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January / February, 2017

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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

EXAM SCHEDULE

CERTIFICATION & OCC (RECERTIFICATION)

EXAMS	LOCATIONS	POSTMARK DATE
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**AOA OMED Conference** **Philadelphia, PA** October 7 - 11, 2017 October 6 - 8, 2017	**April 1, 2017** *Late fee through June 1*
Family Medicine / OMT Certification / OCC Cognitive Exam	**Electronic Testing** Regional Sites October 21, 2017	**April 1, 2017** *Late fee through June 1*

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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.

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Articles submitted for publication must be original in nature and may not be published in any other periodical.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.)*

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VOLUME 9 | NUMBER 1

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OSTEOPATHIC FAMILY PHYSICIAN SPECIALTY PEER REVIEWERS

2017 STUDENT PEER REVIEW & WRITING INTERNS

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 apporpriate 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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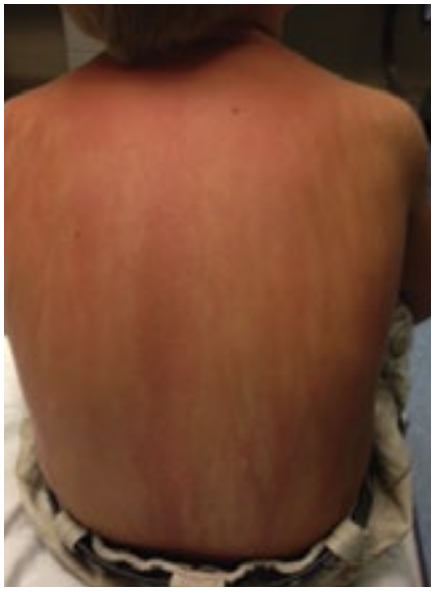

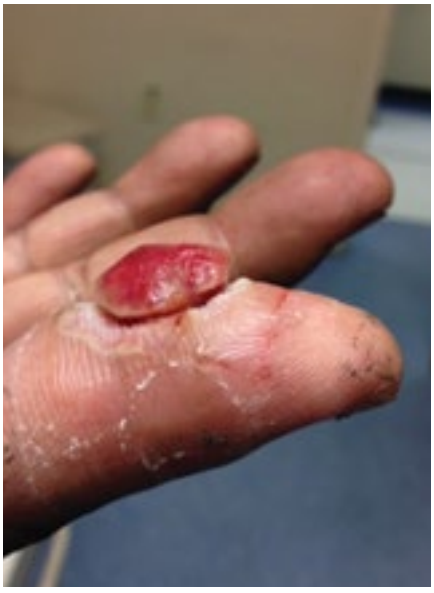

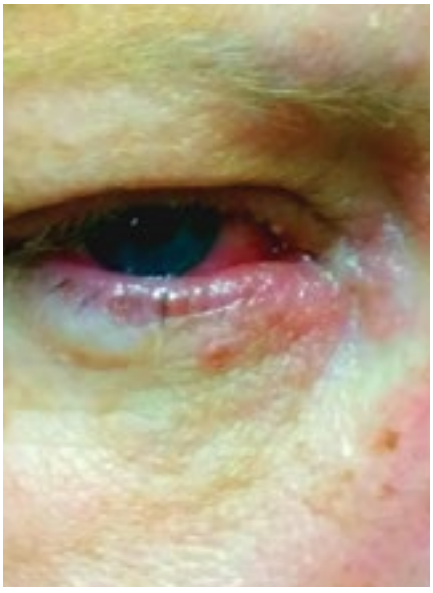

Osteopathic Family Physician

ACCEPTING SUBMISSIONS FOR THE SECTION TITLED "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.

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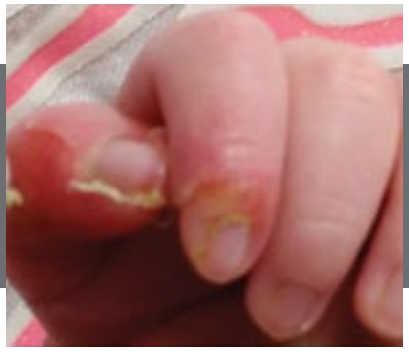

EDITOR'S MESSAGE

Winter Wonderland

Amy J. Keenum, DO, PharmD, Editor, Osteopathic Family Physician

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.

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[1]*A subset of Quality Markers measures qualify for PQRS and QCDR reporting.*
[2]*Provider 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 Readine$$, 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.[1] 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.
2. **Report quality for a continuous 90-day period** starting January 1, 2017 to October 1, 2017. Report on a minimum of one quality measure. You may receive a small incentive payment.
3. **Report quality for the entire calendar year.** Report on a minimum of one quality measure. You may receive a modest 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.[3] 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.[4]

TABLE 1:

The Four CMS Categories Used to Determine an EP's Composite Performance Score[2]

Measurements	2017 - Percentage of CMS Composite Performance Score	Possible Point Score[1]
Quality Reporting	60%	70 points
Resource Use	0% for 2017 - Benchmark Year	No points for 2017
Advancing Care Information (ACI) *Previously Meaningful Use*	25%	100 points
Clinical Practice Improvement Activities	15%	40 points

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:

1. McLaughlin, Jennifer JD. "Under the MACRAscope." MGMA webinar. November 2016.
2. www.cms.gov. Accessed on October 27 and November 27, 2016
3. www.cms.gov. Accessed October 27, 2016
4. www.ncqa.gov Accessed November 22, 2016
5. Quality Payment Program. www.cms.gov. October 14, 2016

*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.

ORIGINAL RESEARCH

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, DO[3]

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[3]New Hanover Regional Medical Center, Wilmington, North Carolina

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.

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 wrapping 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]

CORRESPONDENCE:
Samuel Madore, DO | samadore@mainegeneral.org

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 suffers. 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.[1,11,12,13] 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.[2,15] A study showed that 93% of patients

IMAGE 1:

This dissection shows the lesser and greater occipital nerves passing through myofascial structures.

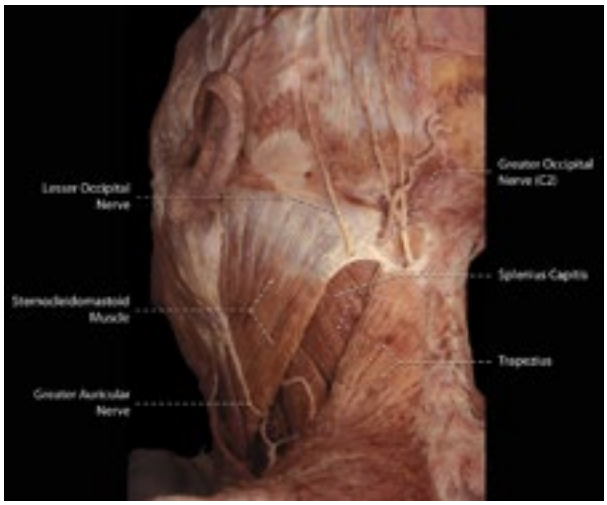

Photo courtesy of Dr. Frank Willard PhD.

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 palpated, 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-.75 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-.75 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.[1,19] 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.

TABLE 1:

80% of patients were female with an average age of forty-seven. All patients were Caucasian and lived in central Maine.

Week Benefit	Patients Who Received ONB	Patients Who Received ONB + TPI
0 Weeks	1pt (5.5%)	5 pts (9.4%)
<4 weeks	1 pt (5.5%	8 pts(15%)
4-12 weeks	15 pts (83%	34 pts (64%)
13-24 weeks	1 pt (5.5%)	6 pts (11%)
Median benefit	8 weeks	8 weeks
Mean benefit	8.6 weeks	9.1 weeks
Abbreviations	ONB-occipital nerve block	TPI- trigger point injection

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.[15,24]

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.[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-syncope, 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.[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.[27] In addition, the inclusion of placebo controls in future studies would help further validate this therapy.

In this study, patients who received less then 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.[1,9] 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.

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CONFLICT OF INTEREST STATEMENT

The Authors declares that there are no conflict of interest.

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REFERENCES:

1. Tobin J, Flitman S. Occipital nerve blocks: when and what to inject? Headache. 2009; 49(10): 1521-1533
2. Giamberardino MA, Tafuri E, Savini A, et al. Contribution of myofascial trigger points to migraine symptoms. J Pain. 2007; 8(11): 869-878
3. Tubbs RS, Watanabe K, Loukas M, et al. The Intramuscular course of the Greater Occipital Nerve : Novel Findings with Potential Implications for Operative Interventions and Occipital Neuralgia. Surg Neurol Int 2014;5:155
4. Dash KS, Janis JE, & Guyuron B. (2005). The lesser and third occipital nerves and migraine headaches. Plastic and reconstructive surgery, 115(6), 1752-1758
5. Simons DG, Travell JG, Simons LS. Travel and Simons' Myofascial pain and dysfunction: The Trigger Point Manual, ed 2, pg. 262, Baltimore, 1999, Williams and Wilkins.
6. Kuhn WF, Kuhn SC, Gilberstadt H. Occipital neuralgias: clinical recognition of a complicated headache. A case series and literature review. J Orofac Pain 1997; 11:158-165
7. Anthony M. Headache and the greater occipital nerve. Clin Neurol Neurosurg. 1992; 94 (4):297-301
8. Ashkenazi A, Young W. The effects of greater occipital nerve block and trigger point injection on brush allodynia and pain in migraine. Headache. 2005; 45(4);350-354
9. Afridi SK, Shields KG, Bhola R, et al. Greater occipital nerve injections in primary headache syndromes- Prolonged effects from a single injection. Headache 2006; 122:126-129
10. Dougherty C. Occipital Neuralgia. Curr Pain Headache Rep. 2014; 18: 411
11. Scattoni L, Di Stani F, Villani V, et al. Great occipital nerve blockade for cluster headache in the emergency department: case report. J Headache Pain. 2006; 7:98-100
12. Hecht J. Occipital Nerve Blocks in Postconcussive Headaches: a retrospective review and report of ten patients. J Head Trauma Rehabil. 2004; 19: 58-71
13. Leinisch-Dahlke E, Jurgens T, Bogdahn U, et al. Greater occipital nerve block is ineffective in chronic tension type headache. Cephalgia. 2005; 25: 704-708

