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- Approach to the Patient with a Tremor
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- Direct Primary Care: Emerging Practice Alternative
- The Food Allergy Revolution
- Gas, Bloating and Belching: Tips for the Primary Care Physician
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The content should include the following:

Abstract
Introduction
Methods
Results
Discussion
Conclusions
Acknowledgments

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Family Medicine / OMT Certification / OCC Performance Evaluation Only	**ACOFP Conference** **Austin, TX** March 22 - 25 , 2018 March 20 - 23, 2018	**November 1, 2017** *Late fee through December 1*
Family Medicine / OMT Certification Only Cognitive Exam	**Electronic Testing** Regional Sites April 28, 2018	**October 1, 2017** *Late fee through December 1*
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VOLUME 9 | NUMBER 4

CONTENTS

OSTEOPATHIC FAMILY PHYSICIAN SPECIALTY PEER REVIEWERS

Nazem Abdelfatta, MD
Family Medicine, Obstetrics, Women's Health

Richard L. Averitte, Jr, MD
Dermatology

Dana Baigrie, DO
Clinical Images

Jeffrey Benseler, DO
Radiology

Shagun Bindlish, MD
Diabetes and Endocrinology

John Bissett, DO
Clinical Images

Warren Bodine, DO
Sports Medicine & Family Medicine

Grace Brannan, PhD
Statistics/Design

Natasha Bray, DO
Ethics

Janis Coffin, DO
Practice Management

Rob Danoff, DO
Emergency Medicine, Preventive

Robin Devine, DO
Statistics/Design

Brian Downs, DO
HIV, Wound Care

Dennis Eckles, DO
Diabetes, Rural Medicine

Gail Feinberg, DO, FACOFP
Academic

Robert Grubb, DO
Sports Medicine

Rose Hall, DO
Family Medicine

Nadia Hasan, DO
Clinical Images

Robert Hunter, DO
Health Policy, Hospice/Palliative Care, ER, Diabetes, Wound Care

Ronald P. Januchowski, DO
Military & Rural/Underserved

Holly Kanavy, DO
Dermatology

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

Uzma Khan, DO
Family Medicine

Frank Komara, DO
Geriatrics

Paul Lazar, DO
Adult Family Medicine, Geriatrics, Academic

Mohammad Mansour, MD
Inpatient Medicine, Cardiology, Pulmonary, Geriatrics, Obstetrics

Sarah Mitchell, DO
Family Medicine

Wadsworth Murad, DO
Psychiatry

Merideth Norris, DO, FACOFP
Addiction

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

Jon Parham, DO
Preventive Medicine, Pulmonary, Public Health, Geriatrics, Medical Errors

Raena Pettitt, DO
Disease Prevention & Wellness

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

Mischa Pollard, MD
Women's Health, Child Sexual Abuse, Pediatrics, Obstetrics

M. Jay Porcelli, DO, FACOFP *dist*.
Pain Management

Joseph Reyes, DO
Pain Management

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

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

Lawrence Sawicki, DO
Clinical Images

Jay Shubrook, Jr., DO, FACOFP
Endocrinology

Leslie Sleuwen, MD
Community Medicine

Frederick Stine, DO
Pediatrics, Nephrology, Emergency & Critical Care

Lindsay Tjiattas-Saleski, DO
Clincial Images, Emerency Medicine

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

Stuart Williams, DO
OMM

Barbara Wolf, DO
Psychology

William Woolery, DO, PhD, FACOFP
Geriatrics

Julian Vega, DO
Clinical Images

Sheldon Yao, DO
Cardiology

Peter Zajac, DO, FACOFP
Patient Education

2017 STUDENT PEER REVIEW & WRITING INTERNS

Vaidehi Ambai
Philadelphia College of Osteopathic Medicine

Kristen Constantine, MPH
Lake Erie College of Osteopathic Medicine

McKenzie Denton
University of Pikeville –Kentucky College of Osteopathic Medicine

Ashton Dixon
University of Pikeville –Kentucky College of Osteopathic Medicine

Nicole Findlay
Texas College of Osteopathic Medicine

Matthew Hadfield
Liberty University College of Osteopathic Medicine

Robert Malinak
University of Pikeville –Kentucky College of Osteopathic Medicine

Sujith Modugular
University of Pikeville –Kentucky College of Osteopathic Medicine

Benjamin Oldach
Ohio University College ofOsteopathic Medicine

Thomas Thacker
University of Pikeville –Kentucky College of Osteopathic Medicine

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EDITOR'S MESSAGE

Are You Ready?

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

Are you having a little anxiety while you are learning the government regulations this summer for the new requirements for the quality measurement program that will apply to your future? *OFP* has you covered with an article on *Osteopathic Approach to Anxiety*. You can do this. Thought you just needed to learn how to diagnose and treat patients? Well that idea should have passed by now. We continue to type, check this box and that box, and provide peer-to-peer prior authorizations (which are only a fraction of the ones the staff is doing.)

I often wonder if someone landed from Mars and watched our crazy system for a day, what would they think? (It is complicated I hear.) The idea that two people in my little office call the insurance company and stay on hold for half an hour to get permission for my patient to get an imaging study that was suggested by the imaging professional or a drug they have been on for years is bizarre. In addition, Medicare is going to increase the number of these calls like this in my office by 50% in six months. Are you ready?

The anxiety article is great and includes diagnosis and treatments with the inclusion of osteopathic manual medicine. Reading it will give you to tools to relax this summer.

Also included in this issue is a review of the literature article, *Adverse Childhood Experiences: A Call to Action for Osteopathic Medicine*, focusing on adverse childhood events and how they may affect the future of a person, especially their health. This information has been building for thirty years in the literature. Make time for some positive childhood experiences this summer with the children in your lives.

However, if you are heading for the beach, the *Melanoma for Primary Care* article will certainly motivate you to avoid sunburn and give you a review of the ways to best to do that.

In the research article, *Tobacco Use & Sleep Problems Among Acticve Duty Service Members*, the small study documents that not only is smoking unhealthy it also affects the sleep of active duty service members.

Continuing with our visual clinical image series, we have a little nugget for you but I'm not going to give you the answer so the suspense will keep you reading the journal through to the end.

Don't forget to take a break and enjoy some summer down time.

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FROM THE PRESIDENT'S DESK

We Can't Afford to Lose Primary Care Physicians Over the CMS Quality Payment Program (QPP) – Help is Here

Rodney M. Wiseman, DO, FACOFP *dist*.
2017 - 2018 ACOFP President

The ACOFP is constantly looking for ways to make our members' lives as physicians easier and to increase stability in these challenging times. The most common thing that members are struggling with, especially in solo, small and rural practices, is making sense of the CMS Quality Payment Program (QPP). This year, Quality Reporting is 60 percent of the Composite Payment Score and affects whether you get an incentive or penalty for your Medicare Part B Fee-For-Service (FFS) patients.

While CMS has tried to help by setting up a training website on the QPP, you can get lost in pages of explanations, which I will refer to as "CMS-speak." It is frustrating to all physicians, but this weighs most heavily on those who "go-it-alone," or have severely restricted resources such as the rural physician communities.

With a shortage of Primary Care Physicians estimated at 14,900 to 35,600 by 2025,[1] and a 55 percent increase of patients age 65 or older since 2010,[2] we can't afford to lose any Primary Care Physicians due to compounding annual CMS penalties. In 2017, the penalty is negative 4 percent of the Medicare FFS revenues, and by 2020 it will grow to negative 9 percent. To avoid these penalties, we need to master the QPP. The plus side is that if you are successful at improving patient outcomes you can gain incentives which can be well above the plus 4 percent to plus 9 percent range.[3] ACOFP would like to share a no-cost way to achieve these incentive payments.

To assist physicians who are in solo, small, and rural practices, ACOFP has partnered with the National Rural Accountable Care Consortium (NRACC), a not-for-profit organization, to bring practices the help they need – at no cost to them. ACOFP is extending an invitation to solo, small and rural practices in all 50 states to join the Practice Transformation Network – as the first step towards the incentives you work so hard for.

What does this look like? The NRACC will provide tools, training, and consulting for you and your practice to maximize patient outcomes and stabilize your revenue streams.

- Modify clinic workflows to address patient care gaps and improve patient outcomes (quality)
- Identify and train a Care Coordinator for your practice to manage the more complex patients, allowing you more time to see patients
- Care Coordinators will be trained and certified, at no cost
- Understand the Merit-Based Incentive Payment System (MIPS), and be guided through the steps by trained healthcare consultants
- Insure that you are conducting and billing for Medicare Prevention Services, many physicians overlook these, despite having patients who need these services
- Implement an innovative care management, population health system to automate patient care and quality management
- Utilize a 24-hour nurse-staffed patient hotline provided for your Medicare patients at no charge
- Improve patient satisfaction at the point of care – your practice will be provided a tablet which will allow patients to quickly complete a patient satisfaction survey
- Assistance will be provided so your practice can be certified as a Patient-Centered Medical Home – earn credits towards your Composite Payment Score for the QPP.

I hope this convinces you to take the next step, which is to sign-up at www.acofp.org/ptn and an Enrollment Specialist from the Practice Transformation Network will contact you. There are limited spaces, and the deadline is October 1, 2017, or when it is full. Please call the ACOFP office for additional information at 847-952- 5523.

Rodney M. Wiseman, DO, FACOFP *dist.*
2017 -2018 ACOFP President

From the American College of Osteopathic Family Physicians.

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RESEARCH ARTICLE

Tobacco Use & Sleep Problems Among Active Duty Service Members

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Keywords:

Behavioral Medicine

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Psychiatry

PTSD

Sleep Apnea

Tobacco

Objective: To determine the consequences of tobacco use on a person's sleep.

Methods: Active duty service members self-disclosing their tobacco use participated in a one month study during which time they completed standardized self-assessment scales which included the Pittsburg Insomnia Rating Scale, Zung Self-Rating Depression Scale, Zung Self-Rating Anxiety Scale, the Epworth Sleepiness Scale, and the Alcohol Use Disorders Identification Test. In addition, subjects completed a baseline and one month later home sleep study.

Results: Twenty - eight active duty subjects reported their tobacco use with half (n=14, 28, 50%) denying use. When comparing the two groups of tobacco users and non-tobacco users, the independent T-Test analyses identified no statistical differences based on the BMI (p=.13) or the Alcohol Use Disorders Identification Test (p=.37). In terms of the respiratory events associated with obstructive sleep apnea significant differences emerged between tobacco users and non-tobacco users. Tobacco users had significantly higher respiratory disturbance index scores, particularly during REM sleep and less oxygen saturation, an observation present at the initial sleep study and in the one month follow-up study. In terms of the apnea/hypopnea index, tobacco users had a nearly significant difference at the initial sleep study and in the one month follow-up study, again with a trend effecting REM sleep.

Conclusion: Tobacco use is a national health concern and motivating reluctant users to quit requires pertinent, evidence based clinical persuasion relevant to the person's life. Preliminary findings in this study suggest tobacco users experience significant sleep disruptions effecting sleep respiration and architecture.

