syndromes are at increased risk of developing delirium. Delirium is multifactorial and is often the first indicator of an acute illness in the geriatric patient. The work-up for delirium should include review of the patient’s medications, evaluation for environmental factors as well as laboratory and radiologic studies. The mainstay for treating delirium is to identify and treat the underlying cause. Many treatment measures are also good preventive measures and include establishing normalcy for the patient by providing a care environment that is as similar to their home environment as possible and maintaining their daily schedule and regimen. Physical restraints should not be used and pharmacologic treatment should only be considered when there is concern about the patient’s safety or the safety of others, non-pharmacologic treatments have already been utilized, and the underlying cause has been treated. Delirium has many long-term effects including distress, cognitive decline, loss of function, and increased morbidity and mortality. Patients with delirium also have longer hospital stays and there is increased economic cost.

Keywords: Delirium, Geriatrics, Elderly, Psychiatry, Behavioral Medicine

INTRODUCTION

“She slept well all night, not a peep.” These were the words of the nurse caring for my 89-year-old patient. Our geriatric medicine team was consulted by the orthopedics service for “medical management.” I learned that the patient was an 89-year-old female who was admitted three days ago after a fall. Her fall resulted in an intratrochanteric hip fracture and she underwent open reduction internal fixation in the OR within 24 hours of admission. Her operative course went well, with no complications. Prior to being hospitalized, she was living in her own home alone and only required occasional assistance from her son with some instrumental activities of daily living including shopping and managing finances. She was a retired college professor who enjoyed spending her time volunteering as an usher at the theater. Her past medical history included osteoarthritis and she took acetaminophen on occasion to control her joint pains. According to her nurse, she was awake briefly in the recovery room but had been sleeping since. The patient’s vital signs had been stable and her routine labs were unremarkable. On review of her hospital medications, I found that she had not received any medications in two days as the nurses were holding all medications due to her somnolence.

DELIRIUM DEFINED

Would you recognize the above clinical case as delirium? Delirium is a common syndrome in the geriatric patient that is under-diagnosed and carries great risks including increased mortality. The word “delirium” is derived from three Latin roots, de which means “away from,” lira which means “furrow in a field,” and ium meaning “going off the ploughed track, a madness.” According to the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5), delirium is defined as an acute syndrome characterized by inattention, cognitive changes that may not be attributed to dementia, acute onset (usually developing over hours to days) with fluctuation, and cause derived from a precipitating factor such as an underlying medical condition, intoxicating substance, adverse drug event, or multifactorial causes. The DSM-5 has not been widely studied yet, but the criteria for delirium appear less subjective than DSM-4. Non-detection rates of delirium using DSM-4 were reported to be 32%-67%.

Many words have been used to describe delirium. “Sundowning” is a term that is commonly used and describes the time period when delirium is most often detected, at night. Patients experiencing delirium tend to demonstrate signs of confusion most at night, after “sun down” when there is less structure or routine in their care setting and more negative stimulation (such as the sounds of beeping alarms and hallway traffic in the hospital setting). Older patients are known to be more vulnerable to the syndrome.

Epidemiology

Epidemiological studies of delirium most commonly include hospitalized older patients as opposed to patients in post-acute and community settings. Studies of hospitalized older patients have reported the prevalence of delirium at admission as 14-24% and the incidence during hospitalization as 6-56%. Rates in the intensive care unit have been reported as 70-87% and rates post-orthopedic surgery have been reported as 15-53%. Furthermore, from the epidemiological studies conducted in long-term care and post-
Acute settings, a rate of up to 60% has been reported. Delirium is thought to occur in up to 83% of patients at the end of life.

It is estimated that approximately 1.5 million older patients with delirium will present to the emergency department each year and emergency physicians fail to diagnose delirium 75% of the time that it is present. This lack of recognition of delirium spans across all specialties with delirium being missed in up to 32-66% of cases. Many factors influence a physician’s ability to recognize delirium. Improving diagnosis of the syndrome may be achieved through physician education and care being taken by the osteopathic family physician to look closely for the syndrome.

Pathophysiology

Although the exact pathophysiology of delirium is still not well known, it is felt that delirium is most likely due to a functional as opposed to a structural lesion. Electroencephalographic (EEG) findings have pointed towards functional derangements and decrease in cortical activity has been noted. The current main hypotheses propose that delirium is the “final common pathway of many different pathogenic mechanisms, resulting from dysfunction of multiple brain regions and neurotransmitter systems.”

Subtypes

There are three subtypes of delirium: hyperactive, hypoactive, and mixed. Patients with hyperactive delirium are most easily recognized. These patients are truly “hyperactive” demonstrating increased psychomotor activity and may appear restless, anxious, or agitated and may have behavioral disturbances that are combative. They may display loud or fast speech, swearing, singing, laughing, anger, wandering, or other increased activity. Patients with hyperactive delirium are often the ones that the nurse calls you about at night as their delirium is most easily recognized in the clinical setting. Hypoactive delirium is the “quiet delirium” that often goes unrecognized. These patients have decreased psychomotor activity and may appear to be sleeping all the time or sedated, thought to be depressed, or possibly even lethargic. They may appear to be staring blankly, have little conversation, or demonstrate slow speech. These are often the patients that do not cause any disturbance at night and appear to be resting comfortably. Because they are “quiet,” they often do not evoke clinical concern. Older patients tend to commonly experience hypoactive delirium. The most commonly diagnosed subtype is mixed. It is composed of characteristics of both hyperactive and hypoactive delirium and thus has fluctuating levels of psychomotor activity.