14. Soma-Srivastava S, Zheng L. Occipital Neuralgia with and without Migraine: Difference in Pain Characteristics and Risk Factors Headache 2011 July ;51:124-128
15. Fernández‐de‐las‐Peñas C , Cuadrado ML, Pareja JA . Myofascial trigger points, neck mobility and forward head posture in unilateral migraine. Cephalalgia. 2006; 26 (9): 1061-1070
16. Calandre EP, Hidalgo J, Garcia-Leiva JM. Trigger point evaluation in migraine patients: an indication of peripheral sensitization linked to migraine predisposition? E J Neurology. 2006 ; 13(3): 244-9
17. Calandre E, Hidalgo J, Garcia-Leiva J, et al. Myofascial trigger points in cluster headache patients: a case series. Head Face Med. 2008;4: 32
18. Alvarez DJ, Rockwell PG. Trigger Points: Diagnosis and Management. Am Fam Physician. 2002;65(4): 653-661
19. Ashkenazi A, Levin M. Greater occipital nerve block or migraine and other headaches: Is it useful? Ashkenazi A, Levin M. Curr Pain Headache Rep. 2007; 11(3): 231-5
20. Keays A, Neher J, Safrenek S. Is osteopathic manipulation effective for headaches? The Journal of Family Practice 2008; 57(3):190-191
21. Fernádez-de-las-Peñas C, Cuadrado ML, Arendth-Nielsen L, et al. Myofascial trigger points and sensitization: an updated pain model for tension-type headache. Cephalgia. 2007 ; 27(5): 383-93
22. Xu YM, Ge HY, Arendt-Nielsen L. Sustained nociceptive mechanical stimulation of latent myofascial trigger point induces central sensitization in healthy subjects. J Pain. 2010:11(12);1348-55
23. Bendtsen L. Central sensitization in tension‐type headache—possible pathophysiological mechanisms. Cephalalgia. 2000; 20(5): 486-508.
24. Olesen J. Clinical and pathophysiological observations in migraine and tension-type headache explained by integration of vascular, supraspinal, and myofascial inputs. J Pain. 1991; 46(2): 125-132
25. Bartsch T, Goadsby P. Stimulation of the greater occipital nerve induces increased central excitability of dural afferent input. Brain. 2002;125:1496-1509
26. Lavin PJ, Workman R. Cushing syndrome induced by serial occipital nerve blocks containing corticosteroids. Headache. 2001; 41(9): 902-904
27. Ashkenazi A , Matro R, Shaw J W, et al. Greater occipital nerve block using local anesthetics alone or with triamcinolone for transformed migraine: a randomized comparative study. J Neurol Neurosurg Psychiatry. 2008; 79(4): 415-417.

REVIEW ARTICLE

Osteopathic Considerations in the Infections of the Respiratory Tract

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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.

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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 mucus 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

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TABLE 1:

Upper Respiratory Infections

Disease	Etiology	Common Symptoms	Common Physical Examination Findings	Considerations	Common Management
Pharyngitis[33, 34,35]	Viral & Bacterial (GAS)	Fever (>38ºC) Sore Throat Myalgia Headahe	Cervical LAD Pharyngeal Erythema Exudates	Respiratory Distress Poor Feeding Resistant to antibiotic therapy	Antimicrobial therapy if high bacterial suspicion
Allergic Rhinitis[36,37]	Viral	>2 sx: Sneezing Nasal pruritus Rhinorrhe Congestion > 1 hour for most days	Inflamed Nasal turbinates Associated with sinusitis, asthma, OM & conjunctivitis	Rule out non-allergic causes including drug induced, & inflammatory disorders, etc.	Nasal decongestants Intranasal steroids
Acute Sinusitis[38,39,40]	Viral with possible secondary bacterial	Nasal obstruction & nasal secretions < 10 days	Sinus swelling Rhinorrhea	**IN THE NEWBORN:** poor feeding & focal signs of sinus involvement	**IN NEWBORNS:** Antibacterial therapy covers S. aureus, GAS & GBS
Rhinosinusitis[41,42]	Viral with possible secondary bacterial	**ACUTE:** > 3 times/ year, with > 2 sx: mucopurulent (not clear) drainage. Nasal obstruction, Facial Pain, & Anosmia **CHRONIC:** sx> 12 weeks	**ACUTE & CHRONIC:** Purulent nasal discharge **CHRONIC:** With or without nasal polyps seen on rhinoscopic exam or sinus CT scan.	Associated with asthma, GERD, OM, immunodeficiencies, defects in mucociliary clearance (CF or PCD)	**CRS:** Antibiotics are controversial, with potential use of a 10-14 day course with or without oral steroids.
Epiglottitis[43, 44,45]	*H.influenza*, *Streptococcus spp.*, *Virall*	Fever (>38□C), sore throat, hoarseness, dyspnea, inspiratory stridor, with a "hot potato" or muffled voice	Unique posture of the head & neck. Gross appearance of the pharynx may appear normal	Posture & Stridor, Unstable vital signs & distress **ADULTS**: stridor not as frequently seen	BSA & steroids; Emergency intervention when necessary
Laryngitis[46,47]	Irritants Viral	Hoarseness & Aphonia ~3 - 4 days duration	Benign examination	If URT, consider alternative diagnosis	Voice Rest
Croup[48,49,50]	Viral (MC Parainfuenza) with possible secondary bacterial	**PRODROME:** URT sx 12 - 48 hours before "barking" cough with inspiratory stridor & hoarseness	**RADIOGRAPH:** AP neck film with "steeple" or "hourglass" sign Westley Score	Rapid course, Drooling & High fever may be present	Conservative Management; Emergent intervention when necessary
Otitis Media	*S.pneumonia*, *H.influenzae*, *M.catarrhalis*	< 3 years old are most susceptible: Fever, otalgia & impaired hearing	Fluid accumulation in the middle ear & erythema of the TM	Unvaccinated children Signs of pharyngeal irritation Recurrent & persistent episodes	Antimicrobial Therapy

ABBREVIATIONS: *MC: most common, GAS: Group A Streptococcus, Sx: symptoms, LAD: Lymphadenopathy, GERD: Gastroesophageal reflux disease, OM: Otitis Media, BSA: Broad Spectrum Antibacterials; CRS: Chronic Rhinosinusitis, GBS: Group B Streptococcus, URT: Upper Respiratory Tract, AP: Anterior-Posterior, CF: Cystic Fibrosis, PCD: Primary Ciliary Dyskinesia, SX: symptoms, TM: tympanic membrane*

TABLE 2:

Lower Respiratory Infections

Disease	Etiology	Common Symptoms	Common Physical Examination Findings	Considerations	Common Management
Acute Bronchitis[51,52]	Viral (Influenza & RSV) Bacteria (*Streptococcus spp*, Atypical Bacteria)	Cough +/- sputum 1-3 weeks	Upper & Lower Respiratory signs without crackles	*Hospitalizations Comorbidities* Vomiting & > 4 weeks duration: Consider B.*pertussis*	Cough suppressants, nasal decongestants, expectorants, beta agonists, antihistamines, & Abx therapy.
Bronchiolitis [53,54,55,56,57,58,59,60,61]	Viral MC is RSV	< 2 yrs old, MC within 1st year Wheezing, Fever, Cough, Rhinorrhea	Decreased lung sounds with crackles Dyspnea Chest retractions	Prematurity, Lower cord blood antibody titers to RSV, lower SES, smoke exposure.	Conservative management Consider Abx if bacterial superinfection suspected
Pneumonia [6,7,62,,63,64,65,66,67,68,69 70,71,72,73]	BACTERIA: *S. pneumonia, S. aureus, H.influenzae* VIRAL (CHILDREN): *RSV, Parainfluenza, Influenza* VIRAL (ADULTS): *Influenza & RSV*	Fever & Chills, Pleuritic chest pain, Productive cough with purulent sputum	Leukopenia Tachypnea Tachycardia Crackles Signs of consolidation Sputum: thick & purulent, possibly rust colored	Older age; Unvaccinated; Comorbidities	Beta-lactam plus a macrolide or fluroquinolone therapy

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.[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.[1] Antibiotic overuse may lead to resistance, increased costs, and increased adverse effects: thus, it is important to differentiate between bacterial and viral etiologies.[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.[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.[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.[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.[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.[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.[17] Table 3 *(pages 21 and 23)* summarizes osteopathic manipulative treatments by region that can be useful in the treatment of apatient 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.[18] Such increased respiratory effort overwhelms 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 atthe associated vertebral levels to help relax and encourage normal thoracic diaphragm motion.[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.[4] Therefore, dysfunctions of the cranial base and facial bones can affect the upper respiratory tract.[4] More specifically, dysfunctions affecting the vagus nerve can affect parasympathetic tone and influence pharyngeal motor activity.4Retro-orbital and retro-auricular pain may be produced by anterior atlas dysfunctions in patients with sinusitis or congestive symptoms.[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.[4]

RESPIRATORY-CIRCULATORY MODEL & METABOLIC-ENERGY MODEL CONSIDERATIONS

The right side of the head and neck and portions of the lung drain into the right lymphatic duct, while the left side of the head and neck, and portions of the lung drain into the left lymphatic duct or thoracic duct.[19] The nose, sinuses, and pharynx, typically drain into the submandibular and retropharyngeal nodes, ultimately draining into cervical lymph nodes.[4,19] Somatic dysfunctions in the head, precervical muscles, neck, and lung can impede proper tissue activity and metabolism.[19] Respiratory infections result in the recruitment of secondary muscles of respiration, leading to increased work of

TABLE 3:

Osteopathic Manipulative Techniques (OMT) to address respiratory disease organized by body region