INTRODUCTION

Motivating individuals to quit tobacco use can be an uphill struggle. Even individuals receiving treatment for a different substance use disorder often resist stopping "everything "- meaning tobacco - by wrapping their reluctance in the desire to conquer one problem at a time. At this decisive moment clinicians have a choice, agree with the person's rationale and place smoking cessation on the back burner or present a counter argument that might lead to a different outcome.

It is probably safe to assume that most individuals with a tobacco use disorder are familiar with the major physical problems associated with smoking. Clinicians may not sway smokers with a lecture pointing out the likelihood of cancer and other associated physical conditions. A better approach would tailor the message to current problems in the person's life. One of the more common complaints clinicians encounter, and the focus of this article, is the myriad of sleep problems patients present with in the doctor's office. A chronic insomniac might be receptive to smoking cessation if the clinician can make an adequate clinical argument connecting the dots between tobacco use and another night of restless sleep.

Clinicians seeking guidance on the comprehensive range of sleep problems associated with tobacco use may be surprised by the paucity of published literature on the subject.[1] In a large subjective study based on participants' self-reports, investigators reported that when compared to non-tobacco users, individuals using tobacco had less than six hours sleep, took longer to fall asleep, and experienced less restful, satisfying sleep.[2] In another subjective study, subjects were queried about the sufficiency of their sleep with the investigators' reporting that users of tobacco, whether smoked, smokeless, or passively inhaled, were more likely to relate insufficient sleep when compared to non-tobacco users.[3]

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Polysomnography offers an objective contribution to the study of tobacco's influence on sleep. In one such study, the investigators reported that smokers had less total sleep time, a longer time falling asleep, more sleep related apneic events, and more restless, leg movements.[4] In another study researchers explored the impact of abstinence from tobacco with its accompanying withdrawal and reported more frequent awakenings along with the subjects' more subjective assessments of increased anxiety, irritability, and daytime fatigue.[5] Among chronic smokers, the onset of the first REM sleep is delayed, deep sleep reduced, and sleep less efficient with more arousals interrupting continuity.[6]

Nicotine's short half-life introduces another complication as its effects diminish overnight producing varying degrees of withdrawal.[7] Researchers reported that a 24-hour nicotine patch, when compared to a 16-hour nicotine patch only worn while awake, had a more favorable result in terms of less sleep disruption as evidenced by polysomnography. Interestingly, the 24 hour study group did not subjectively identify better sleep, despite the polysomnography's results.

In this article, the investigators report the results of tobacco use on sleep, using both self-assessment measures and the objective results obtained from home sleep studies. The investigators pursued this study in an effort to better understand the relationships between tobacco use and sleep problems, both of which seem to mutually reinforce each other. Objective data based literature on the subject is limited, particularly among active duty service members.

METHODS

The investigators collected these data as a convenience sample from active duty military subjects participating in a prospective pilot study approved by the Walter Reed Institutional Review Board (IRB). All subjects were enrolled in a substance abuse treatment program at Walter Reed National Military Medical Center in Bethesda, Maryland. All subjects simply reported their use or non-use of tobacco products. Participants agreed to complete three home sleep studies evenly spaced over a one month period along with standardized self-assessment instruments examining alcohol use, sleep, and mood.

The standard of care for service members' enrolled in the substance abuse treatment program requires urine drug screening. The urine drug screen includes amphetamines, barbiturates, benzodiazepines, cocaine, morphine, methadone, phencyclidine, and tetrahydrocannabinol. Service member's testing positive for any of these substances were excluded from the study.

At baseline and at one month the subjects completed the Pittsburg Insomnia Rating Scale (PIRS), Zung Self-Rating Depression Scale (SDS), Zung Self-Rating Anxiety Scale (SAS), and Epworth Sleepiness Scale (ESS). Subjects completed the Alcohol Use Disorders Identification Test (AUDIT) and the Post Traumatic Stress Disorder Checklist (PTSD) – Military Version (PCL-M) only at baseline.

The AUDIT consists of ten questions and five responses per item. Typical questions include, "How often do you have a drink containing alcohol" and "How often do you have six or more drinks on one occasion?" Scores exceeding seven are associated with harmful drinking.[8] In response to the questions, subjects could choose from:

- Never (which scored zero for that scale item)
- Monthly or less
- 2-4 times a month
- 2-3 times a week
- 4 or more times a week (which earned the maximum score for that scale question of four)

The PIRS is a 20-item self-report instrument assessing sleep over the preceding 7-day period.[9] The range of scores on the PIRS is from 0-60 with scores above 20 suggesting insomnia. Typical questions on the PIRS include: "From the time you tried to go to sleep, how long did it take to fall asleep on most nights?" and "If you woke up during the night, how long did it take to fall back to sleep on most nights?"

The SDS is a 20-item self-report instrument in which respondents choose among four descriptions (i.e., "a little of the time," which scored one for that scale item, "some of the time," "good part of the time," and "most of the time", which earned the maximum score for that item of four) in answering questions regarding depression. Typical questions include: "I feel down - hearted and blue" and "Morning is when I feel the best." Scores above 50 suggest clinically significant depression.[10,11]

The SAS is a 20-item self-report instrument in which respondents choose among four descriptions (i.e., "a little of the time," which scored one for that scale item, "some of the time," "good part of the time," and "most of the time," which earned the maximum score for that item of four) in answering questions regarding anxiety. Typical questions include, "I feel more nervous and anxious than usual" and "I fall asleep easily and get a good night's rest". Scores exceeding 45 suggest clinically significant anxiety.[12]

The ESS consists of eight questions which are answered by subjects on a four point scale ranging from 0 "No chance of dozing" to 3 for "definitely would doze."[13] Typical questions that subjects would answer would involve situations such as "sitting and reading, watching TV or sitting inactive. Scores of 10 or greater suggest daytime sleepiness.

The PCL-M is a 17- item self-report instrument assessing military centric symptoms associated with a traumatic event that allows respondents to identify problems that vary from 1 (not at all) to 5 (extremely).[14] For purposes of this study, the investigators used a cut-off score of 50 or greater as suggesting PTSD.

Investigators used the WatchPat 200 (Itamar Medical Ltd., Caesarea, Israel) for the home sleep studies. This wrist worn device includes an actigraph, peripheral arterial tonometer, a pulse oximeter, and an integrated body position and snoring sensor. Proprietary software analyzes these data collected from the home sleep study.

Sleep study results include such measures as the total sleep time, oxygen saturation levels, pulse rate, and a detailed hypnogram. The device also calculates the apnea/hypopnea index (AHI), oxygen desaturation index (ODI), and the respiratory disturbance index (RDI), which are the commonly accepted respiratory diagnostic determinants of OSA.[15]

Researchers comparing the accuracy of this device in measuring the respiratory parameters and sleep architecture with traditional polysomnography (PSG) report strong correlations between the two procedures.[16-19]

RESULTS

After obtaining the subjects' written research consent, twenty-eight active duty subjects reported their tobacco use with half (n=14/ 28, 50%) denying use. Among the total group most were enlisted service members (n=22/28, 79%), male (n=23/28, 86%), between the ages of 21-35 (n=21/28, 75%), and had been deployed to an area of combat operations (n=18/28, 64%) (See Table 1). The average BMI for the group was 26.9 (n=28, SD 5.0). The average PCL-M (n=28, M=48.9, SD 20.48) score fell below the screening threshold.

When comparing the two groups of tobacco users and non-tobacco users, the independent T-Test analyses identified no statistical differences based on the BMI (p=.13), AUDIT (p=.37), SDS (p=.11), PIRS (p=.18), and the Epworth (p=.1). While the difference between the PIRS scores between tobacco users (n=14, M=41.3, SD 10) and non-tobacco users (n=14, M=34.2, SD 16.3) was insignificant both groups reported scores that exceeded the threshold of 20 suggesting the presence of insomnia. The same analysis did identify a significant difference (p=.05) among the two groups with tobacco users reporting higher anxiety scores on the SAS (See Table 2).

TABLE 1:

Characteristics of Military Subjects (n=28)

AGE	n (%)
21-25	7 (25.0)
26-30	8 (28.6)
31-35	6 (21.4)
36-40	3(10.7)
>41	4 (14.3)
GENDER	
Male	24 (85.7)
Female	4 (14.3)
RANK*	
E1-E4	12 (42.9)
E5-E9	10 (35.7)
O1-O3	3 (10.7)
O4-O6	3 (10.7)
MARITAL	
Single	7 (25)
Married	12 (42.9)
Separated	5 (17.9)
Divorced	3 (10.7)
Widowed	1 (3.6)
DEPLOYED	
Yes	18 (64.3)
No	10 (35.7)

**E1-E4 = Junior enlisted rank*
E5-E9 = Noncommissioned officers
O1-O3 = Junior commissioned officers
O4-O6 = Senior commissioned officers

TABLE 2:

Initial Group Test Results

Test	Tobacco Use / n	Mean	SD	Sig (2-Tailed)
AUDIT	Yes / 14 No / 14	15.4 12.3	9.6 8.0	3.7
SDS	Yes / 14 No / 13	54.6 46.0	9.6 16.2	.11
SAS	Yes / 14 No / 13	42.2 32.2	9.5 14.9	.05*
PIRS	Yes / 14 No / 14	41.3 34.2	10.0 16.3	.18
Epworth	Yes / 14 No / 14	12.2 8.4	6.3 5.6	.10
BMI	Yes / 14 No / 14	28.3 25.4	5.9 3.5	.13

**Significant at the 0.05 level (2-tailed)*

AUDIT – Alcohol Use Disorders Identification Test
SDS – Zung Depression Scale
SAS – Zung Anxiety Scale
PIRS – Pittsburgh Insomnia Rating Scale
Epworth – Epworth Sleepiness Scale
BMI – Body Mass Index

An analysis of individual SAS and SDS questions produced interesting results. Tobacco users were significantly more likely to report losing weight, feeling tired, restless, irritable, experiencing numbness and tingling, and frequent urination (See Table 3). One month later subjects completed the SAS and SDS again and reported roughly similar results with tobacco users feeling restless, panicky, tired, and experiencing trembling, a fast heart rate, dizziness, numbness, and dry hands (See Table 4).

In terms of the respiratory measures associated with OSA significant differences emerged between tobacco users and non-tobacco users. Tobacco users had significantly higher RDI scores, particularly during REM sleep and less oxygen saturation, an observation present at the initial sleep study and in the one month follow-up study. In terms of the AHI, tobacco users had a nearly significant difference at the initial sleep study and in the one month follow-up study, again with a trend towards REM sleep (See Table 5, page 14).