RISK FACTORS

A small insult can precipitate delirium in a geriatric patient who has many risk factors. Some risk factors include:

Age

Patients older than age 65 and of the male sex have increased risk for delirium. This is especially true following procedures and in different care settings. For example, Allen and Frankel have reported that up to 50% of elderly patients suffer from delirium postoperatively. Furthermore, patients who have undergone orthopedic procedures (as the patient in the above case) are more likely to develop delirium than patients who have undergone general surgery procedures. It is estimated that 28% to 61% of geriatric patients with a hip fracture will experience delirium. It is important for the osteopathic family physician to recognize older age alone as a known risk factor. It should also be noted that a patient’s chronologic age may not correlate to their biologic age. Therefore, the patient’s actual age as well as their overall medical condition and determination of their biologic age should be taken into account.

Comorbidities and History of Psychiatric Illness

Patients with multiple acute or chronic medical conditions are more likely to suffer from delirium. The prevalence of older persons with delirium in the intensive care unit has been found to be 60% to 80% for those with mechanical ventilation and 20% to

<table>
<thead>
<tr>
<th>Hyperactive</th>
<th>Hypoactive</th>
<th>Mixed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased psychomotor activity</td>
<td>Decreased psychomotor activity</td>
<td>Characteristics of both hyperactive and hypoactive</td>
</tr>
<tr>
<td>Restlessness / Anxious</td>
<td>Decreased alertness / Sleepy</td>
<td>Fluctuating levels of psychomotor activity</td>
</tr>
<tr>
<td>Loud or Fast Speech</td>
<td>Slow or little speech / Quiet</td>
<td></td>
</tr>
<tr>
<td>Agitation / Combativeness / Anger</td>
<td>Unawareness / Staring blankly</td>
<td></td>
</tr>
<tr>
<td>Laughing, Singing, Swearing</td>
<td>Apathy / Appear Depressed</td>
<td></td>
</tr>
<tr>
<td>Hypervigilance</td>
<td>Lethargy</td>
<td></td>
</tr>
<tr>
<td>Distractability</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tangentiality</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Persistent thoughts</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wandering</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
50% for those without mechanical ventilation. Brummel and Girard have referenced that the average medical ICU patient possesses eleven or more risk factors for delirium. Patients with a history of alcoholism, use of intoxicating substances, and psychiatric illness are also more likely to be afflicted with delirium. Patients who reside in a long-term care setting are at a high risk of delirium as residents in long-term care tend to have more comorbidities and are more likely to have cognitive and physical impairments.

**Cognitive Disorders / Geriatric Syndromes**

Baseline cognitive disorders (such as mild cognitive disorder, dementia or history of memory impairment secondary to stroke) increase a patient’s risk of delirium. The risk of delirium also increases with the severity or stage of dementia. Geriatric syndromes as a whole have been shown to be a predisposing factor for delirium. These include: dementia, immobility or decrease in function, sensory impairments including hearing loss and visual disturbances, malnutrition, depression, frailty and falls, polypharmacy, previous history of delirium, history of elder abuse, and pressure ulcers as well as others.

**CAUSES OF DELIRIUM**

Delirium is typically multifactorial and it may be impossible to isolate just one cause of delirium in a patient. Commonly, delirium is the first indicator of an underlying acute illness. Geriatric patients, especially, may demonstrate delirium prior to the development of vital sign changes such as fever, tachycardia, tachypnea or hypoxia. Some of the most common causes seen in the geriatric patient include:

**Infection**

Infections are one of the most common causes of delirium. Of patients who develop delirium due to infection, urinary tract infections and pneumonia account for 34% to 43% of these cases. Assessing for infection should always be part of the diagnostic evaluation for delirium.

In the geriatric patient, delirium may be the first clinical indication of infection as vital sign changes and other clinical signs often present later in the clinical course. Some geriatricians consider delirium to be the sixth vital sign. In advocating that mental status should be the sixth vital sign, Flaherty et al. have argued that “the brain is as sensitive and vital an organ as the immune (temperature), cardiac (pulse, blood pressure), and respiratory systems (respiratory rate) for heralding that something is amiss.” Furthermore, “each vital sign is nonspecific but an abnormal or changed value may signal something is wrong” and “in frail older patients with an infection, a change in mental status often occurs before a change in pulse, blood pressure, or respirations.” In evaluation of the geriatric patient, it is most important for the osteopathic family physician to consider delirium as a sixth vital sign.