	Techniques	Potential Treatment Effects
Head	Balanced membranous tension	• Treats cranial strain patterns • Decreases dural strainrestrictions
	Sinus drainage technique Cranial bone lifts & effleurage	• Facilitates movement of facial bones • Improves sinus drainage • Decreases facial pain
	Galbreath technique (Mandibular lift)	• Improves Eustachian tube drainage • Decompresses the auditory canal • Decreases auricular pain
	Sphenopalatine ganglia inhibition	• Normalizes parasympathetic tone to nasal mucosa and sinuses • Regulates blood flow to nasal conchae and encourages thinner mucosal secretions • Decreases headache and facial discomfort
	Venous sinus drainage	• Improves venous and gylmphatic flow of the brain • Decreases dural strain • Decreases headache
	Occipito-atlantal decompression Suboccipital release	• Decreases muscle spasms and restores upper cervical mobility • Frees the passage of the vagus nerve, normalizing parasympathetic tone
Neck & Cervical Spine	Soft tissue & myofascial techniques addressing secondary muscles of inspiration	• Relaxes the Sternocleidomastoid and scalene muscles to aid in the drainage of the superficial and deep cervical lymph nodes • Allows improved respiration by relaxing the attachments to the manubrium and clavicle
	Direct techniques (MET, articulatory, HVLA) to cervical spine dysfunctions	• Improves somatic dysfunctions in the cervical spine allowing increased range of motion of the neck • Regulates neural influence over the trigeminal nucleus[4] • Treatment of C3-C5 can affect diaphragmatic innervation • Removes mediastinal fascial restrictions freeing the pathway of the vagus nerves
	Indirect techniques (CS,FPR,BLT) to cervical spine dysfunctions	• CS alleviates acute tenderpoints • Treatment of upper cervical region balances autonomics • 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.
Lumbar	Direct/indirect treatment of the lumbar spine (FPR or BLT of L2-L3)	• Addresses restrictions at diaphragmatic attachments on the lumbar spine at L1-L3[19] • Encourages respiratory diaphragm motion • Improves lymphatic drainage (cisterna chyli lies anterior to the lumbar spine)
	Direct/indirect treatment of the psoas and QL	• Addresses myofascial restrictions at the attachments of the diaphragm • Psoas restrictions affect the upper lumbar spine and cross into the pelvis and lower extremities
Pelvis & Sacrum	Direct/indirect treatments of the sacrum and pelvis (sacral rock, pelvic SD treatment with MET)	• Improves biomechanical restrictions of the sacrum to allow for proper motion of the spine with respiration • Improves motion of the pelvic diaphragm, allowing for the descent of the abdominal diaphragm with inhalation • Balances autonomic innervation affecting overall autonomic tone
Extremities	Direct/indirect treatment of the scapula and upper extremity SD	• Improves head, neck, and thoracic cage mobility due to the musculoskeletal attachments of 17 muscles from the scapula to the regions mentioned • Improves upper extremity restrictions that can decrease thoracic lymph drainage
	Direct/indirect treatment of the hip and lower extremity SD	• Improves diaphragmtic motion through the psoas and additional musculoskeletal attachments from the LE into the pelvis and sacrum

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.[4] Proper thoracic cage compliance is vital to proper lymphatic drainage.[19] 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.[4] 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.[4] Venous and lymphatic drainage from the head and neck are also disrupted by these dysfunctions, leading to decreased clearance of microorganisms.[4]

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.[19] 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.[19] 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.[20]

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,[4] 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.[19] 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.[4] OMT can normalize this balance, reducing the duration and intensity of symptoms and enhancing the efficacy of other therapies.[4] 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.[19]

Somatic dysfunctions in the upper thoracic spine causing facilitation of those spinal segments can increase the activity of the sympathetic nervous system.[4] An increase in sympathetic tone leads to an increase in airway epithelial hyperplasia which leads to increased goblet cells and increased luminal secretions.[19] In order to normalize sympathetic tone in patients with respiratory infections, consider treating the sympathetic chain between T1-T7.[19] 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 bronchiole constriction.[21] Head complaints involving nasal sinuses and lacrimation can be treated with COMM as is seen in Table 3 *(page22)*. 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.[4] Thus, muscles innervated by the trigeminal nerve may be subject to viscerosomatic reflexes.[4] 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.[4] 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.[4]

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.[22, 23,24,25,26,27] 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.[28]

The severity and chronicity of respiratory infections correlates with impairments in well-being, quality of life, and sleep.[23] As upper respiratory infections and chronic diseasessuch as asthma and Chronic Obstructive Pulmonary Disease (COPD) are correlated, these comorbid conditions correspond with an increase in anxiety and depression.[25,29] 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.[30]

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.[31] 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.[31, 32,20] 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.[20]

TABLE 3 (CON'T):

Osteopathic Manipulative Techniques (OMT) to address respiratory disease organized by body region

	Techniques	Potential Treatment Effects
Thorax	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[19] • Improves upper rib motion
	Lymphatic pumps	• Augments lymphatic drainage of lungs[19] • 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: *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*

TABLE 4:

Pertinent Chapman's Reflex Points for Respiratory Infections[4,74]

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

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:

1. Prevention C. Respiratory Infections - Chapter 2 - 2016 Yellow Book | Travelers' Health | CDC. Www.ccdc.gov. 2016. Available at: http://wwwnc.cdc.gov/travel/yellowbook/2016/the-pre-travel-consultation/respiratory-infections. Accessed April 5, 2016.
2. Denny F. The Clinical Impact of Human Respiratory Virus Infections. Am J Respir Crit Care Med. 1995;152(4_pt_2):S4-S12. doi:10.1164/ajrccm/152.4_pt_2.s4.
3. Dasaraju PV, Liu C. Infections of the Respiratory System. In: Baron S, editor. Medical Microbiology. 4th edition. Galveston (TX): University of Texas Medical Branch at Galveston; 1996. Chapter 93. Available from: http://www.ncbi.nlm.nih.gov/books/NBK8142/ Accessed April 5, 2016.
4. Nelson KE and Glonek T. Somatic Dysfunctions in Osteopathic Manipulative Medicine. 2nd Edition. Wolters Kluwer Health,2015.
5. Levitzky MG. Chapter 1. Function and Structure of the Respiratory System. In: Levitzky MG. eds. Pulmonary Physiology, 8e . New York, NY: McGraw-Hill; 2013. http://accessmedicine.mhmedical.com.arktos.nyit.edu/content.aspx?bookid=575&Sectionid=42512979. Accessed March 14, 2016.
6. Lee GE, Lorch SA, Sheffler-Collins S, Kronman MP, Shah SS. National hospitalization trends for pediatric pneumonia and associated complications. Pediatrics 2010;126:204-13.
7. Pfuntner A, Wier LM, Steiner C. Costs for hospital stays in the United States, 2011. HCUP statistical brief #168. Rockville, MD: Agency for Healthcare Research and Quality, December 2013 (http://www.hcup-us.ahrq.gov/reports/statbriefs/sb168 -Hospital-Costs-United-States-2011.pdf)
8. Stover C, Litwin C. The Epidemiology of Upper Respiratory Infections at a Tertiary Care Center: Prevalence, Seasonality, and Clinical Symptoms. Journal of Respiratory Medicine. 2014;2014:1-8. doi:10.1155/2014/469393.
9. Weekly U.S. Influenza Surveillance Report| Seasonal Influenza (Flu) | CDC. Cdcgov. 2016. Available at: http://www.cdc.gov/flu/weekly/#S4. Accessed April 5, 2016.
10. Boloursaz M, Lotfian F, Aghahosseini F et al. Epidemiology of Lower Respiratory Tract Infections in Children. Journal of Comprehensive Pediatrics. 2013;3(3):93-8. doi:10.5812/jcp.10273.
11. Ortqvist A. Treatment of community-acquired lower respiratory tract infections in adults. European Respiratory Journal. 2002;20(Supplement 36):40S-53s. doi:10.1183/09031936.02.00309002.
12. Solomon C, Wunderink R, Waterer G. Community-Acquired Pneumonia. New England Journal of Medicine. 2014;370(6):543-551. doi:10.1056/nejmcp1214869.
13. Rovery C. Use of antibiotics in common respiratory infections. In: Cohen J, Opal S, Powdely W, ed. Infectious Disease. 3rd ed. Elsevier Limited; 2010:344-346. Available at: https://www.clinicalkey.com.arktos.nyit.edu/#!/content/book/3-s2.0-B9780323045797002318. Accessed May 7, 2016.
14. McKay R, Mah A, Law M, McGrail K, Patrick D. Systematic Review of Factors Associated with Antibiotic Prescribing for Respiratory Tract Infections. Antimicrobial Agents and Chemotherapy. 2016:AAC.00209-16. doi:10.1128/aac.00209-16.
15. Zoorob R, Sidani M, Fremont R, Kihlberg C. Antibiotic Use in Acute Upper Respiratory Tract Infections. American Family Physician. 2012;86(9):817-822. Available at: https://www.clinicalkey.com.arktos.nyit.edu/#!/content/journal/1-s2.0-S0002838X12603748. Accessed May 7, 2016.
16. Seffinger MA, King HH, Ward RC, et al. Osteopathic Philosophy. In: Chila AG, ed. Foundations of Osteopathic Medicine. 3rd ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2011:3-22
17. Earley B, Luce H. An Introduction to Clinical Research in Osteopathic Medicine. Primary Care: Clinics in Office Practice. 2010;37(1):49-64. doi:10.1016/j.pop.2009.09.001.
18. Hadjikoutis, S., C.m. Wiles, and R. Eccles. "Cough in Motor Neuron Disease: A Review of Mechanisms." Qjm 92.9 (1999): 487-94. Web.
19. Ward R. Foundations For Osteopathic Medicine. Philadelphia: Lippincott Williams & Wilkins; 2003.
20. Hodge L, Creasy C, Carter K, Orlowski A, Schander A, King H. Lymphatic Pump Treatment as an Adjunct to Antibiotics for Pneumonia in a Rat Model. The Journal of the American Osteopathic Association. 2015;115(5):306. doi:10.7556/jaoa.2015.061.
21. Mitchell Robbins S. Pocket Companion To Robbins &Cotran Pathologic Basis Of Disease, Eighth Edition. Philadelphia, Pa.: Elsevier Saunders; 2011.
22. Chung JH, Lee YJ, Kang TW, et al. Altered Quality of Life and Psychological Health (SCL-90-R) in Patients With Chronic Rhinosinusitis With Nasal Polyps. Ann OtolRhinolLaryngol. 2015;124(8):663-70.
23. Juniper EF, Guyatt GH, Dolovich J. Assessment of quality of life in adolescents with allergic rhinoconjunctivitis: development and testing of a questionnaire for clinical trials. J Allergy ClinImmunol. 1994; 93:413-423.
24. Bousquet J, Bullinger M, Fayol C, Marquis P, Valentin B, Burtin B. Assessment of quality of life in patients with perennial allergic rhinitis with the French version of the SF-36 Health Status Questionnaire. J Allergy ClinImmunol. 1994; 94:182-188.
25. Katotomichelakis M, Simopoulos E, Zhang N, et al. Olfactory dysfunction and asthma as risk factors for poor quality of life in upper airway diseases. Am J Rhinol Allergy. 2013;27(4):293-8.
26. Fu QL, Ma JX, Ou CQ, et al. Influence of self-reported chronic rhinosinusitis on health-related quality of life: a population-based survey. PLoS ONE. 2015;10(5):e0126881.
27. Dalager-pedersen M, Thomsen RW, Schønheyder HC, Nielsen H. Functional status and quality of life after community-acquired bacteraemia: a matched cohort study. ClinMicrobiol Infect. 2016;22(1):78.e1-8.
28. Bramley TJ, Lerner D, Sames M. Productivity losses related to the common cold. J Occup Environ Med. 2002;44(9):822-9.
29. Belfer MH, Reardon JZ. Improving Exercise Tolerance and Quality of Life in Patients With Chronic Obstructive Pulmonary Disease. J Am Osteopath Assoc 2009;109(5):268-278
30. Zanotti E, Berardinelli P, Bizzarri C, et al. Osteopathic manipulative treatment effectiveness in severe chronic obstructive pulmonary disease: a pilot study. Complement Ther Med. 2012;20(1-2):16-22.
31. Hodge L. Osteopathic lymphatic pump techniques to enhance immunity and treat pneumonia. International Journal of Osteopathic Medicine. 2012;15(1):13-21. doi:10.1016/j.ijosm.2011.11.004.
32. Noll D, Degenhardt B, Morley T et al. Efficacy of osteopathic manipulation as an adjunctive treatment for hospitalized patients with pneumonia: a randomized controlled trial. Osteopathic Medicine and Primary Care. 2010;4(1):2. doi:10.1186/1750-4732-4-2.