TABLE 3:

Initial Zung Depression & Zung Anxiety Questions Related to Tobacco Use

Test Question	Tobacco Use / n	Mean	SD	Sig (2-Tailed)
Q7: Losing Weight[1]	Yes / 14 No / 12	1.71 2.67	1.07 1.07	0.03*
Q10: Tired[1]	Yes / 14 No / 12	3.14 2.33	.95 .98	0.04*
Q13: Restless[1]	Yes / 14 No / 12	3.07 2.33	.83 .78	0.03*
Q15: Irritable[1]	Yes / 14 No / 13	2.93 2.15	1.0 .99	0.05*
Q8: Tired[2]	Yes / 14 No / 13	3.07 1.92	.83 1.38	0.01**
Q14: Numbness[2]	Yes / 14 No / 13	2.21 1.08	1.19 1.32	0.03*
Q16: Frequent[2] Urination	Yes / 14 No / 13	2.21 1.08	1.25 .95	0.01**

**Significant at the 0.05 level (2-tailed)*
*** Significant at the 0.01 level (2-tailed)*

[1]Zung Depression
Qv7: I notice that I am losing weight
Q10: I get tired for no reason
Q13: I am restless and can't keep still.
Q15: I am more irritable than usual

[2]Zung Anxiety
Q8: I feel weak and get tired easily
Q14: I get feelings of numbness and tingling in my fingers & toes
Q16: I have to empty my bladder often

TABLE 4:

One Month Follow-Up Zung Depression & Anxiety Questions Related to Tobacco Use

Test Question	Tobacco Use / n	Mean	SD	Sig (2-Tailed)
Q9: Fast Heart Rate[1]	Yes / 7 No / 8	2.0 1.3	.82 .46	.04*
Q13: Restless[1]	Yes / 7 No / 10	2.29 1.5	.49 .71	.02*
Q3: Panicky[2]	Yes / 8 No / 9	2.25 .89	1.04 .93	.01**
Q4: Falling Apart[2]	Yes / 8 No / 8	1.63 .38	1.06 .52	.01**
Q6: Tremble[2]	Yes / 8 No / 9	1.88 .33	1.25 .71	.01**
Q8: Tired[2]	Yes / 8 No / 9	2.13 1.0	1.36 .87	.06
Q10: Fast Heart Rate[2]	Yes / 8 No / 9	1.75 1.08	1.25 .89	.03*
Q11: Dizzy[2]	Yes / 8 No / 8	.88 .00	1.13 .00	.03*
Q14: Numbness[2]	Yes / 8 No / 9	1.88 .13	1.13 .35	.00**
Q17: Dry Hands[2]	Yes / 8 No / 9	2.63 .78	1.30 .83	.00**

**Significant at the 0.05 level (2-tailed)*
*** Significant at the 0.01 level (2-tailed)*

[1]Zung Depression
Q9: My heart beats faster than usual
Q13: I am restless and can't keep still

[2]Zung Anxiety
Q3: I get upset easily or feel panicky
Q4: I feel like I'm falling apart and going to pieces
Q6: My arms and legs shake and tremble
Q8: I feel weak and get tired easily
Q10: I can feel my heart beating fast
Q11: I am bothered by dizzy spells
Q14: I Get feelings of numbness and tingling in my fingers and toes
Q17: My hands are unusually dry and warm

TABLE 5:

Relationships Between Tobacco Use & Respiratory Measures

Sleep Variable	Tobacco Use / n	Mean	SD	Sig (2-Tailed)
AHI[1]	Yes / 13 No / 12	11.7 3.4	13.7 4.8	.06
RDI[1]	Yes / 13 No / 12	19.2 9.4	11.4 5.6	.01**
RDI REM[1]	Yes / 11 No / 8	25.6 14.7	8.7 4.5	.01**
Oxygen Saturation[1]	Yes / 13 No / 12	94.5 95.9	1.7 1.4	.04*
RDI REM[2]	Yes / 5 No / 12	25.5 13.5	14.2 8.8	.05*
AHI REM[2]	Yes / 5 No / 12	16.7 5.8	15.4 6.9	.06
Oxygen Saturation[2]	Yes / 8 No / 13	94.5 95.7	1.5 .85	.03*

**Significant at the 0.05 level (2-tailed)*
*** Significant at the 0.01 level (2-tailed)*

[1]Initial Sleep Study Results
[2]One Month Follow -Up Sleep Study Results

AHI = apnea-hypopnea index
RDI = respiratory disturbance index
RDI REM = respiratory disturbance index during REM
AHI REM = Apnea Hypopnea Index during REM

COMMENT

Motivating tobacco users to quit is generally an uphill struggle. Even among patients receiving intensive treatment for other substance use disorders a reluctance to quit tobacco commonly arises. Most patients are familiar with the long term physical risks associated with tobacco use and are usually unresponsive to a clinician's appeal along this line. On the other hand, tobacco users may be receptive to quitting if current problems will be alleviated. One of the more common clinical complaints that falls into that category would be sleep problems.[20]

As this preliminary study suggests, even among a cohort of individuals reporting insomnia, tobacco users experience a different set of problems. Perhaps most important is this study's finding pointing towards a higher rate of OSA among self-identified tobacco users. Drilling down a bit further into the sleep study results also seems to suggest that the breathing problems are more likely to occur during REM sleep. Tobacco users' breathing problems were fairly constant based on the results of the two sleep studies subjects completed during the course of the one month study.

Fractured REM sleep may have important clinical consequences by disrupting a key component of the sleep architecture. In one example, REM sleep appears to play an important role in memory consolidation which in turn may affect recovery from post-traumatic stress disorder (PTSD).[21] Chronic sleep disorders that habitually impact REM sleep may be a modifiable risk factor promoting recovery from PTSD.[22] As this study suggests, quitting tobacco could be a modifiable risk factor that would contribute not only to a better night's sleep but might also help individuals with PTSD.

Tobacco users also subjectively report more somatic symptoms than non-tobacco users such as feeling tired, restless, irritable, and numb; all of which constitute common components of many psychiatric disorders which can only can confuse and complicate their treatment. Again, the findings were replicated one month after the subject's completed their initial self-assessments instruments. Clinicians might consider the negative contributions of tobacco use when managing treatment resistant cases.

This study does have limitations, perhaps chief among these being the investigators' reliance on the subjects' self-disclosure of tobacco use without quantifying type, frequency of use, or how recently the subjects quit using tobacco. Another factor that may limit the study's generalizability is the military sample that may not be representative of America's population. Future research could address these limitations as well as including a more heterogeneous sample. In defense of these obvious limitations the investigators would suggest that typical clinical practice may not extend beyond asking the screening question regarding tobacco use, a clinical scenario incorporated in this study. The fairly consistent results the investigators obtained over the one-month study period also bolsters the findings.

Clinicians counseling patients to quit tobacco often face substantial resistance. One possible way clinicians can breech that barrier is by connecting tobacco use with sleep problems. Everyone wants a restful night's sleep and tobacco users rarely have one. The findings in this study suggest the reasons why as tobacco users subjectively report more somatic symptoms and objectively demonstrate REM based breathing problems.

CONCLUSION

Motivating reluctant patients to quit tobacco can be a challenging task. One way, and the focus of this research is for Osteopathic physicians, as part of a holistic approach, is to explain the negative relationship between tobacco consumption and sleep problems. Aside from that, the root cause of a chronic sleep problem that is resistant to interventions, may suggest that tobacco use is the confounding variable preventing relief, and until that is addressed, a restful night's sleep will remain just a dream.

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REVIEW ARTICLE

Melanoma for Primary Care

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The incidence of newly diagnosed malignant melanoma is rapidly increasing. In 2016, it is estimated that there will be 76,380 new cases with 10,130 deaths in the United States. Incidence has increased from 1 in 1500 persons born in the early 1900s to 1 in 50 of Caucasian persons born in 2014, 1 in 200 Hispanics, and 1 in 1000 for African-Americans. Unlike basal and squamous cell carcinoma, malignant melanoma correlates more with intense intermittent UV radiation exposure. Malignant melanoma is the aggressive therapy-resistant skin cancer of melanocytes where thickness of the tumor is the most important prognostic factor. The ABCDs (Asymmetry, Border Irregularity, Color Variation, Diameter >6 mm) of melanoma serve as the foundation for patient education, but the use of EFGs (Evolving, Elevation, Firmness, Growth) provide a more comprehensive screening method for malignant melanomas. The authors recommend self-skin examinations and that family physicians maintain a high clinical suspicion in high risk patients since early diagnosis with appropriate treatment significantly impacts survival.

INTRODUCTION

The incidence of malignant melanoma is rapidly increasing in the United States. It is estimated that there will be 76,380 reported new cases with 10,130 deaths in 2016 in the U.S.[1] Incidence has increased from 1 in 1500 persons born in the early 1900s to 1 in 50 of Caucasian persons born in 2014, 1 in 200 Hispanics, and 1 in 1000 for African-Americans. Melanoma is the most common cancer among women ages 25-29.[2] There is a strong correlation between sun exposure and development of malignant melanoma. Unlike basal and squamous cell carcinoma, malignant melanoma correlates better with intense intermittent UV radiation exposure.[3] Survival is directly related to early detection with appropriate treatment.[4] A patient's prognosis remains extremely dependent on the stage present when initially diagnosed.[5]

Melanoma is the 5th leading cancer in men and the 7th in women in the United States.[6] Ultraviolet (UV) light exposure is a major risk factor for development of melanoma. Intense sun or tanning bed exposure, as well as exposure to areas not normally exposed to UV radiation, are risk factors. A history of multiple sunburns in childhood also increases risk for development of melanoma. Those with a family history of melanoma or FAMMM Syndrome (Familial Atypical Multiple Mole and Melanoma Syndrome) are also at increased risk.[7] Six risk factors to watch for in the development of malignant melanoma include the following:

- Family history of malignant melanoma
- Blond or red haired individuals
- Freckling on the skin of the upper back
- Three or more sunburns with blistering before age 20 years
- History of an outdoor summer job as a teenager for three or more summers
- Actinic keratosis present

Anyone with one to two of these six factors has a 3.5 times increased risk as compared to the general population. Anyone with three or more of these factors has a risk 20 times that of the general population. Additional risk factors to consider include location of the patient (those that live closer to the Earth's equator are at an increased risk), and people who have a decreased ability to tan. Also note that a history of a prior melanoma automatically increases the risk for development of an additional melanoma at a future time (Table 1).[8,9]

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Classic cutaneous melanoma exists as four main subtypes:

- Superficial spreading melanoma,
- Lentigo maligna melanoma
- Acral lentiginous melanoma
- Nodular melanoma

There is also a rare variant amelanotic melanoma. Superficial spreading melanoma is the most common type, most often seen in young people. Lentigo maligna is the most common type in the elderly. When it gets invasive it is referred to as lentigo maligna melanoma. Acral lentiginous melanoma is most common in African-Americans and Asians. It is found under the nails or on the soles of the feet or palms of the hands. Melanoma of the nail bed is a variant of acral lentiginous melanoma, which includes subungual, ungual and periungual melanoma and can affect any finger or toenail. Nodular melanoma is the most aggressive of the melanomas. It has a rapid growth and most commonly occurs on the trunk and limbs in the 5th or 6th decade, more commonly in males. Nodular melanomas are ulcerated and do not have radial growth; they only have vertical growth.[10] It can present as a darkly pigmented papule which may be polypoid or pedunculated.[11]

Rapidly growing nodular melanomas do not follow the ABCD rule. The EFG rule helps identify these types of melanomas that can go undetected. E refers to elevation and evolving lesion. A mole or lesion that looks different from previous appearance or is changing in size, shape or color.[2] Any lesion that is elevated raises a suspicion and should immediately be referred or biopsied. F refers to firmness- a lesion that feels thick or hard on palpation. G is for any growth, which includes any change in size, shape, color, elevation or new symptom such as bleeding, itching or crusting.[12] These moles do not need to be dark or have any color. They are raised, symmetrical, firm and evolving. It can affect anyone but is more common in men over 50. Rapidly growing nodular melanomas are dangerous because of the very rapid vertical growth.