**External Devices, Environmental Factors, & Sleep**

Any changes from the norm for a geriatric patient may contribute to delirium. When one thinks about the multiple changing factors that occur when a geriatric patient transitions from living at home to being hospitalized, it can be overwhelming just to think about. Imagine what this experience is like for a geriatric patient. The more transitions that occur, the more likely he/she is to develop delirium.

---

### TABLE 2:

<table>
<thead>
<tr>
<th>Risk Factors for Delirium&lt;sup&gt;1,4&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age greater than 65</td>
</tr>
<tr>
<td>Male sex</td>
</tr>
<tr>
<td>Comorbidities</td>
</tr>
<tr>
<td>Alcoholism / substance abuse</td>
</tr>
<tr>
<td>Depression and history of psychiatric illness</td>
</tr>
<tr>
<td>History of chronic pain</td>
</tr>
<tr>
<td>Dementia and other cognitive disorders</td>
</tr>
</tbody>
</table>

### TABLE 3:

<table>
<thead>
<tr>
<th>Class</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotics</td>
<td>Quinolones, Macrolides, Linezolid, Antimalarials</td>
</tr>
<tr>
<td>Antidizziness, Vertigo</td>
<td>Scopolamine, Meclizine</td>
</tr>
<tr>
<td>Antihistamines</td>
<td>Diphenhydramine, Hydroxyzine</td>
</tr>
<tr>
<td>Antiemetics</td>
<td>Promethazine</td>
</tr>
<tr>
<td>CNS System / Psych</td>
<td>Benzodiazepines, Anticonvulsants, Sedatives, TCAs</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>Amiodarone, Digoxin, Diltiazem, Beta blockers, Clonidine</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Metoclopramide, Cimetidine, Ranitidine, Atropine</td>
</tr>
<tr>
<td>Pain / Anti-Inflammatory / Musculoskeletal</td>
<td>Corticosteroids, NSAIDs, Muscle Relaxants, Narcotics</td>
</tr>
</tbody>
</table>

It is estimated that the average ICU patient carries 11 or more risk factors for delirium. The setting of the intensive care unit alone also places them at risk as it is far from the norm of their daily life. Often, these patients are in isolation and everyone that enters their room is not easily recognizable to them due to all the protective clothing that must be worn by healthcare team members and visitors. There are many “tethers” on the intensive care patient. These may include a bladder catheter, telemetry monitor, continuous pulse oximetry, perhaps endotracheal tube, gastric tube, routine blood pressure monitor, etc. All of these may cause overstimulation and contribute to delirium. Lack of sleep appears to be
a major factor in the development of delirium in the ICU. Several studies have found the correlation of lack of sleep to delirium and it has been found that the average amount of sleep in ICU patients is approximately 1 hour and 51 minutes in a 24-hour time period.1

**Meds, Meds, Meds Until Proven Otherwise**

Medications should be considered to be a cause of delirium in the geriatric patient until proven otherwise. As the number of medications in a patient’s regimen increases, so does the risk for delirium. The highest incidence of medication-induced delirium is noted in patients taking more than three medications.2 Medications with anticholinergic properties are the most notable for precipitating delirium in the geriatric patient. These include diphenhydramine, promethazine, hydroxyzine, meclizine, amitriptyline among others.7 Some medications, such as benzodiazepines can contribute to delirium in patients but also have a protective effect in others.7 It is important to review the patient’s medication list daily to investigate for any medications that may be causing delirium or place the patient at risk. Some medications are more obvious than others.

Other causes include inadequate pain control, dehydration, metabolic abnormalities (such as hepatic or renal failure, electrolyte disturbances, hypo/hyperglycemia), cerebrovascular accident, acute myocardial infarction, seizure, subdural/epidural hematoma, meningitis or encephalitis, hypoxia/respiratory failure, hypoten:sion, hypoperfusion, congestive heart failure, trauma, shock, constipation, and urinary retention.7,8

The osteopathic family physician must keep all systems in mind as well as medications and environmental factors.

**ASSESSMENT & DIAGNOSIS**

Assessment for delirium should begin on initial evaluation in the emergency department and ongoing assessment should occur regularly as signs of delirium may fluctuate throughout day and night.2 Several tools exist to assess for delirium, but the Confusion Assessment Method (CAM) is the most widely embraced by healthcare providers.7 The CAM has 4 features:7

1. Acute mental status change and fluctuating course
2. Inattention
3. Disorganized thinking
4. Altered level of consciousness

In order to meet criteria for the diagnosis of delirium, a patient must have features 1 and 2 and either feature 3 or 4.7 The CAM has been found to have sensitivity of 94%-100% and specificity of 90%-95% in screening hospitalized patients.13

Geriatric patients with delirium should be admitted to the hospital for further investigation as geriatric patients who are discharged from the emergency department have higher death rates than patients without delirium.7 The diagnostic evaluation should be focused on finding the underlying cause.6 In addition to taking a complete history (including medications and any medication changes, history of drug/alcohol use) and performing a thorough physical examination (including neurological), the evaluation of the geriatric patient with delirium includes laboratory and perhaps radiologic studies. Table 4 summarizes these studies that should be considered. Clinical judgment must be used to determine studies that are appropriate for each patient.