33. Harris AM, Hicks LA, Qaseem A. Appropriate Antibiotic Use for Acute Respiratory Tract Infection in Adults: Advice for High-Value Care From the American College of Physicians and the Centers for Disease Control and Prevention. Ann Intern Med. 2016;164(6):425-34.

34. Gerber MA, Shulman ST. Rapid diagnosis of pharyngitis caused by group A streptococci. ClinMicrobiol Rev. 2004;17(3):571-80

35. Cunningham MW. Pathogenesis of group A streptococcal infections and their sequelae. AdvExp Med Biol. 2008;609:29-42.

36. Greiner A, Hellings P, Rotiroti G, Scadding G. Allergic rhinitis. The Lancet. 2011;378(9809):2112-2122. doi:10.1016/s0140-6736(11)60130-x.

37. Murray CS, Simpson A, Custovic A. Allergens, viruses, and asthma exacerbations. Proc Am ThoracSoc2004; 1: 99–104.

38. Dykewicz MS, Hamilos DL. Rhinitis and sinusitis. J Allergy ClinImmunol. 2010;125(2 Suppl 2):S103-15.

39. Kaliner MA, Osguthorpe JD, Fireman P, et al. Sinusitis: bench to bedside. Current findings, future directions. J Allergy ClinImmunol. 1997;99(6 Pt 3):S829-48.

40. Rudmik L, Soler ZM. Medical Therapies for Adult Chronic Sinusitis: A Systematic Review. JAMA. 2015;314(9):926-39.

41. Bachert C, Holtappels G. Pathophysiology of chronic rhinosinusitis, pharmaceutical therapy options. GMS Curr Top Otorhinolaryngol Head Neck Surg. 2015;14:Doc09.

42. Peters AT, Spector S, Hsu J, et al. Diagnosis and management of rhinosinusitis: a practice parameter update. Ann Allergy Asthma Immunol. 2014;113(4):347-85.

43. R.K.Shah, D.W.Roberson, D.T.Jones, Epiglottitis in the Hemophilusinfluenzae type B vaccine era: changing trends, Laryncoscope 114 (3) (2004) 557–560.

44. C.C. Chiou, N.L. Seibel, F.A. Derito, D. Bulas, T.J. Walsh, A.H. Groll, Concomitant candida epiglottitis and disseminated varicella zoster virus associated with acute lymphoblastic leukemia, J. Pediatr. Hematol. Oncol. 28 (11) (2006) 757–759.

45. J.L. Goldhagen, Supraglottitis in three young infants, Pediatr. Emerg. Care 5 (3) (1989) 175–177.

46. Klassen TP. Recent advances in the treatment of bronchiolitis and laryngitis. PediatrClin North Am. 1997;44(1):249-61.

47. Dworkin JP. Laryngitis: types, causes, and treatments. OtolaryngolClin North Am. 2008;41(2):419-36, ix.

48. RosychukRJ,KlassenTP,MetesD,etal.Crouppresentations to emergency departments in Alberta, Canada: a large population-based study. PediatrPulmonol. 2010;45:83-91.

49. WeinbergGA,HallCB,IwaneMK,etal. Parainfluzenzainfection of young children: estimates of the population- based burden of hospitalization. J Pediatr. 2009;154: 694-699

50. Russell KF, Liang Y, O'Gorman K, et al. Glucocorticoids for croup. Cochrane Database Syst Rev. 2011;(1):CD001955.

51. Hament J, Kimpen JL, Fleer A, Wolfs TF. Respiratory viral infection predisposing for bacterial disease: a concise review. FEMS Immunol Med Microbiol. 1999;26:189-95. [PMID: 0010575129]

52. Gonzales R, Anderer T, McCulloch CE, et al. Less is more: a cluster randomized trial of decision support strategies for reducing antibiotic use in acute bronchitis. JAMA Intern Med. 2013;173:267-273.

53. Bush A, Thomson A. Acute bronchiolitis. Br Med J. 2007; 335:1037-1041.

54. Thorburn K, Harigopal S, Reddy V, Taylor N, van Saene H. High incidence of pulmonary bacterial co-infection in children with severe respiratory syncytial virus bronchiolitis. Thorax 2006;61(7):611–5.

55. Kneyber M, van Oud-Alblas H, van Vliet M, Uiterwaal C, Kimpen J, van Vught A. Concurrent bacterial infection and prolonged mechanical ventilation in infants with respiratory syncytial virus lower respiratory tract disease. Intensive Care Medicine 2005;31:680–5.

56. Christakis D, Cowan C, Garrison M, Molteni R, Marcuse E, Zerr D. Variation in inpatient diagnostic testing and management of bronchiolitis. Pediatrics 2005;115:878–84.

57. Kabir M, Haq N, Hoqu M, Ahmed F, Amin R, Hossain A, et al.Evaluation of hospitalised infants and young children with bronchiolitis - a multi-centre study. Mymensingh Medical Journal 2003;12(2):128–33.

58. Vogel AM, Lennon DR, Harding JE, Pinnock RE, Graham DA, Grimwood K, et al.Variations in bronchiolitis management between five New Zealand hospitals: can we do better?. Journal of Pediatric Child Health 2003;39:40–5

59. Bordley WC, Viswanathan M, King VJ, Sutton SF, Jackman AM, Sterling L, et al.Diagnosis and testing in bronchiolitis: a systematic review. Archives of Pediatrics & Adolescent Medicine 2004;158:119–26.

60. Lozano J, Wang E. Bronchiolitis. Clinical Evidence 2002;8: 291–303.

61. Williams JV, Harris PA, Tollefson SJ, Halburnt-Rush LL, Pingsterhaus JM, Edwards KM, et al.Humanmetapneumovirus and lower respiratory tract disease in otherwise healthy infants and children. New England Journal of Medicine 2004;350:443–50.

62. Bochud PY, Moser F, Erard P, et al: Community-acquired pneumonia. A prospective outpatient study, Medicine (Baltimore) 80:75–87, 2001.

63. File TM Jr, Marrie TJ: Burden of community-acquired pneumonia in North American adults, Postgrad Med 122:130–141, 2010.

64. Christ-Crain M, Opal SM: Clinical review: the role of biomarkers in the diagnosis and management of community-acquired pneumonia, Crit Care 14:203, 2010.

65. Lim WS, Baudouin SV, George RC, et al: BTS guidelines for the management of community acquired pneumonia in adults: update 2009, Thorax 64(Suppl 3):iii1–iii55, 2009.

66. Mandell LA, Wunderink RG, Anzueto A, et al: Infectious Diseases Society of America/American Thoracic Society consensus guidelines on the management of community-acquired pneumonia in adults, Clin Infect Dis 44(Suppl 2):S27–S72, 2007.

67. Waterer GW, Rello J, Wunderink RG: Management of community-acquired pneumonia in adults, Am J Respir Crit Care Med 183:157– 164, 2011.

68. Grijalva CG, Nuorti JP, Arbogast PG, Martin SW, Edwards KM, Griffin MR. De- cline in pneumonia admissions after routine childhood immunization with pneumococcal conjugate vaccine in the USA: a time-series analysis. Lancet 2007;369: 1179-86.

69. Nelson JC, Jackson M, Yu O, et al. Impact of the introduction of pneumococcal conjugate vaccine on rates of community acquired pneumonia in children and adults. Vaccine 2008;26:4947-54.