TABLE 1:

Risk Factors for Melanoma[8,9]

Ultraviolet light exposure	Fair skin
History of atypical moles	Natural light colored hair
History of FAMMM Syndrome	Freckled skin
History of congenital melanocytic nevi	Prior melanoma or other skin cancers
Immunosuppressed status	Male gender
Increasing age	Xeroderma pigmentosum
3 or more sun burns before age 20	Actinic keratosis
Outdoor summer jobs for 3 or more summers	Living closer to the Earth's equator

ILLUSTRATIVE CASE REPORT

A 69-year-old Caucasian male of Irish descent presented for a routine physical examination with no complaints. He had not seen a doctor in 10 years. He had a large facial lesion that was non-painful but pruritic and associated with intermittent bleeding while shaving. Patient stated the lesion had increased in size. He had no history of prior sun exposure or family history of skin cancer. Patient had a five pack years smoking history, denied any alcohol or drug use. Patient was not taking medications. On physical examination the lesion was a 2.5 cm dark pigmented plaque with coalescing center. There was a 3-4 mm thick papule with 2-3 mm satellite lesion (Image 1). A 4-5 mm deep full thickness wedge biopsy was done. Biopsy and pathology showed a 2.4 mm nodular malignant melanoma with ulceration and no metastasis. There was extension into the reticular dermis. The tumor was staged T3b. Nuclear scan and sentinel lymph node biopsy were negative. There was considerable activity and blue staining in the superficial parotid gland. The patient had a wide excision with superficial parotidectomy and local flap closure (Image 2). The patient is being followed closely by oncology. Six months after excision he was found to have a hyperechoic focus in the left occipital area on PET/CT scan. A follow up PET/CT scan six months later showed no abnormalities. It has now been 18 months and the patient is doing well with no current oncological treatment.

IMAGE 1:

Lesion before biopsy

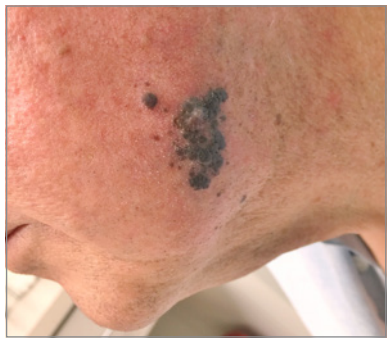
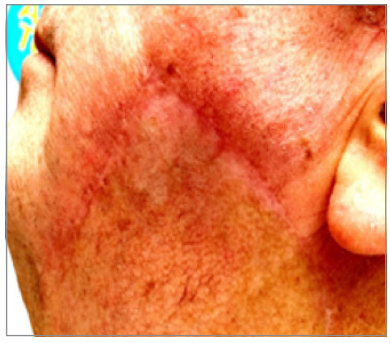


IMAGE 2:

Lesion after flap closure



PROGNOSIS & TREATMENT

Melanomas are aggressive skin cancers that may spread to involve nearly any component of the body. Early diagnosis and appropriate treatment plays a significant role in minimizing morbidity/mortality.[11] Melanoma thickness and extent of metastatic disease are key determinants for the prognosis of a patient with melanoma. Thickness, location of the primary tumor, and sex of the patient are all independent prognostic factors for melanoma. Regarding melanoma of the head/neck areas, a study conducted by the Swedish Melanoma Study Group showed corrected 10-year survival rates to be higher for women at 83%, vs men at 68%, however these were cases of stage I disease. Ten-year survival rates drastically plummeted for patients with higher stage disease with regional and lymph node metastasis.[12]

Thickness of the tumor is the most important factor regarding the prognosis. Ten year survival rate is 92% with thickness at or less than 1 mm, however 10-year survival decreases to 50% for tumors thicker than 4mm. Melanomas have vertical and radial phases of growth. Absence of vertical progression, as in a lack of excessive thickness, indicates a better prognosis with a near zero risk for metastatic disease.[13] Early malignant melanoma presents very similarly regardless of location on the body. Melanomas present as asymmetric, borders are often irregular, typically have obvious color variety present and diameters typically greater than 6 mm (usually larger than a pencil eraser). Benign pigmented lesions are typically round, flat, symmetric, nearly uniform in color, and less than 6 mm in diameter. As a melanoma evolves, its cells will begin to invade from the epidermis into the dermis.

Five-year and 10-year survival rates for melanoma are dependent upon the stage of disease when diagnosed. As tumor thickness increases, survival rate declines.[14] Those at high risk for melanoma based on family history, past medical history, age 50 years or older, sunburn history, as well as presence of atypical nevi or moles, should perform self-examinations of their skin as well as seek dermatologist examination regularly. If the patient has a finding on self-examination, or notes a mole change, he or she should inform their physician as soon as possible.

The TNM (tumor, node, metastasis) staging system relies on examination of the primary tumor, lymph nodes in the region associated with the primary tumor, as well as distant sites of metastasis. Examination of the primary tumor consists of assessment of thickness, mitotic rate, and presence/absence of ulceration at the site.[15] Lymph node assessment is staged based on the extent of regional lymph node involvement determined from immunohistochemical staining. Presence and extent of distant metastatic sites are also incorporated in the TNM staging system. Key findings are summarized in the Table 3.

Imaging and laboratory studies may be necessary to accurately stage prior to initiation of therapy, as well as for follow-up once initial therapy has been instituted. Extensive imaging is not recommended for localized stage I or II primary melanoma due to low yield and high false positive rates.[16,17] Lymphatic mapping with sentinel lymph node biopsy is recommended for intermediate or high risk lymph node metastatic, however not for low risk. Stage III disease warrants obtaining a CBC, LDH, as well as additional imaging prior to lymphadenectomy.[18] To detect and monitor for any recurrences, a physical exam and additional studies should be conducted every 3-12 months. Regarding stage IV disease, MRI of the brain as well as CT Chest/Abdomen/Pelvis are recommended.[19] A PET/CT may also prove useful if surgical resection is being considered when minimal metastasis consisting of a single site is suspected.[18]

A dermatoscope can aid in distinguishing benign from malignant lesions. A dermatoscope is a handheld device with 10x magnification that uses polarized and nonpolarized light to visualize detailed skin structures. There is a two-step process that can aid in the interpretation of dermoscopic structures (Figure 1).[20] The first step is to differentiate melanocytic lesions from nonmelanocytic lesions, which is done by identifying certain structural features in nonmelanocytic lesions. If these structures are not identified, then the lesion should be biopsied. Once it is determined that a lesion is melanocytic the next step is to determine if it is a benign nevus or malignant melanoma.[20] This can be done by a point system or by pattern analysis. There are certain features typical of a nevus and certain melanoma-specific structures that can help determine if a lesion should be biopsied.

Most biopsies are within the scope of family medicine and can be done in the office. The first step is to determine the type of biopsy. Techniques include shave biopsy, punch biopsy and elliptical biopsy (Table 4). There are two types of shave biopsies: tangential and saucerization. Tangential shave biopsy is not appropriate if a lesion might be malignant since this does not include sufficient depth. Saucerization, also known as scallop shave or deep shave biopsy, is used when depth is required and the lesion is suspicious for melanoma. A deep shave biopsy is recommended by the National Comprehensive Cancer Network for this use. Punch biopsy can also be used for suspicious melanocytic lesions. To obtain adequate tissue for pathology a minimum depth of 3-4 mm should be used. To perform a punch biopsy the clinician should apply the cutting surface of the device perpendicular to the skin and press firmly. The punch biopsy unit should be rotated clockwise and counterclockwise until there is a release that indicates penetration into the dermis. The lesion is then removed. The punch site can be closed with sutures or left to heal by secondary intention.[21]

Elliptical excision biopsy can be used for larger lesions. When possible the length of the ellipse is 2 to 3 times the width and the angles of the ellipse should be around 30 degrees. The point of the excision should start at one apex and move along the arc of the other apex. Traction on the surrounding tissue allows for a clean and precise cut. Then the lesion should be lifted with a forceps and removed from the surrounding subcutaneous fat with a scalpel.[21] The biopsy specimen should include the underlying subcutaneous fat for melanomas. The wound is then closed with sutures. Undermining the edges of the wound should be avoided in melanocytic lesions since undermining may disrupt lymphatic flow that can impair the ability to perform sentinel node studies.

TABLE 2:

SURVIVAL RATES FOR MELANOMA[14]
Based on 2008 AJCC Melanoma Staging Database

Stage	5-Year Survival Rate	10-Year Survival Rate
IA	97%	95%
IB	92%	86%
IIA	81%	67%
IIB	70%	57%
IIC	53%	40%
IIIA	78%	68%
IIIB	59%	43%
IIIC	40%	24%
IV	15-20%	10-15%

TABLE 4:

Biopsy Techniques[21]

Type of Biopsy	When to Use	Tools Needed
Tangential Shave	Depth not required	Scalpel blade or razor blade
Saucerization Shave	Depth required Removal of entire lesion Suspicious lesions	RazorBlade
Punch	Suspicious lesions Full thickness required Small skin surface	Disposable or nondisposable units with at least 3-4 mm diameter
Elliptical Excision	Larger lesions Complete excision	Scalpel and forceps

FIGURE 1:

TWO-STEP DERMOSCOPY ALGORITHM[20]
With Permission from Dr. Marghoob.