**TREATMENT & PREVENTION**

Many of the treatment measures for delirium are also good preventive measures. When a hospitalized geriatric patient becomes delirious, the patient benefits from efforts to maintain a regular schedule and create surroundings that are as close to their home life as possible. This includes insuring that the patient is out of bed for meals unless contraindicated, establishing early mobility, occupational and physical therapy, setting a day/night and wake/sleep schedule with positive cognitive stimulation during the day (including turning on the lights, opening the blinds so that sunlight is in the room, and avoiding daytime naps) and limited interruptions to allow for restful sleep at night, surrounding the patient with familiar items from home and encouraging family and friends to visit regularly.12 If a patient wears hearing aids or glasses at home, he/she should be wearing them in the hospital (even in the ICU). Interruptions at night should be limited and noise kept to a minimum when the patient is sleeping. As physicians, we should avoid ordering frequent checks of vital signs, procedures, lab draws, and radiologic studies (especially at night) unless absolutely needed for patient safety.6 Regular medications should be given during daytime hours when possible.

Physical restraints are not recommended for managing delirium or for use in patients at risk of delirium. In fact, the use of physical restraints increases the risk of a patient developing delirium and also have been found to increase the severity of delirium.14 Oftentimes, physicians order physical restraints because they believe that this will prevent injury from falls. This is a misconception as studies have demonstrated an increased fall rate with the use of physical restraints.14

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**TABLE 4:**

<table>
<thead>
<tr>
<th>Laboratory</th>
<th>Radiologic / Other</th>
</tr>
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<tr>
<td>Complete blood count</td>
<td>12-lead electrocardiogram</td>
</tr>
<tr>
<td>Comprehensive metabolic panel (electrolytes, glucose, BUN/Creatinine, LFTs)</td>
<td>Chest radiograph</td>
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<tr>
<td>Ammonia</td>
<td>CT of the head</td>
</tr>
<tr>
<td>Urinalysis / urine culture</td>
<td>Electroencephalography (if seizure expected or delirium is unclear)</td>
</tr>
<tr>
<td>Cardiac biomarkers</td>
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<tr>
<td>Lumbar puncture</td>
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<td>Blood cultures</td>
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<td>Thyroid-stimulating hormone</td>
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<tr>
<td>Vitamin B12 &amp; Folate levels</td>
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<tr>
<td>Urine drug screen</td>
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<tr>
<td>Arterial blood gas</td>
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<td>Rapid plasma reagin</td>
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Before considering pharmacologic treatment for delirium, the above interventions should be taken and the underlying cause should be treated. The patient should be evaluated for pain. Sometimes, patients with hypoactive delirium and/or those with cognitive decline are unable to voice their pain. A regular regimen for pain control often results in resolution of their delirium. If delirium persists after the underlying medical condition has been treated and environmental interventions have been taken, pharmacologic management may be needed. In general, benzodiazepines should be avoided as they are known to not only cause but also exacerbate delirium. The American Psychiatry Association advises only using benzodiazepines in the setting of alcohol and benzodiazepine withdrawal, not as monotherapy in delirious patients. Instead, antipsychotic medications are recommended. Haloperidol is known as the “agent of choice” but as with all antipsychotics, must be prescribed with caution and attention paid to possible adverse effects such as extrapyramidal effects, prolonged corrected QT interval/torsades de pointes, among others. Halo- peridol should be avoided if a patient has underlying parkinsonism, withdrawal syndrome, hepatic insufficiency, or neuroleptic malignant syndrome. Osteopathic family physicians should consult the U.S. Food and Drug Administration boxed warnings for medications prior to prescribing them for delirium and also evaluate risks/benefits to the patient and discuss this openly with the patient when possible and the family/health care surrogate. Pharmacologic treatment for delirium should be the last option chosen in treatment and used only when there is concern about the patient’s safety or that of others.

LONG-TERM EFFECTS: DISTRESS, COST, & DEATH

Only 14% of patients with delirium have returned to their baseline level of cognitive functioning at discharge. Many times, this results in the need for placement in long-term care as opposed to discharge to home. Delirium is a strong prognostic indicator and is associated with increased morbidity and mortality. Post-operative delirium is linked to increased morbidity as well as a 1 year mortality of 40%. Han and colleagues found that delirium in geriatric patients in the emergency room is an independent predictor of increased 6-month mortality. Patients with delirium also stay hospitalized for an average of 5-10 days longer than patients who have the same medical problems but have not had delirium. The economic cost for care is also increased. On average, patients in the ICU with delirium have health care costs that are 31% higher than patients with the same medical problems but without delirium. The national burden of delirium on the health care system is somewhere between $32 billion to $152 billion per year. Grover and Shah studied distress due to delirium and found that the overall experience of delirium distresses the patient and the majority of patients studied reported at least a moderate level of distress post-delirium.