70. Griffin MR, Zhu Y, Moore MR, Whit- ney CG, Grijalva CGUS. U.S. hospitalizations for pneumonia after a decade of pneumococcal vaccination. N Engl J Med 2013;369:155-63.

71. Lexau CA, Lynfield R, Danila R, et al. Changing epidemiology of invasive pneumococcal disease among older adults in the era of pediatric pneumococcal conjugate vaccine. JAMA 2005;294:2043-51.

72. Pilishvili T, Lexau C, Farley MM, et al. Sustained reductions in invasive pneumococcal disease in the era of conjugate vaccine. J Infect Dis 2010;201:32-41.

73. Jain. Community-Acquired Pneumonia Requiring Hospitalization among U.S. Adults. NEJM 2015

74. Nicholas, Alexander S., and Evan A. Nicholas. Atlas of Osteopathic Techniques. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2012. Print.

REVIEW ARTICLE

Knee Pain in Adults with an Osteopathic Component

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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.

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 individual and societal costs of knee pain, along with the debilitating long-term consequences, make diagnosis and effective management a top priority for a primary care physician. This paper presents an osteopathic approach to the diagnosis and treatment of knee pain in the primary care setting, with a focus on etiologies, clinical presentations, diagnostic strategies, and treatment. Major anatomical, biomechanical, and environmental considerations are also discussed. Finally, a summary of evidence-based studies investigating the effectiveness of Osteopathic Manipulative Treatment (OMT) and other non-pharmacologic treatments in the management of common knee conditions is presented.

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

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sometimes correlated with rickets.[6] 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.[4] 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.[41] Knee joint stability is conferred mainly by the soft tissues of the capsule: ligaments, tendons, and menisci.[7,8] The ligaments confer static stability to the knee joint, while the muscles and tendons provide dynamic stability during motion.[8] 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.[9] It contains the medial collateral ligament (MCL), which is the most commonly injured ligament in the knee, the medial meniscus, and the medial patellofemoral (MPFL) ligament.[10] 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.[9] 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.[10] 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.[10] 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[10] 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.[11-13] Non-modifiable risk factors include gender, age, race, and genetic predisposition.[12-15] 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 1 *(page 29 and 31)*.[9,16-19] 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.[20]

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-

risk activities. Traumatic injuries are most often elicited based on history and can be confirmed by physical exam findings. The need for radiographic studies to rule out a fracture may be determined by the Ottawa knee rules (see Management).[16] 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 *(Table 2, page 32)*[6,44] 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.[16] For older patients with chronic knee pain, a physician should consider osteoarthritis high on the differential *(Table 1, Figure 1 - page 30)*.

Patients with knee pain can also present with a joint effusion for which they may need an arthrocentesis. Various etiologies such as soft tissue injuries, fracture, septic arthritis, infectious, autoimmune, crystalline deposits, and tumors can lead to this clinical presentation (Table 1). Evidence of inflammation in the synovial fluid can indicate a more serious cause, while the lack of inflammation can point to a strain or sprain.[16] If the patient's history is suspicious for autoimmune disease, serum markers should be obtained. If a physician is unable to diagnose the underlying cause of the patient's knee pain and conservative management is unsuccessful, they should consider referral to sports medicine, orthopedics or rheumatology based on the clinical history.

When evaluating a patient with knee pain, it is important for osteopathic physicians to perform a thorough osteopathic structural exam. This includes closely observing the patient's gait throughout the visit and noting any signs of discomfort. Patients with knee pain often present with a limp because they are unable to bear weight on one or both knees. Such a drastic change in gait may suggest an alteration in the patient's knee function due to the loss of muscular or ligamentous support.[6] Considering the interrelatedness between anatomical structures and function, landmarks as well as surrounding musculature and fascia should be palpated for any tissue texture changes.[6] Other potential causes should always be considered like leg length discrepancies, functional muscle imbalances and Q angle It is also important to diagnose somatic dysfunctions within the lower extremity and throughout the rest of the body. Somatic dysfunctions in proximal regions like the hip can often lead to the body compensating elsewhere in order to maintain normal gait and posture. This can lead to referred pain in areas like the lower back or the knee. Diagnosing and treating all somatic dysfunctions throughout the body is hence critical before a patient's knee pain can be directly attributed to the knee itself.

MANAGEMENT

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.[17,18] The Ottawa Knee rules are highly sensitive, evidence-based guidelines dictating that an x-ray is required in a patient with acute knee injury only if one or more of 5 criteria are met: Age 55 years or older; tenderness at head of fibula, isolated tenderness of the patella, inability to flex to 90 degrees, and inability to bear weight on the leg (take 4 steps) immediately following injury and in the emergency department. Patients who are older tend to have more fragile bones and are more likely to have fractures. The other criteria are based on common symptoms seen in acute knee fractures.[18] Surgical referral should be considered in the presence of specific types of ligamentous or meniscal injury, if the patient is young or an athlete, if meniscal injury results in locking of the knee due to a displaced fragment, or if a high degree of instability is present.[19] Immediate treatment of acute knee injury should begin with the application of "RICE"- rest, ice, compression, and elevation. Next, a combination of pharmacologic and non-pharmacologic treatments may be integrated to reduce inflammation and pain, strengthen the affected muscle groups, and correct somatic dysfunctions (see Treatments in Table 1).[21]

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.[21] 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.[22,23] 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.[21]

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.[24,25,26,27] The most common conditions for which non-pharmacologic management is used are osteoarthritis (OA) and patellofemoral pain syndrome (PFPS). A study by Deyle[28] 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.[29] The strengthening of the quadriceps muscle was shown to improve joint stability and significantly decrease pain.[28,29] 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.[30,31] Pinto[32] found that exercise therapy and manual therapy were more cost effective when compared to pharmacological therapy for OA of the knee.

TABLE 1:

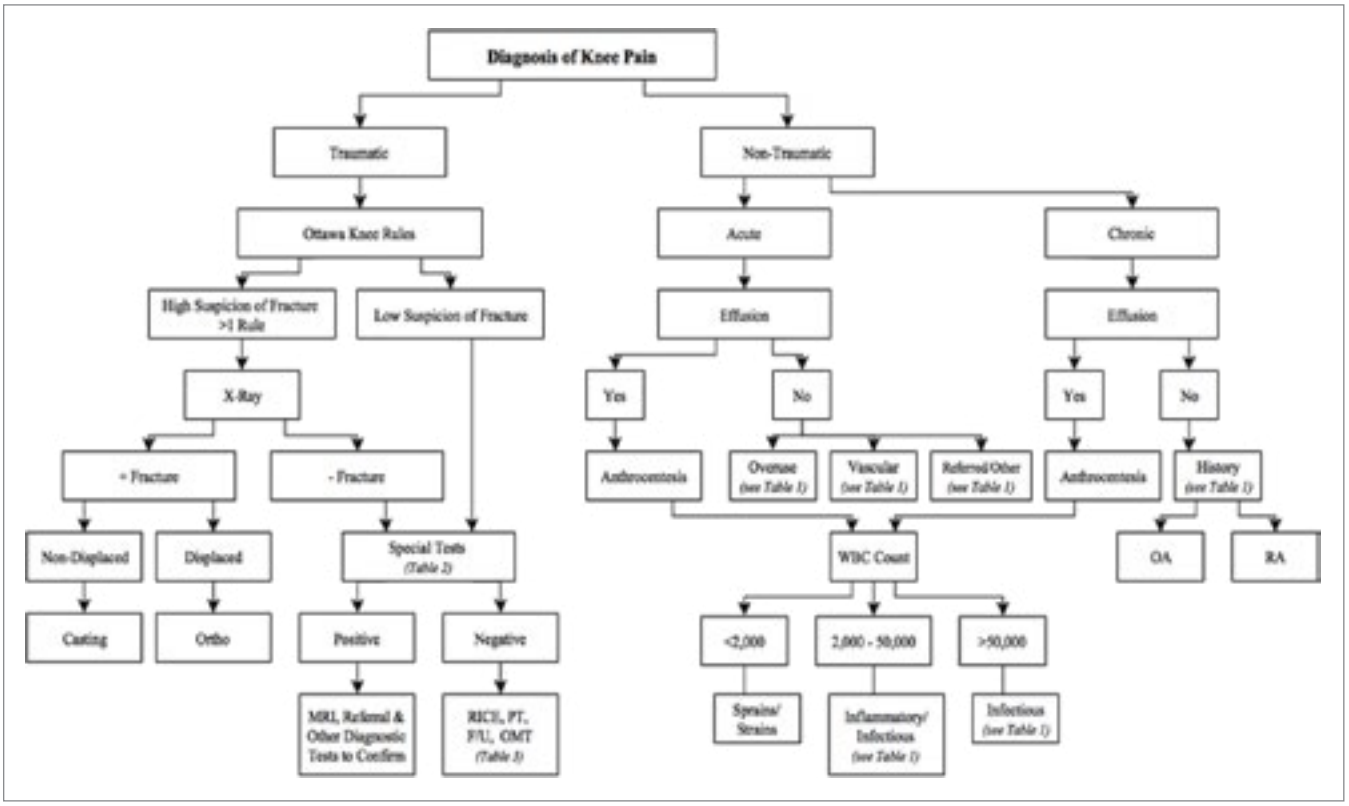
Etiologies, Diagnosis, and Treatment of Knee Pain