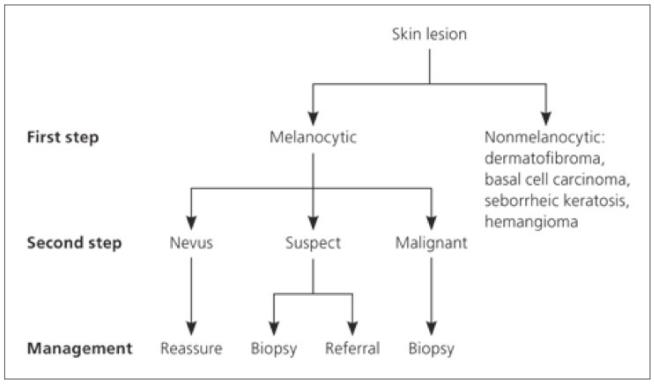


TABLE 3:

2010 AJCC TNM Staging for Cutaneous Melanoma[15]

PRIMARY TUMOR (T)	
TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Melanoma in situ
T1	Less than or equal to 1.0 mm A: Without ulceration & mitoses <1/mm^2 B: With ulceration and mitoses ≥1/mm^2
T2	1.01– 2.0 mm A: Without ulceration B: With ulceration
T3	2.01 - 4.0 mm A: Without ulceration B: With ulceration
T4	> 4.0 mm A: Without ulceration B: With ulceration
REGIONAL LYMPH NODES (N)	
NX	Patients in whom the regional nodes cannot be assessed
N0	No regional metastases detected
N1	One lymph node A: Micrometastases B: Macrometastases
N2	Two or three lymph node A: Micrometastases B: Macrometastases C: In-transit met(s) / satellite(s) without metastatic lymph nodes
N3	Four or more metastatic lymph nodes, or matted lymph nodes, or in-transit met(s)/ satellite(s) with metastatic lymph node(s)
DISTANT METASTASIS (M)	
M0	No detectable evidence of distant metastases
M1a	Metastases to skin, subcutaneous, or distant lymph node, normal serum LDH
M1b	Lung metastases, normal LDH
M1b	Metastasis to other visceral metastases with a normal LDH or any distant metastases and an elevated LDH

PATIENT EDUCATION

Education about melanoma encourages behavioral change in terms of sun exposure and also promotes early detection.[22] Screening and prevention decreases the risk of melanoma. Primary prevention refers to decreasing known risk factors. Secondary prevention refers to early diagnosis and treatment.[10] The main goal of follow-up for patients with a history of melanoma is early detection of recurrent disease and additional primary melanoma. There is no specific follow-up interval. Expert opinion recommends follow up at least annually, with a 3-12 month range based on risk for recurrence. Factors include disease stage, multiple primary melanomas, atypical nevi, family history, and patient's awareness. Patients should be educated about monthly self-examinations. The most important part of follow-up for patients with melanoma is a thorough history and total body physical exam with focus on lymph nodes.

The U.S. Preventative Task Force (USPSTF) concludes that skin cancer screening in asymptomatic adults is a grade I recommendation meaning there is insufficient evidence to assess the benefits and harms of visual skin examination by a clinician. USPSTF recommends counseling children, adolescents, and young adults 10 to 24 years who have fair skin about minimizing their exposure to ultraviolet radiation and gives this a grade B recommendation. For adults older than 24, this is a grade I recommendation. The current evidence is insufficient.[23]

The authors recommend self-skin examinations and maintaining high clinical suspicion in high-risk patients. A thorough self-skin examination requires a well-lit room, in addition to full length, hand held mirrors and a hair dryer (Figure 2).[24] It is necessary to examine areas of skin that are hard to see during self-examination. These areas include portions of the back, scalp, buttocks, and perineal area. A spouse, relative or friend can help with the examination of these areas. The self-skin exam should be carried out step-by-step, and assistance obtained to ensure full viewing of areas difficult to view otherwise.[25]

SUMMARY

The incidence of malignant melanoma is increasing at an alarming rate. Melanoma affects nearly every body part from the eyes to the nail beds. Early diagnosis and treatment requires education by primary care providers to patients. Increased patient education and provider awareness may be able to decrease incidence and mortality. This means maintaining an elevated clinical suspicion in asymptomatic patients. The ABCDs along with the EFGs of melanoma can be used as a guideline and screening for melanomas. A dermatoscope can help in the diagnosing of suspicious melanocytic lesions. Patients with suspicious lesions should have a biopsy or be referred to a dermatologist for additional evaluation. Patients with a personal and/or family history of melanoma require particularly close follow up. By the time a melanoma is palpable it is often too late, therefore early detection is key. Fortunately most melanomas grow slowly and can be detected early to prevent morbidity and mortality.

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REVIEW ARTICLE

Adverse Childhood Experiences: A Call to Action for Osteopathic Medicine

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Keywords:
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It has been nearly 20 years since the first Adverse Childhood Experiences (ACEs) study was published. It is time for the osteopathic profession to embrace these findings – that adversity and trauma in childhood foster ill health in adults and children. We need to champion a well-informed work force of medical providers who practice trauma-informed care. This should be completely intuitive for us, as our credo of mind, body and spirit aligns perfectly with this knowledge.

INTRODUCTION

In 1998, Felitti et al. published the ground breaking article, "Relationship of Childhood Abuse and Household Dysfunction to Many of the Leading Causes of Death in Adults: The Adverse Childhood Experiences (ACE) Study."[1] With this publication and the more than 1,500 published studies that followed,[2] Felitti et al. scientifically substantiated what we have all observed and "known" for years from our own anecdotal observations: when traumatic events happen in people's lives, they experience a multitude of medical, psychological and social ills.[1] However, nearly 20 years later, ACEs continue to be under-addressed in clinical practices, public healthcare, and medical education,[3-5] despite affecting as many as 1 in 8 children.[6] In our current state of exorbitant healthcare costs and an unresolved opioid epidemic, we cannot afford to ignore the connection between ACEs, chronic disease[1] and chronic pain.[7]

As osteopathic physicians and followers of A.T. Still, we are called to enact his entreaty for considering "first the material body, second the spiritual being, third a being of mind which is far superior to all vital motions and material forms, whose duty is to wisely manage this great engine of life. This great principle known as mind, must depend for all evidences on the five senses."[8] In treating the whole person, as we have been trained to do, we already embrace the concept that our patients are a product of their environment (their five senses). Therefore, we are uniquely prepared and perfectly positioned to lead a reform of the practice of medicine in which it becomes routine to consider our patients' trauma histories and practice trauma-informed care throughout their lifespans. Furthermore, we must ensure our student physicians are educated, self-aware and ready from day one to meaningfully interact with patients who have experienced ACEs.

THE IMPACT OF TRAUMA & ACES

The term "trauma" can refer to a wide variety of experiences, ranging from emotional/psychological to strictly physical contexts. The Substance Abuse and Mental Health Services Administration (SAMHSA) Division of the United States Department of Health and Human Services provides a comprehensive definition: "Individual trauma results from an event, series of events, or set of circumstances that is experienced by an individual as physically or emotionally harmful or life threatening and that has lasting adverse effects on the individual's functioning and mental, physical, social, emotional, or spiritual well-being."[9]

In Felitti's study of almost 10,000 adults who answered questions related to seven categories of ACEs (such as abuse, intimate partner violence, substance abuse or mental illness in the home and a household member incarcerated), researchers found that the higher the exposure to these adversities, the greater the risk for serious medical problems and death in the adults (See Figure 1). Fifty-two percent of respondents experienced one or more ACEs and 6.2% experienced four or more. The most common ACE was substance abuse in the home at 25.6%.1 Later studies added three more categories of adverse experience to include loss of a parent and two types of neglect.[2]

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The effect of ACEs can also be dose-dependent. When comparing people who experienced four or more ACEs to those who had none, there was a four- to twelve-fold increase in health risks of substance abuse, depression and suicidality and a two- to four-fold increase in smoking and sexually transmitted infections, to name a few. The authors concluded: "persons with multiple categories of childhood exposure were likely to have multiple health risk factors later in life."[1]

Further study has shown these adverse effects can be observed as early as childhood.[11,12] Flaherty et al.'s study of child health after early childhood adversity looked at over one thousand children at high risk for child abuse and neglect. They found that one adverse exposure almost doubled the risk of poor health while four adverse events or more almost tripled the risk of illness requiring medical attention by the age of six years.[11]

Cronholm and colleagues studied a more socioeconomically and racially diverse urban population[13] – compared to Felitti's study of an insured, not racially diverse population[1] – and created an expanded list of ACEs which included the following: witnessing violence, feeling discrimination, unsafe neighborhood, experiencing bullying and living in foster care. Their study involved almost 1800 respondents, 14% of whom experienced only the expanded ACEs and would not have been recognized as experiencing adversity using conventional ACEs.[13]

These studies addressing general categories of adversity have been shored up by extensive research regarding the mechanism of the toxic effect of ACEs on the developing brain, also referred to as the neurobiological consequences of trauma. These studies are well summarized in the American Academy of Pediatrics (AAP) Technical Report: The Lifelong Effects of Early Childhood Adversity and Toxic Stress.[3] Namely, the plasticity of the young brain makes it sensitive to stress and the chemical influences created by toxic stress which in turn can interrupt normal brain development in a structural way. As an example, glucocorticoid receptors found in the hippocampus, amygdala and prefrontal cortex, when overstimulated, can influence the size and architecture of these areas of the brain with resultant pathologic functioning.[3]

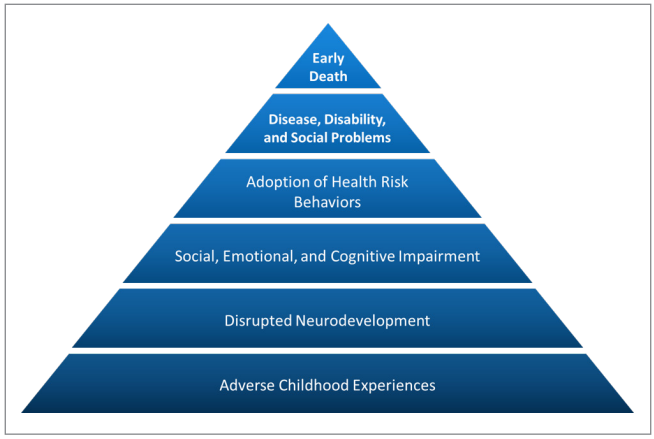
In addition to the degree of impact that ACEs have on individuals, it is striking how prevalent these adversities are. As Felitti et al. discussed, 52% of respondents had one ACE, and one-quarter of respondents experienced substance abuse in the home.[1] Studies on child maltreatment, such as the JAMA Pediatrics article "The Prevalence of Confirmed Maltreatment Among US Children, 2004-2011" by Wildeman et al., suggest that 12.5%, or one in eight children, will experience confirmed maltreatment by the age of 18 years.[6] The deep, lasting impact of ACEs, coupled with their markedly high prevalence, therefore demand significant attention both in our medical practice, and in our training of future medical professionals.