THE FUTURE

“Not a peep” as described in the introductory case should raise concern for hypoactive delirium. For this post-operative patient, treatment involved a scheduled regimen of pain medication and nursing staff was educated about the clinical signs of hypoactive delirium and the importance to not hold the patient’s doses unless there were signs of respiratory depression, bradycardia, hypotension, or other clinical concern. Nursing staff was asked to contact the geriatric medicine team to evaluate the patient if there was any concern about giving the patient’s scheduled pain medication. With the patient receiving her scheduled regimen of pain medication, she slowly returned to her normal cognition. With time, she only required pain medication prn and was subsequently discharged to sub-acute rehab.

Although a large amount of research has been conducted on delirium and much is understood, there remain many opportunities for investigation as the causes of delirium are multifactorial and the treatments (pharmacologic and non-pharmacologic) are numerous. Continuity of care is very beneficial to the patient with delirium and thus there is a great role for the osteopathic family physician to affect outcomes. The approach to treating delirium must involve educating the healthcare team and be multidisciplinary with the osteopathic family physician serving as the team leader.

REFERENCES:

Underlying Appendicitis Leading to Chorioamnionitis in Preterm Rupture of Membranes

Jennifer Gibbs, DO, Firas Bridges, MD, John J. Vullo, DO, & Anthony Sampino, DO

Department of Obstetrics & Gynecology and Department of General Surgery, Good Samaritan Hospital Medical Center, West Islip, NY

**Background:** PPROM complicates 3% of pregnancies, the most commonly identified etiology is infection. Appendicitis is a well-known cause of peritonitis and systemic illness, complicating approximately 1/1700 pregnancies.

**Case:** A healthy 26 year old primagravida female at 24 weeks gestation presented with PPROM. She was managed expectantly and delivered at 26 weeks gestation due to suspected chorioamnionitis, manifested by abdominal pain and tenderness. Postpartum the patient complained of mild abdominal pain and nausea that was deemed appropriate for her post-operative state, and she was discharged home on post-operative day 3. The following day she presented to our emergency department with worsening abdominal pain. Imaging was suggestive of appendicitis, and the patient subsequently underwent surgery. Intra-operative findings were significant for an inflamed appendix matted to the posterior surface of the uterus and diffuse erythema of the uterine serosa. Final pathology reports confirmed acute appendicitis, chorioamnionitis and funisitis.

**Conclusion:** It is possible that an underlying appendicitis lead to intrauterine infection and subsequent preterm delivery in our patient.

**INTRODUCTION**

Preterm birth complicates 11% of all pregnancies, and 3% of all pregnancies are affected by preterm premature rupture of membranes (PPROM). In cases of PPROM, the most commonly identified etiology is infection. Here we will discuss a case of a healthy 26-year-old primagravida female, with an antenatal period complicated by PPROM occurring at 24 weeks’ gestation, with delivery at 26 weeks secondary to suspected chorioamnionitis. In the immediate post-operative period, the patient was re-admitted to the hospital and underwent surgery for acute appendicitis; leading to the question of which came first, the chorioamnionitis or the appendicitis?

**Teaching Point:**

Inflammation, from any organ system, can lead to the pathological cascade causing PPROM subsequent preterm delivery.

**CASE**

26-year-old G3P0020 African American female at 24 6/7 weeks’ gestation by first trimester sonogram presented to the labor and delivery unit of a small community hospital with complaint of leakage of fluid. PPROM was confirmed and the patient was transferred to our facility for further management. She was admitted to the antepartum service for expectant management. Following ACOG recommendations the patient received antenatal corticosteroid for fetal lung maturity, magnesium sulfate for neuroprotection and a 7-day course of antibiotics for latency. She was monitored closely with serial abdominal exams and daily lab work trending white blood cell count. Fetal monitoring consisted of continuous tocometry and external fetal monitoring. On hospital day 7, the patient developed a mild leukocytosis of 14,000. At this time, she complained of abdominal pain, but on physical exam, the abdomen was soft with no fundal tenderness, rebound or guarding. She remained afebrile with reassuring fetal heart rate tracing and was monitored closely.

On hospital day 10, at 26w1d gestation, the patient developed worsening abdominal pain. On exam, she displayed obvious abdominal tenderness. In conjunction with maternal fetal medicine, the decision was made to proceed to delivery for suspected chorioamnionitis. She underwent a primary classical cesarean section with a double layer uterine closure under spinal anesthesia. Intra-operative findings were significant for: 1) viable female infant with APGARs of 3 and 8, weighing 1lb 10oz (775g); 2) no amniotic fluid 3) friable placenta, noted to be unhealthy in appearance 4) diffuse irritation and erythema of the uterine serosa. Placenta cultures were obtained at time of delivery. The patients post-operative course was significant for mild leukocytosis of 13,000 and abdominal discomfort which was deemed appropriate for procedure. On post-operative day 3 the patient was discharged home.
The following day, the patient presented to the emergency room with complaint of lower abdominal pain, worsening since hospital discharge. On arrival to the ED, she was afebrile with WBC 14,000. Pfannenstiel skin incision was healing well without obvious evidence of infection. On exam the patient was noted to have tenderness in the right and left lower quadrant and a CT scan was significant for a dilated appendix and multiple appendicoliths. The patient was evaluated by general surgery, and the decision was made to proceed with surgical management. She subsequently underwent a laparoscopic appendectomy. Intraoperative findings were significant for inflamed appendix, markedly adherent to the posterior uterus, with diffuse erythema of the uterine serosa and surrounding peritoneum. Additionally there were multiple adhesions from the uterus to nearby structures including the bowel and anterior abdominal wall. Post-operatively she recovered well and was discharged home on post-operative day.¹