		Clinical Presentation	Diagnosis	Treatment
Trauma	Knee Fracture	Axial loading / falls onto flexed knee. Limited ambulation.	Radiographs. Assess neurovascular integrity	Non-displaced Fracture: Casting Displaced Fracture: Surgery
	Patellar Dislocation	Noticeable deformity, retinacular pain, inability to flex knee	Patella apprehension test. Radiographs	Patellar reduction with casting
	MCL / LCL	Medial or lateral pain and swelling with focal tenderness	Valgus / Varus stress tests, Radiograph / MRI	PRICE, NSAIDs, Brace, Surgical Correction
	ACL / PCL	"Pop" / Deep pain / Immediate swelling / Weakness / Instability	Lachman, Pivot shift, Anterior / Posterior drawer / MRI	PRICE, NSAIDs, Brace, Surgical Correction
	Meniscal Injuries	Medial / lateral pain / tenderness catching / locking / popping	Thessaly / McMurray / Apley tests / X-ray / MRI	PRICE, NSAIDs, PT, Activity Modification, Surgery
	Osteochondral Lesions / Osteochondritis Dessicans	Dull pain/ mild locking or clicking of the knee	X-ray/ MRI	PRICE, NSAIDS, PT, Brace, Surgical Correction
Overuse	Popliteus Tendinitis	Posterolateral knee pain, worse with downhill running	Webb test: painful internal rotation with 20-30 degrees of knee flexion	RICE, Quadriceps strengthening
	Patellar Tendinitis	Pain at inferior pole of the patella exacerbated by jumping	Focal patellar pain with activity	RICE, cryotherapy
	Iliotibial Band Syndrome	Lateral knee pain radiating towards hip; common in runners	Ober Test: abduct / adduct leg with hip hyperextended	RICE, iliotibial tract stretch; lateral wedge if heel is varus
	Patellofemoral Syndrome	Chronic, aching anterior bilateral pain with flexion; instability	Theatre sign; Pain with squatting and patellar compression	PRICE OMM, quadriceps & gluteus strengthening, tape, orthoses
	Bursitis (Pes anserinus pain syndrome (PAPS))	Medial knee pain along proximal tibia worse with ascending / descending stairs	Focal tenderness at bursa without swelling/induration	PRICE; hamstring and quad strengthening
	Bursitis (Pre-Patellar) Housemaid's Knee	Pain/swelling anterior to patella; History of direct pressure from repetitive kneeling	Fluctuant subcutaneous swelling anterior to lower patella;	PRICE
	Synovial Effusion	Tightness and extra-articular swelling anterior to patella	Arthrocentesis / Synovial fluid analysis	Fluid removal; treat underlying cause
Age / Rheumatic	Osteoarthritis	Insidious, bilateral swelling, pain with use, crepitus, and decrease ROM, stiffness	Weight bearing X-rays, joint narrowing, osteophyte, subchondral sclerosis	PRICE, NSAIDs, Weight loss, Lifestyle modifications, Total knee replacement if refractory
	Rheumatoid Arthritis	Morning stiffness > 1 hour, polyarticular swelling	Serum rheumatologic assays	Medications, PT, OT, Surgery if needed
	Gout	Swelling and tenderness often in big toe, onset of pain at night	Arthrocentesis (sodium urate crystals- treatment goal <6)	Medications (NSAIDS, Colchicine, steroids etc.)
	Pseudogout	Sudden joint pain and tenderness, often in the knee	Arthrocentesis (calcium pyrophosphate crystals)	Medications (NSAIDS, Colchicine, steroids etc.)

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), Collins[31] 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. Perlman[33] 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] Suter[35] 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.[6,34,38] The best studied conditions with proven OMT efficacy are osteoarthritis, patellofemoral pain syndrome and post-surgical care.[26,34]

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 Deyle[29] 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

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

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[33] 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.[37] Anterior cruciate ligament (ACL) tear is one of the most common and debilitating knee injuries. A JAOA Case report by Gugel[38] 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

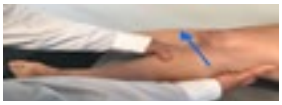
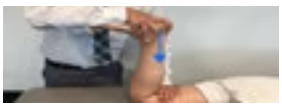
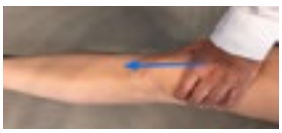
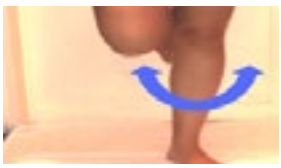
Test	Method or Appearance	Pictures	Significance
Varus-Valgus Stress Test	Abduction/adduction motion to the proximal tibia with knee extended and flexed	 	Laxity at 30 degrees = Injury to the MCL (valgus) or LCL (varus) Laxity at 0 degrees = Injury to the MCL/LCL and PCL
Lachman Test (most sensitive)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

TABLE 3:

OMT Treatments of Knee Pain

Technique	Region of Treatment	Clinical Findings	Diagnosis
Muscle Energy: Place bone or joint into barrier and apply isometric resistance against patient's active contraction of muscle for 3-5 sec; Repeat 3-5 times	Posterior Fibular Head	Foot inversion, forefoot adduction, tibial rotation	Symptoms of compression of peroneal nerve
	Anterior Fibular Head	Foot eversion, forefoot abduction, tibial external rotation	Lateral Knee Pain
	Tibiofemoral joint: Knee Extension /Flexion, Internal / External Rotation Somatic Dysfunction	Internal rotation of femur, external rotation of tibia (due to relaxation of popliteus)	OA, RA, Baker's Cyst
		External rotation of femur, internal rotation of tibia (due to contraction of popliteus)	
	Hip: anterior / posterior rotation, superior / inferior shear, inflare / outflare somatic dysfunction	Flexion / Extension	Extrinsic causes / Referred Pain (see Figure 2)
		Abduction / Adduction	
		Internal / External Rotation	
	Lumbar Spine	Type I SD	Neutral Group Curve
		Type II SD	Non-neutral Group Curve
Counterstrain: Position joint to shorten muscle until pain is relieved / "mobile point" is reached. Hold positioning for 90 seconds to allow for reduction in proprioceptive firing. Return joint slowly to neutral to prevent re-initiation of inappropriate firing	Anterior Patella	T.P - Patellar tendon	Patellofemoral pain syndrome
	Medial/Lateral Patella	T.P - Medial or lateral patellar surface	
	Posterior Knee	T.P - Medial or Lateral ACL	ACL/PCL injury; Gastrocnemius sprain; Popliteal (Baker's cyst); DVT
		T.P - Center of Popliteal Fossa	
		T.P - Lower popliteal fossa	
	Medial Knee	T.P - Medial Joint Line	Medial Meniscus injury, OA, pes anserine bursitis, medial plica syndrome, medial collateral ligament sprain, medial meniscal tear
		T.P - Medial hamstring muscle, distal attachment	
	Lateral Knee	T.P - Lateral joint line	Lateral meniscus injury, lateral compartment OA, lateral collateral ligament sprain, lateral meniscal injury, iliotibial band tendonitis
		T.P - Lateral hamstring, distal attachment, near fibular head	
FPR: 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	Tibiofemoral joint	Point tenderness at and medial to midpoint of knee joint	OA, pes anserine bursitis medial plica syndrome, medial collateral ligament sprain, medial meniscal tear
HVLA: 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	Anterior / Posterior Fibular Head	Lateral Knee Pain; if Posterior Fibular Head symptoms of peroneal nerve compression	Lateral Compartment OA, lateral collateral ligament sprain, lateral meniscal tear, iliotibial band tendonitis

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,[41] 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:

1. Domino F, Baldor R, Golding J, Stephens M. Page 676. The 5-Minute Clinical Consult Standard. Philadelphia, PA: Wolters Kluwer Health; 2015.
2. Baker P. Knee disorders in the general population and their relation to occupation. Occupational and Environmental Medicine. 2003;60(10):794-797.
3. Nguyen U, Zhang Y, Zhu Y, Niu J, Zhang B, Felson D. Increasing Prevalence of Knee Pain and Symptomatic Knee Osteoarthritis: Survey and Cohort Data. Annals of Internal Medicine. 2011;155(11):725.
4. Murphy L, Helmick C. The Impact of Osteoarthritis in the United States. Orthopaedic Nursing. 2012;31(2):85-91.
5. Guccione A, Felson D, Anderson J et al. The effects of specific medical conditions on the functional limitations of elders in the Framingham Study. Am J Public Health. 1994;84(3):351-358.
6. DiGiovanna E, Schiowitz S, Dowling D. Chapter 93-94. An Osteopathic Approach to Diagnosis and Treatment. Philadelphia, PA: Lippincott Williams and Wilkins; 2005.
7. Koval K, Zuckerman J. Chapter 34. Handbook of Fractures. Philadelphia, PA: Lippincott Williams & Wilkins; 2006.
8. Miller M, Hart J. Pages 199-201. Review of Orthopaedics. Philadelphia, PA: Saunders/Elsevier; 2008.
9. Anderson B. Chapter 9. Office Orthopedics for Primary Care. Philadelphia, PA: W.B. Saunders Co.; 2006.
10. Bollen S. Epidemiology of knee injuries: diagnosis and triage. British Journal of Sports Medicine. 2000;34(3):227-a-228.
11. Rossignol M. Primary osteoarthritis of hip, knee, and hand in relation to occupational exposure. Occupational Environmental Medicine. 2005;62(11):772-777.
12. Felson D, Zhang Y. An update on the epidemiology of knee and hip osteoarthritis with a view to prevention. Arthritis & Rheumatism. 1998;41(8):1343-1355.
13. Felson D. Risk Factors for Osteoarthritis. Clinical Orthopaedics and Related Research. 2004;427:S16-S21.
14. Cooper C, Snow S, McAlindon T et al. Risk factors for the incidence and progression of radiographic knee osteoarthritis. Arthritis & Rheumatism. 2000;43(5):995.
15. Blagojevic M, Jinks C, Jeffery A, Jordan K. Risk factors for onset of osteoarthritis of the knee in older adults: a systematic review and meta-analysis. Osteoarthritis and Cartilage. 2010;18(1):24-33.
16. Jackson J. Evaluation of Acute Knee Pain in Primary Care. Annals of Internal Medicine. 2003;139(7):575.
17. Skinner H. Chapter 3. Current Diagnosis & Treatment In Orthopedics. New York: Lange Medical Books/McGraw-Hill Medical Pub. Div.; 2006.
18. Calmbach W, Hutchens M. Evaluation of Patients Presenting with Knee Pain: Part I. American Family Physician. 2003;68(5):907-912.
19. Calmbach W, Hutchens M. Evaluation of Patients Presenting with Knee Pain: Part II. American Family Physician. 2003;68(5):917-922.
20. Chila A. Chapter 42. Foundations Of Osteopathic Medicine. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2011.
21. Barron MC, Rubin BR. Managing osteoarthritic knee pain. The Journal of the American Osteopathic Association. 2007;107(10):506-507.
22. Day H. Office Orthopedics for Primary Care: Diagnosis and Treatment. Annals of Internal Medicine. 1999;130(10):868.
23. Marcus D. Pharmacologic Interventions for Knee Osteoarthritis. Annals of Internal Medicine. 2015;162(9):672.
24. Fransen M, Crosbie J, Edmonds J. Physical therapy is effective for patients with osteoarthritis of the knee: a randomized controlled clinical trial. Journal of Rheumatology. 2001;114(2):574.
25. Collins N, Bisset L, Crossley K, Vicenzino B. Efficacy of Nonsurgical Interventions for Anterior Knee Pain. Sports Medicine. 2012;42(1):31-49.
26. Abbott J, Robertson M, Chapple C et al. Manual therapy, exercise therapy, or both, in addition to usual care, for osteoarthritis of the hip or knee: a randomized controlled trial. 1: clinical effectiveness. Osteoarthritis and Cartilage. 2013;21(4):525-534.
27. Maziares B, Thevenon A, Coudeyre E, Chevalier X, Revel M, Rannou F. Adherence to, and results of, physical therapy programs in patients with hip or knee osteoarthritis. Development of French clinical practice guidelines. Joint Bone Spine. 2008;75(5):589-596.
28. French H, Brennan A, White B, Cusack T. Manual therapy for osteoarthritis of the hip or knee - A systematic review. Manual Therapy. 2011;16(2):109-117.
29. Deyle G. Effectiveness of Manual Physical Therapy and Exercise in Osteoarthritis of the Knee. Annals of Internal Medicine. 2000;132(3):173.
30. Fakuda T, Rossetto F, Magalhaes E, Bryk F, Garcia Lucareli P, De Almeida Carvalho N. Short-Term Effects of Hip Abductors and Lateral Rotators Strengthening in Females With Patellofemoral Pain Syndrome: A Randomized Controlled Clinical Trial. Journal of Orthopaedic & Sports Physical Therapy. 2010;40(11):736-742.
31. Mascal C, Landel R, Powers C. Management of Patellofemoral Pain Targeting Hip, Pelvis, and Trunk Muscle Function: 2 Case Reports. Journal of Orthoapedic & Sports Physical Therapy. 2003;33(11):647-660.
32. Pinto D, Robertson M, Abbott J, Hansen P, Campbell A. Manual therapy, exercise therapy, or both, in addition to usual care, for osteoarthritis of the hip or knee. 2: economic evaluation alongside a randomized controlled trial. Osteoarthritis and Cartilage. 2013;21(10):1504-1513.
33. Bjordal J, Klovning A, Ljunggren A, Slordal L. Short-term efficacy of pharmacotherapeutic interventions in osteoarthritic knee pain: A meta-analysis of randomized placebo-controlled trials. European Journal of Pain. 2007;11(2):125-138.
34. Brantingham J, Globe G, Pollard H, Hicks M, Korporaal C, Hoskins W. Manipulative Therapy for Lower Extremity Conditions: Expansion of Literature Review. Journal of Manipulative Physiological Therapeutics. 2009;32(1):53-71.
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

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

36. Van den Dolder P, Roberts D. Six sessions of manual therapy increase knee flexion and improve activity in people with anterior knee pain: a randomized controlled trial. Australian Journal of Physiotherapy. 2006;52(4)261-264.

37. Suter E, McMorland G, Herzog W, Bray R. Decrease in quadriceps inhibition after sacroiliac joint manipulation in patients with anterior knee pain. Journal of Manipulative and Physiological Therapeutics. 1999;22(3):149-153.

38. Patterson M. Basic Mechanisms of Osteopathic Manipulative Treatment: A Must Read. The Journal of the American Osteopathic Association. 2015;115(9):534.

39. Ebert JR, Joss B, Jardine B, Wood DJ. Randomized Trial Investigating the Efficacy of Manual Lymphatic Drainage to Improve Early Outcome After Total Knee Arthroplasty. Archives of Physical Medicine and Rehabilitation. 2013;94(11):2103-2111.

40. Gugel Mjohnston W. Osteopathic Manipulative Treatment of a 27-Year-Old Man After Anterior Cruciate Ligament Reconstruction. The Journal of the American Osteopathic Association. 2006;106(6):346.

41. Sadovsky R. Evaluating Patient's with Acute Knee Pain: A Review. American Family Physician. 2004; 69(11): 2695.

42. Willliams Davis I. Hamill J, Buchanan TS. Lower Extremity Kinematic and Kinetic Differences in Runners With High and Low Arches. Journal of Applied Biomechanics. 2001;17(2):153–163.

43. Gross K, Felson D, Niu J et al. Association of flat feet with knee pain and cartilage damage in older adults. Arthritis Care & Research. 2011;63(7):937-944.

44. Crespo B, James E, Metsavaht L, LaPrade R. Injuries to posterolateral corner of the knee: a comprehensive review from anatomy to surgical treatment. Revista Brasileira de Ortopedia (English Edition). 2015;50(4):363-379.

REVIEW ARTICLE

Not a Peep: Delirium in the Geriatric Patient

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Keywords:
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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.

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 intertrochanteric 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."[1] 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.[2] 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%.[3]

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[1] (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.[4] Studies of hospitalized older patients have reported the prevalence of delirium at admission as 14-24% and the incidence during hospitalization as 6-56%.[4] Rates in the intensive care unit have been reported as 70-87% and rates post-orthopedic surgery have been reported as 15-53%.[4] Furthermore, from the epidemiological studies conducted in long-term care and post-

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acute settings, a rate of up to 60% has been reported.[4] Delirium is thought to occur in up to 83% of patients at the end of life.[4]

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.[5] This lack of recognition of delirium spans across all specialties with delirium being missed in up to 32-66% of cases.[6] 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.[4] Electroencephalographic (EEG) findings have pointed towards functional derangements and decrease in cortical activity has been noted.[4] 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."[4]

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.[7] They may display loud or fast speech, swearing, singing, laughing, anger, wandering, or other increased activity.[8] 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.[7] 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.[7] They may appear to be staring blankly, have little conversation, or demonstrate slow speech.[8] 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.[8] 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.[7]

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.[8] 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.[9] 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.[9] It is estimated that 28% to 61% of geriatric patients with a hip fracture will experience delirium.[8] 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 1:

Characteristics of Delirium Subtypes[1,7,8]

Hyperactive	Hypoactive	Mixed
Increased psychomotor activity	Decreased psychomotor activity	Characteristics of both hyperactive and hypoactive
Restlessness / Anxious	Decreased alertness / Sleepy	Fluctuating levels of psychomotor activity
Loud or Fast Speech	Slow or little speech / Quiet	
Agitation / Combativeness / Anger	Unawareness / Staring blankly	
Laughing, Singing, Swearing	Apathy / Appear Depressed	
Hypervigilance	Lethargy	
Distractability		
Tangentiality		
Persistent thoughts		
Wandering		

50% for those without mechanical ventilation.[8] Brummel and Girard have referenced that the average medical ICU patient possesses eleven or more risk factors for delirium.[10] 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.[8]

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.[3] The risk of delirium also increases with the severity or stage of dementia.[6] 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.[7,8]

CAUSES OF DELIRIUM

Delirium is typically multifactorial and it may be impossible to isolate just one cause of delirium in a patient.[9] 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.[7] 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.[7] 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."[11] 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."[11] 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:

Risk Factors for Delirium[7,8]

Age greater than 65	Terminal illness
Male sex	Polypharmacy
Comorbidities	Immobility / functional decline
Alcoholism / substance abuse	Sensory impairments including hearing/vision loss
Depression and history of psychiatric illness	Malnutrition
History of chronic pain	Advanced illness / end-stage organ disease
Dementia and other cognitive disorders	Geriatric syndromes (including those not listed within this table)

TABLE 3:

Medications Likely to Induce Delirium[1,8,12]

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

It is estimated that the average ICU patient carries 11 or more risk factors for delirium.[10] 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.[1] 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, hypotension, 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.

TABLE 4:

Laboratory & Radiologic Studies-Evaluating the Geriatric Patient with Delirium[7,8]

Laboratory	Radiologic / Other
Complete blood count	12-lead electrocardiogram
Comprehensive metabolic panel (electrolytes, glucose, BUN/Creatinine, LFTs)	Chest radiograph
Ammonia	CT of the head
Urinalysis / urine culture	Electroencephalography (if seizure expected or delirium is unclear)
Cardiac biomarkers	
Lumbar puncture	
Blood cultures	
Thyroid-stimulating hormone	
Vitamin B12 & Folate levels	
Urine drug screen	
Arterial blood gas	
Rapid plasma reagin	

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.[8] 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]

Before considering pharmacologic treatment for delirium, the above interventions should be taken and the underlying cause should be treated.[7] 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.[7] 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.[8] Haloperidol should be avoided if a patient has underlying parkinsonism, withdrawal syndrome, hepatic insufficiency, or neuroleptic malignant syndrome.[8] 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.[8]

LONG-TERM EFFECTS: DISTRESS, COST, & DEATH

Only 14% of patients with delirium have returned to their baseline level of cognitive functioning at discharge.[1] 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.[7,9] Post-operative delirium is linked to increased morbidity as well as a 1 year mortality of 40%.[9] Han and colleagues found that delirium in geriatric patients in the emergency room is an independent predictor of increased 6-month mortality.[5] 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.[1] 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.[1] 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.[15]

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:

1. Maldonado J: Delirium in the Acute Care Setting: Characteristics, Diagnosis, and Treatment. Crit Care Clin.2008;24:657-722.
2. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. Washington, DC: American Psychiatric Association; 2013.
3. Stall N,Wong C: Hospital-acquired Delirium in Older Adults. CMAJ.2014;186(1):E61.
4. Halter, Jeffrey B, et al. Hazzard's Geriatric Medicine and Gerontology. Ch53.Delirium.The McGraw-Hill Companies Inc; 2009.
5. Han J, Shintani A, Eden S, Morandi A, Solberg L, Schnelle J, et al: Delirium in the Emergency Department: An Independent Predictor of Death Within 6 Months. Annals of Emergency Medicine.2010;56(3):244-252.
6. Inouye SK: Delirium in hospitalized older patients: recognition and risk factors. J Geriatr Psychiatry Neurol.1998;11(3):118-125.
7. Han J, Wilson A, Ely E: Delirium in the Older Emergency Department Patient: A Quiet Epidemic.Emerg Med Clin N Am.2010;28:611-631.
8. Kalish V, Gillham J, Unwin G: Delirium in Older Persons: Evaluation and Management. American Family Physician.2014;90(3):150-158.
9. Allen S, Frankel H: Postoperative Complications: Delirium.Surg Clin N Am.2012;92:409-431.
10. Brummel N, Girard T: Preventing Delirium in the Intensive Care Unit.Crit Care Clin.2013;29:51-65.
11. Flaherty J, Rudolph J, Shay K, Kamholz B, Boockvar K, Shaughnessy M, et al: Delirium is a Serious and Under-recognized Problem: Why Assessment of Mental Status Should be the Sixth Vital Sign. Journal of the American Medical Directors Association.June 2007:273-275.
12. Houman J, Tulebaev S: Management of Common Postoperative Complications.Clin Geriatr Med.2014;30:271-278.
13. Inouye SK, van Dyck CH, Alessi CA, et al: Clarifying Confusion: the Confusion Assessment Method. A new method for detection of delirium. Ann Intern Med.1990;113(12);941-8.
14. Flaherty J: The Evaluation and Management of Delirium Among Older Persons.Med Clin N Am.2011;95:555-577.
15. Grover S, Shah R: Distress due to delirium experience.General Hospital Psychiatry.2011;33:637-639.

BRIEF REPORT

Underlying Appendicitis Leading to Chorioamnionitis in Preterm Rupture of Membranes

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Keywords:

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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 effected by preterm premature rupture of membranes (PPROM)1. In cases of PPROM, the most commonly identified etiology is infection.[1,2,3] 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. Intraoperative 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.

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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.[1,2,3]

During expectant management, 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.[3,4] 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.[2,3]

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.[2,3] 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 fecaliths or hypertrophy of lymphoid tissue. This closed-loop obstruction leads to multiplication of the resident bacteria, leading to distension 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 pregnancies6. 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,[7] none have examined non-obstetric surgical interventions in the face of PPROM. We cannot definitely say whether an under lying 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, chorioamnonitis 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:

1. ACOG: Premature Rupture of Membranes, Practice Bulletin #139. Oct 2013.
2. Cunningham, F. Gary. Williams Obstetrics. 24th ed. New York: McGraw-Hill Professional 2014. 36:804-831. Print.
3. Creasy, Robert K. Creasy and Resnik's Maternal-fetal Medicine: Principles and Practice. 7th ed. Philadelphia, PA: Saunders/Elsevier, 2014. 39: 599-623. Print.
4. Goldbenberg RL, Flatow Culhane J. Infection as a cause of preterm birth. Clinics in perinatology. 30 (2003) 677-700.
5. Brunicardi, F. Charles. Schwartz's Principles of Surgery 10th ed. New York: McGraw-Hill, Health Pub. Division, 2015. 30. Print.
6. Mourad J, Elliott JP, Erickson L, Lisboa L. Appendicitis in pregnancy: New information that contradicts long-held clinical beliefs. Am J Obstet Gynecol 2000; 182: 1027-9.
7. Corneille MG, Gallup TM, Bening T, et al. The use of laparoscopic surgery in pregnancy: evaluation of safety and efficacy. The American Journal of Surgery. (2010) 200, 363-376.

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Bilateral Painless Eye Lesions

Craig Bober, DO

Lankenau Medical Center , Wynnewood, Pennsylvania

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

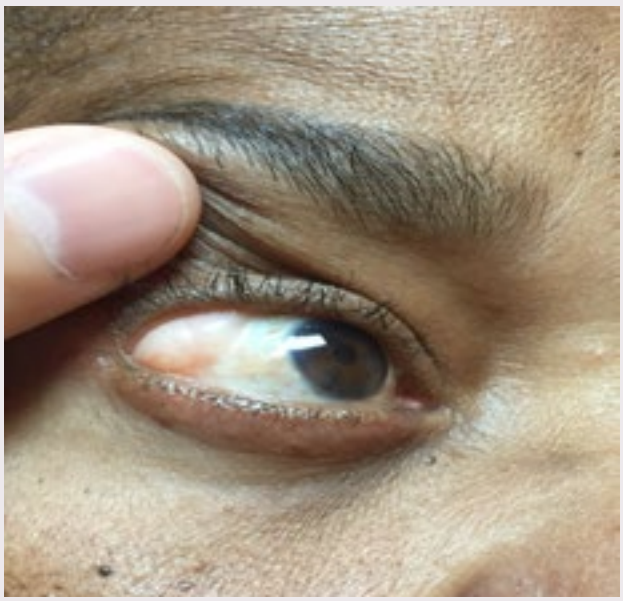
B. Orbital fat prolapse

C. Pinguecula

D. Pterygium

E. Squamous Cell Carcinoma

FIGURE 1:



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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.[1] 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.[2,3]

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.[4] These lesions are almost always found laterally and frequently unilaterally; however, case reports have noted rare medial presence of dermolipomas.[5]

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 thin younger(mean age 22.5) females.[2] 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.[6]

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 period of months, and is usually seen in older men.[3] Pingueculas are yellow, well demarcated nodules that are very common in adults. These benign, immobile growths are usually only slightly raised, clearly distinct from the peripheral eye borders and more often located nasally—unlike dermolipomas.[3] A pterygium is a fleshy appearing, triangularly shaped, conjunctival growth that advances nasally toward the cornea, also seen in older men.[3]

The diagnosis of a dermolipoma is made clinically but if uncertain, imaging studies such as CT or MRI can help differentiate the mass based on fat extension and location.[4] Very rarely does a biopsy need to be performed to confirm the diagnosis. Most commonly no treatment is needed and conservative therapy such as observation is elected. Consideration of surgery should be limited but may be appropriate if there is mechanical compromise of lid function, cosmetic concerns, or persistent irritating foreign body sensation.[7] However, patients must be counseled on the many potential negative post-operative complications (xerophthalmia, diplopia, ptosis) due to close proximity to vital structures of the eye (lacrimal gland, lateral rectus muscle, conjunctiva) that often outweigh the benefits.[7]

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.[8,9] 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.[10]

Ophthalmologic anomalies occur in about 50 percent of Goldenhar-Gorlin syndrome cases, one of the most common of which is a dermolipoma.[8] The dermolipomas seen in this condition however, often are quite pronounced, located in the infratemporal quadrant and are most often unilateral.[5] 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.[9] 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.[10]

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:

1. Mansour AM, Barber JC, Reinecke RD, et al. Ocular Choristomas. Surv Ophthalmol. 1989 Mar-Apr; 33(5):339-58.
2. McNab AA, Wright JE, Caswell AG. Clinical features and surgical management of dermolipomas. Aust N Z J Ophthalmol. 1990 May; 18(2):159–62.
3. Riordan-Eva P, Cunningham E, Jr: Vaughan & Asbury's General Ophthalmology. New York, Lange Medical /McGraw-Hill Medical Pub. Division, 2011, ed 18, pp 107-112.
4. Kim E, KimHJ, KimYD, et al. Subconjunctival Fat Prolapse and Dermolipoma of the Orbit: Differentiation on CT and MR Imaging. AJNR Am J Neuroradiol. 2011 Mar; 32(3): 465-467
5. Rajabi MT, Ramezani K. Lipodermoid cyst: A report of a rare caruncular case. MEAJO 2015; 22(4): 528-530.
6. Kim YD, Goldberg RA. Orbital fat prolapse and dermolipoma: two distinct entities. Korean J Ophthalmol 1994; 8:42-43.
7. Fries P, Kazim M. 2002. Benign Pediatric Orbital Tumors. Katowitz J, Editor. New York (NY): Springer. Chapter 13, Pediatric Oculoplastic Surgery; p. 442-44.
8. Mansour AM, Wang F, Henkind P, et al. Ocular findings in the facioauriculovertebral sequence (Goldenhar-Gorlin syndrome). Am J Ophthalmol 1985 Oct; 100(4): 555-9.
9. Kumaresan R, Srinivasan B, Narayanan M, et al. Craniofacial abnormalities in goldenhar syndrome: a case report with review of the literature. Plast Aesthetic Res. 2014;1(3):108
10. Sharma JK, Pippal SK, Raghuvanshi SK, Shitij A. Goldenhar-Gorlin's syndrome: A case report. Indian J Otolaryngol Head Neck Surg. 2006; 58(1):97-101.

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Iowa ACOFP Midwinter Osteopathic Family Practice Conference
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RESPIRATORY TRACT INFECTIONS

Peter Zajac, DO, FACOFP, Author

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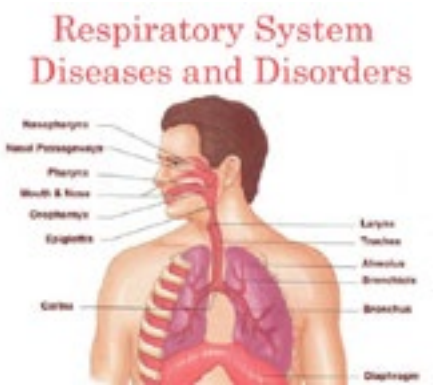

Respiratory tract infections are any infection that affect the nose, sinuses, and throat (i.e. the upper respiratory tract) or airways and lungs (i.e. the lower respiratory tract). Viruses are the main cause of the infections, but bacteria can cause some. You can spread the infection to others through 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.***

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