ADDRESSING TRAUMA & ACES

While the prevention of the initial occurrence of ACEs is a worthwhile endeavor[14] that may occur through methods such as home visitation programs[15] and the Safe Environment for Every Kid (SEEK) Model,[16] osteopathic physicians are also in the position of screening for ACEs and addressing their sequelae within our own offices. The identification of ACEs and individuals at high risk for toxic stress naturally leads to supportive efforts for our patients.[17] Furthermore, adults receiving care in overcoming the sequelae of their ACEs may be better able to cope with the stresses of raising children, reducing the trauma their children experience. Therefore, it is imperative to routinely screen patients for ACEs and trauma exposure, whether through non-judgmental, open-ended questions or even brief checklists given in the waiting room.[16]

FIGURE 1:

Proposed mechanism of the relationship between ACEs and health consequences over the lifespan (adapted from Felitti et al.[1] and the CDC).[10]



SAMHSA explains that a trauma-informed care system "realizes the widespread impact of trauma and understands potential paths for healing; recognizes the signs and symptoms of trauma in staff, clients and others involved with the system; and responds by fully integrating knowledge about trauma into policies, procedures, practices, and settings."[9] Machtinger et al., a national strategy group, published an instructive article on the promise of trauma-informed primary care (TIPC) for women in 2015.[18] They recommend that all staff be trauma trained and the physical space be calm, safe and welcoming. Confrontations should be avoided and empowerment should be supported. It is likely that interdisciplinary teams will be necessary to properly progress the healing of these patients. Practices should screen for trauma histories and be prepared in response to disclosures both personally and professionally, being aware of available community programs and safety planning. Machtinger et al. stress the need for a strong organizational foundation with trauma-informed values, partnerships and champions. As always, there should be support for providers and staff as well as monitoring success and quality improvement.[18]

SAMHSA's statement[9] notes that trauma-oriented clinical teams must be self-aware; they must attend to the traumas of not only their patients, but also those experienced by staff members. Therefore, beyond treating our patients and educating our teams, we are called to acknowledge the effects of traumas and ACEs on the members of our healthcare teams and promote self-care, including in physicians and medical students. Educating medical students about understanding, screening, preventing, and intervening in ACEs, and even having them calculate their own scores during their education,5 we may create better prepared, healthier, more resilient and compassionate physicians.

Seriously addressing what we now know about ACEs does not only decrease the number of children affected by a downward trajectory of mental and physical health, but also improves the quality of all of our lives since criminality has been linked to ACEs.[19] A study of 22,575 delinquent youth referred to juvenile justice suggests that "each additional adverse experience…increases the risk of becoming a serious, violent, and chronic juvenile offender by 35."[19] Therefore, addressing ACEs in the clinic may lead to a safer, more productive society.

Even more astounding, the effect of trauma and ACEs can transcend generations. Recent research in epigenetics reveals that our DNA can be altered by stress, and those changes can be passed from one generation to another.[20] However, not every child who experiences trauma will suffer adverse health outcomes, as resilience is a major component in a child's individual ability to cope.[1] There is therefore hope that if we screen for ACEs and provide proper interventions while supporting resilience in our clinics, these changes can be reversed.[21] Once again, Still's faith in the human organism to heal from within is validated.[8]

As stated in the AAP's Technical Report: "this growing scientific understanding about the common roots of health, learning and behavior in the early years of life presents a potentially transformational opportunity for the future of pediatrics."[3] This is true for all systems of care. In addition to being informed and humane, this approach helps clinicians effectively instruct caretakers in trauma-informed behavior modification for children[22] and can help motivate adult patients in need of behavior modification in order to control or improve their disease, and reduce instances of abuse and neglect. The gains for patients and families make the effort to become trauma-informed well worth the effort.[23]

CONCLUSION

We should no longer question nature versus nurture but understand that nurture directly influences nature, whereby childhood trauma can turn on or off genetic predispositions and alter genetic material.[24] Whereas in the past we looked for broken bones, bruises and burns to assess whether children were safe in their homes, this body of research tells us the lasting psychological injury of adverse events impacts children in profound ways. Therefore, we need to redefine what a safe, healthy home looks like and begin to consider early trauma and adverse experiences in our clinical decision-making. The knowledge gained from these studies should inform our professional roles and be incorporated into the education of our medical students. We must add extensive course work on ACEs and trauma recognition, prevention, and treatment to medical curricula, including trauma-informed care, lifestyle medicine and self-care within trauma-informed offices.

Creating trauma-informed medical systems has the potential to help children and adults who receive care in overcoming the sequelae of their ACEs and should be a priority for physicians. This research and the need for trauma-informed care plays to our osteopathic strengths. Let us make the effort and take charge in creating trauma-informed practices in every corner of osteopathic care.

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REVIEW ARTICLE

Osteopathic Approach to Anxiety

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Keywords:
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Psychiatry

Anxiety disorders are one of the most common psychiatric disorders presenting to the family physician. Anxiety disorders are both biologic and psychologic in origin. Anxiety is a signal alerting the individual of 'danger.' This danger can be unknown, internal, conflictual and vague. The anxiety signal allows the individual to respond to, and resolve the 'danger.' This is to be differentiated from fear which is the emotional response to a real or perceived imminent threat. These two states overlap but differ in that fear more often triggers the physiologic response of fight or flight. Anxiety disorders result when one or both of these systems are in a chronic 'hyper reactive' state for either biologic or psychologic reasons. This article reviews the criteria for anxiety disorders and the range of therapeutic interventions, pharmacologic and non-pharmacologic.

INTRODUCTION

Anxiety disorders are one of the most common psychiatric disorders presenting to the family physician. Anxiety disorders are both biologic and psychologic in origin. Anxiety is a signal alerting the individual of 'danger.' This danger can be unknown, internal, conflictual and vague. The anxiety signal allows the individual to respond to, and resolve the 'danger.' This is to be differentiated from fear which is the emotional response to a real or perceived imminent threat.[1] These two states overlap but differ in that fear more often triggers the physiologic response of fight or flight.

Anxiety disorders result when one or both of these systems are in a chronic 'hyper reactive' state for either biologic or psychologic reasons. A brief example is making a presentation to your department at work. This situation is likely to trigger a small degree of anxiety in all individuals which allows the individual to take steps to make sure the presentation goes well such as checking the materials to be presented for accuracy and clarity. Once the materials are reviewed the individual feels confident and successfully completes the presentation. If the anxiety/fear systems are hyper reactive the individual may experience a panic attack with a range of physiologic responses such are rapid heart rate, hyperventilation, and light headedness to name a few. This physiologic response is clearly inappropriate as this is not a life and death situation in which we need the fight and flight response for survival. In this situation the presentation may not occur possibly resulting in negative outcome for the individual.

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Anxiety disorders often start in childhood and must be differentiated from normal childhood worries. Pediatricians and family physicians are familiar with the startle of infants and the fear of monsters in the toddler years. The preschool age child has fears about safety such as being kidnapped or worries about storms, thunder and lightning. These worries may persist into the school age years with the addition of worries related to school performance and social relationships and/or rejection. Fear of bodily harm and illness may arise during this time. Through the teenage years the main worry is about performance, both social and academic.

Life time prevalence of any anxiety disorder in children and adolescents is between 15% and 32%, and the period prevalence (one year or six months) for any anxiety disorder ranges from 3.1% to 18%.2,3 Children with anxiety disorders are at greater risk of developing substance abuse and conduct problems and have increased use of long-term psychiatric and medical services and greater overall functional impairment.[3,4]

One in four adults have been found to have an anxiety disorder. A replication of the National comorbidity study by Kessler, et al. found an 18.1% 12-month prevalence rate for any Diagnostic and Statistical Manual of Mental Disorders IV (DSM IV) anxiety disorder.[5]

EVALUATION & DIAGNOSIS (CRITERIA)

Anxiety disorders overlap but can be differentiated based on the particular presentation of symptoms. Identifying and treating anxiety disorders early can prevent long term morbidity. Mandates for improved mental health screening in the family practice medical home are based on the affordable care act.[6] This includes screening for children, youth, and adults.

Use of validated rating scales in conjunction with the patient interview and examination can assist the busy clinician in evaluating for and following treatment of anxiety. Below are key features of the main anxiety disorders followed by abbreviated Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5) criteria. Validated rating scales that may be used at no cost are referenced under each diagnosis. See the DSM-5 for complete diagnostic criteria.[1]

TREATMENT

Treatment of the patient with an anxiety disorder is based on the evaluation and resultant biopsychosocial formulation for that patient (part of the five model approach to osteopathic patient centered care). This may range from parent guidance for the young child with separation anxiety to aggressive pharmacologic interventions with referral to a mental health specialist or child and adolescent psychiatrist for evaluation and therapy. Osteopathic manipulative medicine should be considered as part of the overall treatment plan for the patient.

In general, Cognitive Behavioral Therapy (CBT) is the treatment of choice for all anxiety disorders, alone or in combination with medications and other interventions.

COGNITIVE BEHAVIORAL THERAPY:

Although cognitive behavioral therapy is typically provided by a therapist for anxiety, depression, and other mental health disorders, the family practice physician can learn the underlying principals and skills necessary to assist their patients achieve improved mental health.

Cognitive behavioral therapy (CBT) was develop by Aaron Beck, MD, a psychoanalyst, in the early 1960s. CBT grew out of Dr. Beck's research on the psychoanalytic theory of depression. His research, which Dr. Beck expected would validate the psychoanalytic therapy of depression, 'anger turned toward the self', did just the opposite. Rather, distorted thoughts and beliefs were the primary feature of depression.[7] Cognitive behavioral therapy assumes that a patient's misconceptions and attitudes about the world and themselves precede and produce symptoms such as anxiety and depression. Therapy identifies habitual ways in which patients distort information (e.g., automatic thoughts) and teaches patients to identify, evaluate, and respond to their dysfunctional thoughts and beliefs, using a variety of techniques to change thinking, mood, and behavior. Cognitive therapy is a structured, goal-oriented, problem-focused, and time-limited intervention. This active approach involving principles of learning, help the patient develop new and adaptive ways of behaving. Treatment also attempts to alter behavior by systematically changing the environment that produces the behavior; such behavioral changes are believed to lead to changes in thoughts and emotions. Beck's treatment model is based on what he terms the cognitive formulation; the beliefs and behavioral strategies that characterize a specific disorder (Alford and Beck, 1997).

The cognitive formulation is an understanding of the patients' problems and an understanding of the patient's thinking related to the problem. This includes the current thinking that contributes to the problem ("I can't lose weight, I'm a failure, I'll always be fat."). The problematic behaviors (drinking a soda rather than water or driving one block to pick up a child rather than walking). And the developmental events or patterns of thinking that predisposed or hold the behaviors causing the problem (developed earlier than many children and was teased, becoming overly self-conscious and critical of self).

The process of treatment is based on the cognitive formulation with the key goal of having the patient identify and change dysfunctional thinking (cognitions).

PSYCHOEDUCATION

For the purpose of this article, psychoeducation refers to the didactic informing of patients and their relatives about the illness, its treatment, and empowerment to handle the illness.[8] Psychoeducation has been shown to be as effective as CBT for youth with anxiety disorders.[9] Goals for psychoeducation include;[8]

- Ensuring patients and their family/relatives have a basic understanding of the illness and treatment
- Empowering the patient and family/relatives to handle the illness
- Helping the patient take on the role of the "expert"
- Strengthen the role of family/relatives
- Information to improve treatment compliance
- Relapse prevention
- Crisis management and prevention
- Support healthy choices

NUTRITION

The osteopathic physician is skilled at communicating the importance of healthy nutrition for physical, mental and emotional health. There is considerable debate on the effect or usefulness of supplements in the treatment of mental health disorders. The support for nutritional supplements is strongest for depressive disorders with more limited support for anxiety disorders. Because depression and anxiety are frequently comorbid it may be helpful to consider the complementary and alternative medicine (CAM) treatments for depression. The most support can be found for the B vitamins, Omega-3 Fatty Acids, and inositol.[10] A review study by Shaheen Lakah, et al.[11] found evidence for the use of herbal supplements containing extracts of passionflower or kava and combinations of L-lysine and L-arginine as treatments for anxiety symptoms and disorders. It should be noted Kava has been removed from the market secondary to concerns for hepatic toxicity.