DISCUSSION

Preterm premature rupture of membranes (PPROM) complicates 3% of all pregnancies and is defined as rupture of membranes before 37 weeks’ gestation. Management of PPROM is based on the gestational age. Delivery is recommended when PPROM occurs in late preterm gestations (34 0/7 – 36 6/7 weeks). Regarding PPROM in patients ranging from fetal viability to 33 6/7 weeks, such as our patient, expectant management is warranted to prolong the latency period. Latency is defined as time of rupture to time of delivery. This expectant management includes: antenatal corticosteroids, GBS prophylaxis, antibiotics and in those patients less than 32 0/7, magnesium sulfate for neuroprotection. As discussed previously, our patient was managed according to ACOG guidelines.²³

During expectant management, patients must be monitored closely for fever, uterine tenderness, membranes, the most reliable indicator is fever, defined as >38C. Patients must be monitored closely for the development of chorioamnionitis, defined as acute inflammation of the placental membranes. In the face of rupture of membranes, the most reliable indicator is fever, defined as >38C. Patients must be monitored closely for fever, uterine tenderness, maternal or fetal tachycardia and malodorous vaginal discharge. Fetal and neonatal morbidity is increased significantly in the case of chorioamnionitis. Those affected have higher incidence of RDS, IVH, sepsis and periventricular leukomalacia. Additionally, PPROM with intrauterine inflammation has been associated with an increased risk of neurodevelopmental impairment.⁴ The development of chorioamnionitis is an indication for prompt delivery to minimize fetal morbidity. Other indications for delivery include, but are not limited to, non-reassuring fetal status and placental abruption.

The most commonly identified risk factor for PPROM is infection.¹ Reviews of several large studies demonstrated bacteria within amniotic fluid in one-third of cases. The placental membranes are composed of an outer layer, the chorion, and the inner amnion. The amnion provides almost all of the tensile strength to the membranes, composed primarily of collagen types I and III. For this reason, collagen break down has been linked to rupture of membranes. Matrix metalloproteinases (MMP) are a family of enzymes involved in collagen break down. Studies of the amnion-chorion have demonstrated that MMP expression is increased with the inflammatory mediators IL-1, IL-6, TNF alpha. Similarly, other recent studies have demonstrated that bacteria endotoxin directly elicits release of fetal fibronectin (FFN) from the amnion. FFN activates a signaling cascade that leads to the synthesis of prostaglandins and increased activity of MMP.²³

There are multiple pathways in which bacteria and inflammatory mediators gain access to the uterine cavity. The most commonly recognized pathway is ascending infection from the vagina, through the cervix, to the uterine cavity. This is based on the fact the most commonly identified organisms in chorioamnionitis are also found in the vagina. Other potential pathways of spread include hematogenous dissemination. As in the case of bacteremia, organisms gain access via the placenta. Organisms from the abdominal cavity may enter the fallopian tubes and seed the uterus via retrograde spread. Systemic illness and local inflammation has also been linked to preterm delivery, including pneumonia, urinary tract infections and pyelonephritis.⁴ Additionally, well documented evidence exists for the association of periodontal disease and increased risk of preterm birth. The association is not clear, but evidence suggests a variation in the inflammatory response in the oral cavity alters genital tract flora and systemic inflammatory mediators.²³ This further illustrates that infectious source remote from the genital tract is able to initiate cascade of events ultimately culminating in preterm delivery.

The life time risk of developing appendicitis is 6.7% for females. The pathophysiology of appendicitis is incompletely understood, but thought to be due to luminal obstruction, secondary to fecoliths or hypertrophy of lymphoid tissue. This closed-loop obstruction leads to multiplication of the resident bacteria, leading to dispersion and inflammation that eventually spreads to the serosal surface. The initial distension of the appendix is responsible for the dull umbilical/epigastric pain and associated nausea. As the inflammation progresses to involve the serosa, the surrounding peritoneum becomes inflamed, producing the characteristic migration of pain to the right lower quadrant. Additional signs and symptoms of appendicitis include fever and leukocytosis.⁵ Acute appendicitis involves approximately 1/1700 pregnancies. In a large study conducted over a nine-year period involving 66,993 patients, the most common presentation of appendicitis in pregnancy mirrored that of the non-pregnant population. Despite the theory of appendiceal displacement by the gravid uterus, these investigators found that the most common location of pain regardless of trimester was found to be right lower quadrant, with associated nausea, fever, and leukocytosis.⁶

Can intra-abdominal infections cause chorioamnionitis and PPROM? At 20 weeks’ gestation, the average fundal height of the uterus is at the level of the umbilicus. Even at this early gestational age, the uterus spans well outside of the pelvis and lies in contact with other intraabdominal structures, most notably the bowel. As previously discussed, the connection between inflammatory mediators and rupture of membranes has been well documented. Based on knowledge of the inflammatory process and pathology involved in the development of chorioamnionitis, the presence of acute appendicitis and the associated local inflammatory response, could have spread to involve the nearby gravid uterus. Inflammation of the uterine serosa, eventually spreading to involve the myometrium, endometrium and placental membranes is one theory. Our patient’s pathology was significant for confirmation of acute appendicitis, chorioamnionitis and funisitis. Interestingly, cultures from the maternal and fetal placenta surfaces did not show bacterial growth, indicating acute inflammation without clear bacterial infection.
Would suspecting appendicitis have altered the management of our patient? As discussed previously, in the event of intra-amniotic infection, delivery is indicated to decrease neonatal mortality, regardless of the source of the initial infection. The symptoms of chorioamnionitis are markedly similar to many other intra-abdominal infections. Fever, abdominal pain, and tachycardia are generalized symptoms that apply to many disease states. The traditional teaching has been this: PPROM plus fever and/or abdominal pain, equals chorioamnionitis. Although multiple studies have demonstrated the safety of laparoscopic appendectomy in pregnancy, none have examined non-obstetric surgical interventions in the face of PPROM. We cannot definitely say whether an underlying early appendicitis was the sentinel event leading to PPROM, or the later development of chorioamnionitis. This case serves to remind us that inflammation, from any organ system, can lead to the pathological cascade causing PPROM, chorioamnionitis and subsequent preterm delivery. Suspicion of chorioamnionitis should prompt delivery for fetal benefit, but the differential diagnosis of potential other disease processes should remain for appropriate maternal management.

REFERENCES:


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A thin 65-year-old African American female with a past medical history of hypertension presented to her primary care physician with a chief complaint of bilateral “eye bumps”. She stated that they were present for years and only decided to come to the office after her relatives insisted it be evaluated. The patient did not endorse any other ocular problems including decreased vision, photophobia, pain, irritation or tearing. She denied any change in size, color or shape of the bumps over the years. She had no past history of other ocular lesions, no known allergies or drug use. The patient did not present with any other notable craniofacial abnormalities including auricular appendages, fistulas, cleft palate or postural dysfunction. Review of systems was otherwise unremarkable.

Physical exam revealed the presence of bilateral, symmetric, 1cm in diameter, pinkish-yellow, circular masses superior-temporally to the lateral canthus of the eyes. Lifting of the eyelid superiorly further elucidated the size of the mass to be closer to 3cm in diameter. Upon palpation, the mass was soft, non-mobile and could not be indented with a cotton-tip applicator. The mass appeared to be lying on the lateral bulbar conjunctiva. Extraocular muscle motility was within normal limits. Pupils were round, equal, reactive, and with no afferent pupillary defect. A funduscopic examination revealed an unremarkable optic disc without papilledema or signs of neovascularization.

**QUESTION:**

What is the diagnosis?

A. Dermolipoma
B. Orbital fat prolapse
C. Pinguecula
D. Pterygium
E. Squamous Cell Carcinoma
**ANSWER**

**What is the diagnosis?**

The correct answer is: A) Dermolipoma

**DISCUSSION**

An orbital dermolipoma is a benign congenital tumor of the bulbar conjunctiva. It can be more broadly described as a type of dermoid cyst, or choristoma which a group of normal cells in an abnormal location in the body. Histologically composed of adipose tissue surrounded by an outer connective tissue covering, these typically small tumors often go undetected until later in life due to their asymptomatic nature. Their incidence is noted to be rare (less than one in 10,000 live births) and they have an overall female predominance.

The classic description on physical exam, consistent with the patient noted on page 45, includes a well demarcated, pinkish-yellow, gelatinous appearing immobile mass in the superior temporal region of the bulbar conjunctiva. These lesions are almost always found laterally and frequently unilaterally; however, case reports have noted rare medial presence of dermolipomas.

Orbital fat prolapse is often noted to be the closest mimicker of dermolipomas given that both of these conditions typically present as soft yellowish masses in the superotemporal bulbar conjunctiva. However, a closer examination reveals key differentiating features between the two conditions. First, demographically, orbital fat prolapse has a propensity to occur in older (mean age 65-72) obese, males, unlike dermolipomas which classically is first noticed in thinner (mean age 22.5) females. Next, orbital fat prolapse’s convex, freely mobile anterior margin can be easily reduced back into the orbit which sharply contrasts the concave, immobile, non-reducible anterior margin of dermolipomas. Finally, upon magnification of the margin’s surface, superficial blood vessels are more often seen in the orbital fat prolapse while fine hairs can be appreciated on the outer layer of dermolipomas.