EXERCISE

As with nutrition, the osteopathic physician is skilled at communicating the importance of exercise for physical, mental and emotional health. Both exercise and yoga have support for their use in anxiety disorders. [12,13]

OSTEOPATHIC INTERVENTIONS

In the five-model approach to osteopathic patient centered care, anxiety disorders fall under the biopsychosocial model. Though there is much research on the effectiveness of cognitive behavioral therapy in anxiety, research in OMT intervention and anxiety is scant. Recent research on the rat model has demonstrated that cannabinoid receptor agonism suppresses anxiety like behavior in rats with essential tremor.[14] We have a body of research theorizing that OMT increases endocannabinoids in the brain through stimulation of the periaqueductal grey matter as well as down-regulates sympathetic stimulation through the Vagus nerve, Cranial Nerve X.[15]

Stephen W Porges, Ph D, discusses a polyvagal theory in vertebrates, in which the action of the autonomic nervous system can vary based on phylogenetic stages of development. These autonomic subsystems are social communication, mobilization and immobilization, and each are used in to provide an adaptive response in safe, dangerous and life-threatening events.[16]

Regardless of the cause, often the ANS becomes activated and never fully return to a pre-traumatic state of functioning, thus anxiety results. We can see this physiologically in the heart rate variability, where low variability is associated with a high sympathetic tone. Yergagani et al found low heart rate variability correlated with emotional disorders in children.[17] This enhanced stress reactivity in pediatric patients' increases all- cause mortality and can be a possible predictor for future cardiovascular events.[18]

Given the correlation of heart rate variability, the best results using OMT would be likely achieve using techniques that affect the heart rate variability. Cervical HVLA has been shown to improve heart rate variability in one study of volunteer patients with neck pain.[19] Osteopathic cranial manipulative medicine has been felt to affect the heart rate variability through upregulation of the parasympathetic nervous system. Proximity to this region is likely a factor for the effectiveness of these techniques. Listed below is a possible treatment regime for the patient with anxiety. Obviously, the physician must use clinical judgment to know if the patient is appropriate to receive an osteopathic treatment, and permission should be obtained prior.

1. Cervical Soft Tissue/long axis kneeding – this technique is performed by having the physician at the head of the table. With fingertips lateral to the spinous processes, the physician uses a superior and lateral pressure on the entire length of the cervical spine.

2. Cervical High Velocity/Low Amplitude – As described above, cervical HVLA, either sidebending or rotational focus, is thought to improve heart rate variability. Somatic dysfunction barrier is engaged in the cervical spine and short quick thrust applied to localized segment at the end of patient.

3. Sacral decompression – Due to the proximity of the parasympathetic nervous system to the sacral region, a simple sidelying sacral decompression in appropriate patients may helpful to decreasing the level of a patients' perceived anxiety. The patient place the thenar and hypothenar eminence over the base of the sacrum at L5 and uses a constant pressure inferior for a period of 1-3 minutes.

FIGURE 1:

Suboccipital/oa decompression

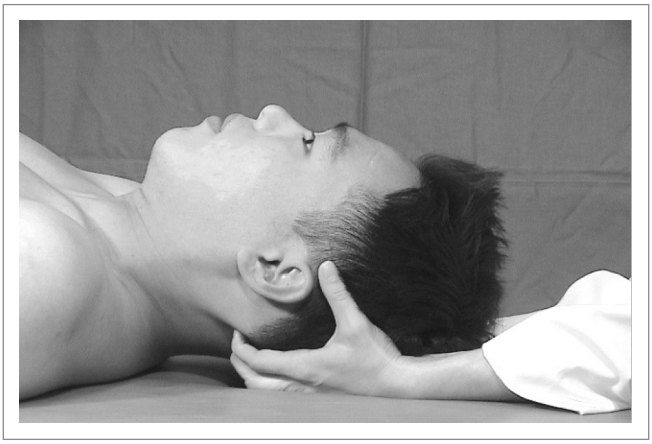
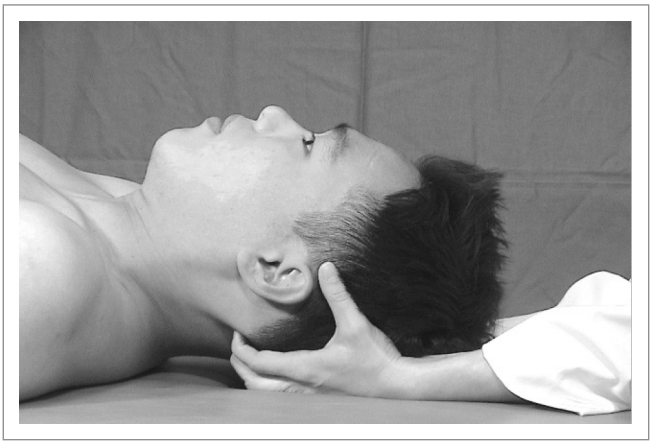


FIGURE 2:

Doming of the thoracic diaphragm



4. Suboccipital/OA decompression- Thought to be beneficial due to the proximity of the Vagus nerve. The patient lies supine on the table while physician placed index and middle fingers in the suboccipital musculature. Anterior pressure is held for a period of 1-3 minutes until relaxation is felt.[20]

5. Doming of the respiratory diaphragm – Patient is in the supine position, physician inserts thumbs under the costal margin but lateral to the xyphoid process and has the patient breath in and out while exerting a superior pressure on the thumbs.

6. Compression of the Fourth Ventricle – An OCMM technique that address the periaqueductal grey area around the fourth ventricle. The physician sits at the head of the supine patient. Physician's hands are placed palmer side up and medial to the occipitomastoid suture. Using thenar eminances on the occiput the physician encourages cranial extension while discouraging cranial flexion until a 'still point' is achieved.[21]

This protocol is just one possible scenario for anxiety which can be performed in approximately 10 minutes at the bedside of the patient, or in the office setting. Additionally, coherence training can be helpful as an adjuctive or take home exercise. Many offices have handheld biofeedback tools, such as the "EmWave" that help the patient with improving heart rate variability. Even without the devices, focused mindful breathing can be very helpful in decreasing anxiety levels and can be used in any setting.

PHARMACOLOGIC MANAGEMENT

Note: Hydroxyzine is only medication that has FDA approval for treatment of children (≥ 6 years) with anxiety. A range of medications are used off label in the treatment of anxiety disorders in children and will be included as supported by the literature.

Separation Anxiety Disorder (ICD 10 code F93.0)

In children the treatment of choice for separation anxiety disorder is CBT with parent guidance and/or Parent-Child Interaction Therapy (PCIT). If medications are considered the SSRI medications are effective and considered the first line pharmacologic treatment. There is evidence for sertraline (25-200mg daily), fluoxetine (20mg daily), and fluvoxamine (50-250 mg daily).[4]

Although there is little research in adults with separation anxiety disorder, CBT and the SSRI medications are considered the treatment of choice.

Social Anxiety Disorder (ICD 10 code F40.10)

All SSRI medications are effective and considered the first line pharmacologic treatment of social anxiety disorder. Dosing strategies for the SSRI medications are the same as for depressive disorders. Venlafaxine 75mg daily (may use higher doses) also demonstrated effectiveness. Buspirone (max dose 60mg/day as bid dosing) has been shown to be effective when used to augment the SSRI medications.[22]

Benzodiazepine (alprazolam 1-6mg daily and clonazepam 0.25-3mg daily) are effective in the treatment of social anxiety at standard doses. Use of Benzodiazepines is considered short term for a period of weeks. The most common clinical mistake utilizing benzodiazepines for treatment is to continue treatment indefinitely.

The MAOI medications including phenelzine (15-90mg daily as tid) have been reported to be effective in cased of severe social anxiety disorder.

Treatment of social anxiety disorder of the performance type can be effective treated with the

beta adrenergic receptor antagonistic medications atenolol (50-100mg about one or two hours prior to the event) and propranolol (20-40mg one or two hours prior to the event) or the benzodiazepines lorazepam and alprazolam.

Panic Disorder (ICD 10 code F41.0)

All SSRI medications are effective and considered the first line pharmacologic treatment of panic disorder. The particular agent chosen is based on the particular effects of the medication such as sedation, activation, and weight gain. Paroxetine is more sedating and calming but also has increased weight gain compared to other SSRI agents. Citalopram, escitalopram, sertraline, and fluvoxamine are next best tolerated. Fluoxetine can be activating and should be started a low dose such as 10mg daily and titrated upward slowly.[22]

Clomipramine and imipramine have demonstrated effectiveness in the treatment of panic disorder. Desipramine (100-200mg daily) has limited evidence. The tricyclic agents are less widely used than the SSRIs because of the increased adverse effects that are seen at the doses needed for clinical response.[22]

MAOI medications including phenelzine (15-90mg daily as tid) and tranylcypromine (30-60mg daily) have data to support their use in panic disorder. The dietary restrictions limit their use.[22]

The atypical antidepressant venlafaxine is effective in the panic disorder but is considered second line treatment to the SSRI medications. Buspirone (max dose 60mg/day as bid dosing) has been suggested as an augmentation to other medications for panic disorder.[22]

If a patient fails to respond to one class of medications changing to another class of medications is suggested.

Alprazolam (3-6mg daily) is a benzodiazepine that is FDA approved for panic disorder. Based on the current trend to avoid the possible longer term complications of the benzodiazepines they have not been included as a primary treatment for panic disorder.[22]

Generalized Anxiety Disorder (ICD 10 code F41.1)

For adults, the benzodiazepines have long been considered the drug of choice for generalized anxiety disorder (GAD) prescribed for a short course or on an 'as needed' basis. Based on the current trend to avoid the possible longer term complications of the benzodiazepines alternative medications such as the SSRI medications, Venlafaxine, and Buspirone are effectively utilized for GAD. As previously noted, the most common mistake in utilizing the benzodiazepines is ongoing, indefinite treatment. The benzodiazepines with an intermediate half- life are typically utilized in GAD (alprazolam, clonazepam, lorazepam).[22]

Buspirone (max 60mg daily) have been suggested to be effective in 60-80% of individuals with GAD. Individuals previously treated with benzodiazepines do not demonstrate this response. Some studies suggest use of benzodiazepines with buspirone as they appear to target different aspects of the anxiety.[22]

Venlafaxine (37.5-225mg daily) has demonstrated effectiveness for GAD.