Squamous cell carcinoma can also appear as a gelatinous unilateral conjunctival mass but is typically more central in location, near the limbus of the eye, with pronounced red blood vessels visible on a pinkish-white base. It can grow very fast, over a period of months, due to close proximity to vital structures of the eye (lacrimal gland, lateral rectus muscle, conjunctiva) that often outweigh the benefits.

While these lesions are benign, they can be associated with a constellation of other symptoms portending an overall worse prognosis. One of the most well-known examples of this is Goldenhar-Gorlin syndrome, a rare syndrome, estimated to be seen in 1 out every 3,500 to 5,600 live births, characterized by ocular anomalies, auricular appendages, and vertebral anomalies. Additional associated abnormalities in this disorder may also include renal or facial hypoplasia, as well as cardiac defects (example ventral septal defects) which occur in 5 to 58 percent of all cases.

Ophthalmologic anomalies occur in about 50 percent of Goldenhar-Gorlin syndrome cases, one of the most common of which is a dermolipoma. The dermolipomas seen in this condition however, often are quite pronounced, located in the infratemporal quadrant and are most often unilateral. The collection of symptoms required for diagnosis are noticeable at birth but because there is such a wide range of overlapping anomalies the diagnosis may be missed. When Goldenhar-Gorlin syndrome is suspected, an echocardiogram and CT imaging can be used to confirm or detect the additional internal defects noted above that can be associated with the condition. The treatment of this disease varies with age and systemic associations but typically involves cosmetic reconstruction based on clinical presentation after the age of five.

This patient was completely asymptomatic, with no other associated findings and satisfied with the reassurance of the lesions benign nature. She will continue to monitor the dermolipoma as she had for the past 60 years.

**REFERENCES:**

### CALENDAR OF EVENTS

#### 2017

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<td>ACOFP Future Leaders Conference</td>
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<td>California ACOFP Annual Scientific Medical Seminar</td>
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Respiratory tract infections are any infection that affect the nose, sinuses, and throat (i.e. the upper respiratory tract) or airways and lungs (i.e. the lower respiratory tract). Viruses are the main cause of the infections, but bacteria can cause some. You can spread the infection to others through the air when you sneeze or cough. You can also spread the infection by indirect contact, for example, by rubbing your nose or eyes before touching a surface that another person may then touch. Common symptoms of an upper respiratory tract infection may include a cough, mild fever, headaches, a runny or stuffy nose, sore throat, sneezing, body aches, and fatigue. Whereas, common symptoms of a lower respiratory tract infection may include a severe cough with phlegm and mucus, difficulty in breathing, wheezing, a tight feeling in your chest, weakness, fever, and fatigue.

HOME MANAGEMENT INCLUDES:

- Drinking plenty of clear fluids and rest. Vitamin-C may help boost your immune system. Over-the-counter pain relievers such as acetaminophen and ibuprofen can be helpful for fevers and to ease any aches. Saline (salt) nose drops, lozenges, and vapor rubs can also help symptoms when used as directed by your physician.
- A cool mist humidifier can make breathing easier by thinning mucus.
- If you smoke, you should try to stop smoking for good! Avoid second-hand smoking also.
- In most cases, antibiotics are not recommended because they are only effective if bacteria caused the infection.
- Other treatments, that your Osteopathic Family Physician may prescribe, include Osteopathic Manipulative Therapy (OMT). OMT can help clear mucus, relieve congestion, improve breathing and enhance comfort, relaxation, and immune function.
- Generally, the symptoms of a respiratory tract infection usually pass within one to two weeks.
- To prevent spreading infections, sneeze into the arm of your shirt or in a tissue. Also, practice good hygiene such as regularly washing your hands with soap and warm water. Wipe down common surfaces, such as door knobs and faucet handles, with a disinfectant spray. Do not share cups or utensils.
- To avoid any possible complications of an acute respiratory tract infection, it is strongly recommended that very young children, older adults, and people with immune system disorders, heart disease and/or other chronic conditions such as lung problems who develop a respiratory infection visit their Family Physician.
- You can also be vaccinated against some respiratory tract infections, such as the flu and pneumonia.

MEDICAL CARE & TREATMENT OPTIONS:

If you have any questions about respiratory tract infections, please contact your Osteopathic Family Physician. Your physician can diagnose an upper or lower respiratory tract infection with a thorough history and physical exam along with any appropriate tests. Management includes the right treatment plan and any necessary follow-up with your doctor. Your family doctor will help you determine which current recommended treatment(s) will work best for you. In case of any emergency, you should call your doctor or 911 right away.

SOURCE(S): Centers for Disease Control & Prevention (CDC), Upper & Lower Respiratory Tract Infections. Gov, and Up-To-Date.

The Osteopathic Family Physician Patient Handout is a public service of the ACOFP. The information and recommendations appearing on this page are appropriate in many instances; however, they are not a substitute for medical diagnosis by a physician. For specific information concerning your personal medical condition, ACOFP suggests that you consult your family physician. This page may be photocopied noncommercially by physicians and other health care professionals to share with their patients.

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