SSRI medications have demonstrated effectiveness for GAD although few have an FDA indication for this purpose (paroxetine and escitalopram). There is some concern that the SSRI medications may initially increase the level of anxiety. The SSRI medications are often prescribed in conjunction with a 2-3 week course of benzodiazepines.[22]

The beta adrenergic agents such as atenolol and propranolol may be used to address the physiologic response and somatic symptoms of anxiety. These agents do not treat the underlying anxiety disorder.[22]

Post Traumatic Stress Disorder (ICD 10 code F43.10)

Trauma focused cognitive behavioral therapy (TF-CBT) is considered the primary treatment for post-traumatic stress disorder (PTSD). Eye movement desensitization reprocessing (EMDR) is also commonly utilized. Both TF-CBT and EMDR require a clinician with specialized training.

All SSRI medications are effective and considered the first line pharmacologic treatment of PTSD. The particular agent chosen is based on the particular effects of the medication such as sedation, activation, and weight gain. Paroxetine is more sedating and calming but also has increased weight gain compared to other SSRI agents. Citalopram, escitalopram, sertraline, and fluvoxamine are next best tolerated. Fluoxetine can be activating and should be started a low dose such as 10mg daily and titrated upward slowly.[22]

Minipress (1-15mg at bedtime) is effective for the nightmares/sleep disorder associated with PTSD.[23]

The tricyclic agents imipramine (100-200mg daily) and amitriptyline (75-300mg daily) also have clinical data supporting their effectiveness in PTSD. The minimum trial of the tricyclic agents should be eight weeks.[22]

Other medications that may be useful in the treatment of PTSD include phenelzine, trazodone, and the anticonvulsants (e.g., carbamazepine, valproate).[22]

CONCLUSION

Anxiety is a multifactorial disease process which requires an individualized treatment plan for each patient. Treatment may include cognitive behavioral therapy, psychoeducation nutritional support, medications and osteopathic manipulative medicine. When used in conjunction with osteopathic manipulative medicine, the biopsychosocial approach of the five models of osteopathic care will assist in decreasing symptoms and support the treatment of the patient with anxiety.

Separation Anxiety Disorder (ICD 10 code F93.0)

The key feature of Separation Anxiety Disorder is excessive fear concerning separation from home or parents (attachment figures).[1]

ABBREVIATED CRITERIA:

Individuals with separation anxiety disorder have symptoms that meet at least three of the following criteria:

- They experience recurrent excessive distress when separation from home or major attachment figures is anticipated or occurs.
- They worry about the well-being or death of attachment figures, particularly when separated from them, and they need to know the whereabouts of their attachment figures and want to stay in touch with them.
- They also worry about untoward events to themselves, such as getting lost, being kidnapped, or having an accident, that would keep them from ever being reunited with their major attachment figure.
- Individuals with separation anxiety disorder are reluctant or refuse to go out by themselves because of separation fears.
- They have persistent and excessive fear or reluctance about being alone or without major attachment figures at home or in other settings.
- They have persistent reluctance or refusal to go to sleep without being near a major attachment figure or to sleep away from home.
- Children with this disorder often have difficulty at bedtime and may insist that someone stay with them until they fall asleep.
- Physical symptoms (e.g., headaches, abdominal complaints, nausea, vomiting) are common in children when separation from major attachment figures occurs or is anticipated.
- The symptoms must last a period of at least four weeks in children and adolescents and typically six months or more in adults.

Prevalence (12 month prevalence):

Child:	4%
Adolescent:	1.6
Adult:	0.9-1.9%
Male: Female:	1:1

Child/Adolescent Rating Scale:

Screen for Child Anxiety Related Emotional Disorders (SCARED); parent rating and self-rating (children 8-11 y/o)

Adult Rating Scale:

Adult Separation Anxiety Questionnaire - ASA-27

Separation Anxiety Disorder (ICD 10 code F93.0)

The key feature of social anxiety disorder is a marked or intense fear/anxiety of social situations in which they may be scrutinized by others.[1]

ABBREVIATED CRITERIA:

- Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others. Note: In children, the anxiety must occur in peer settings and not just during interactions with adults.
- The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (i.e., will be humiliating or embarrassing; will lead to rejection or offend others).
- The social situations almost always provoke fear or anxiety. Note: In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, shrinking, or failing to speak in social situations.
- The social situations are avoided or endured with intense fear or anxiety.
- The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context.
- The fear, anxiety, or avoidance is persistent, typically lasting for six months or more.
- The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- The fear, anxiety, or avoidance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
- The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder, such as panic disorder, body dysmorphic disorder, or autism spectrum disorder.
- If another medical condition (e.g., Parkinson's disease, obesity, disfigurement from burns or injury) is present, the fear, anxiety, or avoidance is clearly unrelated or is excessive.

Prevalence (12 month prevalence):

Child:	7%
Adolescent:	7%
Adult:	7%
Geriatric:	2-5%
Male:Female	1:1

Child/Adolescent Rating Scale:

Screen for Child Anxiety Related Emotional Disorders (SCARED); parent rating and self-rating (children 8-11 y/o)

Adult Rating Scale:

Adult Separation Anxiety Questionnaire - ASA-27

Social Anxiety Disorder (ICD 10 code F40.10)

The key feature of social anxiety disorder is a marked or intense fear/anxiety of social situations in which they may be scrutinized by others.[1]

ABBREVIATED CRITERIA:

- Marked fear or anxiety about one or more social situations in which the individual is exposed to possible scrutiny by others. Note: In children, the anxiety must occur in peer settings and not just during interactions with adults.
- The individual fears that he or she will act in a way or show anxiety symptoms that will be negatively evaluated (i.e., will be humiliating or embarrassing; will lead to rejection or offend others).
- The social situations almost always provoke fear or anxiety. Note: In children, the fear or anxiety may be expressed by crying, tantrums, freezing, clinging, shrinking, or failing to speak in social situations.
- The social situations are avoided or endured with intense fear or anxiety.
- The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context.
- The fear, anxiety, or avoidance is persistent, typically lasting for six months or more.
- The fear, anxiety, or avoidance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- The fear, anxiety, or avoidance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition.
- The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder, such as panic disorder, body dysmorphic disorder, or autism spectrum disorder.
- If another medical condition (e.g., Parkinson's disease, obesity, disfigurement from burns or injury) is present, the fear, anxiety, or avoidance is clearly unrelated or is excessive.

Prevalence (12 month prevalence):

Child:	7%
Adolescent:	7%
Adult:	7%
Geriatric:	2-5%
Male:Female	1:1

Child/Adolescent Rating Scale:

Screen for Child Anxiety Related Emotional Disorders (SCARED); parent rating and self-rating (children 8-11 y/o)

Adult Rating Scale:

Severity Measure for Social Anxiety Disorder (Social Phobia)—Adult

Panic Disorder (ICD 10 code F41.0)

The key feature is a recurrent and abrupt surge of intense fear or discomfort (panic attack) lasting minutes during which at least four of 13 physical and cognitive symptoms occur.[1]

ABBREVIATED CRITERIA:
Physical and cognitive symptoms:

- Palpitations, pounding heart, or accelerated heart rate.
- Sweating.
- Trembling or shaking.
- Sensations of shortness of breath or smothering.
- Feelings of choking.
- Chest pain or discomfort.
- Nausea or abdominal distress.
- Feeling dizzy, unsteady, light-headed, or faint.
- Chills or heat sensations.
- Paresthesias (numbness or tingling sensations).
- Derealization (feelings of unreality) or depersonalization (being detached from oneself).
- Fear of losing control or "going crazy."
- Fear of dying.

At least one of the attacks has been followed by one month (or more) of one or both of the following:

- Persistent concern or worry about additional panic attacks or their consequences (e.g., losing control, having a heart attack, "going crazy").
- A significant maladaptive change in behavior related to the attacks (e.g., behaviors designed to avoid having panic attacks, such as avoidance of exercise or unfamiliar situations).

Prevalence (12 month prevalence):

Child:	< 0.4%
Adolescent:	2 - 3%
Adult:	2 - 3%
Male:Female	1:2

Child/Adolescent Rating Scale:

Screen for Child Anxiety Related Emotional Disorders (SCARED); parent rating and self-rating (children 8-11 y/o)

Adult Rating Scale:

Severity Measure for Panic Disorder - Adult

Generalized Anxiety Disorder (ICD 10 code F41.1)

The key feature of generalized anxiety disorder is excessive anxiety and about a number of events or activities that are difficult to control and interfere with psychosocial functioning.[1]

ABBREVIATED CRITERIA:

- Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).
- The individual finds it difficult to control the worry.
- The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past 6 months): Note: Only one item is required in children.
  - Restlessness or feeling keyed up or on edge.
  - Being easily fatigued.
  - Difficulty concentrating or mind going blank.
  - Irritability.
  - Muscle tension.
  - Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep).
- The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Prevalence (12 month prevalence):

Child:	0.2 - 3.6%
Adolescent:	0.2 - 3.6%
Adult:	2.9%
Male:Female	1:2

Child/Adolescent Rating Scale:

Screen for Child Anxiety Related Emotional Disorders (SCARED); parent rating and self-rating (children 8-11 y/o)

Adult Rating Scale:

Hamilton Anxiety Rating Scale (HAM-A)
Generalized Anxiety Disorder Assessment (GAD 7)

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Post Traumatic Stress Disorder (ICD 10 code F43.10)

The key feature of post traumatic stress disorder (PTSD) is the development of characteristic symptoms following exposure to one or more traumatic events. The clinical presentation of PTSD varies. For children six years and younger, see the adapted criteria in the DSM-5 Text.[1]

ABBREVIATED CRITERIA:

A) Exposure to actual or threatened death, serious injury, or sexual violence in one (or more) of the following ways:

- Directly experiencing the traumatic event(s).
- Witnessing, in person, the event(s) as it occurred to others.
- Learning that the traumatic event(s) occurred to a close family member or close friend. In cases of actual or threatened death of a family member or friend, the event(s) must have been violent or accidental.
- Experiencing repeated or extreme exposure to aversive details of the traumatic event(s) (e.g., first responders collecting human remains; police officers repeatedly exposed to details of child abuse).
- Note: Criterion A4 does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.

B) Presence of one (or more) of the following intrusion symptoms associated with the traumatic event(s), beginning after the traumatic event(s) occurred:

- Recurrent, involuntary, and intrusive distressing memories of the traumatic event(s).
- Note: In children older than 6 years, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.
- Recurrent distressing dreams in which the content and/or affect of the dream are related to the traumatic event(s). Note: In children, there may be frightening dreams without recognizable content.
- Dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring. (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings. Note: In children, trauma-specific reenactment may occur in play.
- Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).
- Marked physiological reactions to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).

C) Persistent avoidance of stimuli associated with the traumatic event(s), beginning after the traumatic event(s) occurred, as evidenced by one or both of the following:

- Avoidance of or efforts to avoid distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).
- Avoidance of or efforts to avoid external reminders (people, places, conversations, activities, objects, situations) that arouse distressing memories, thoughts, or feelings about or closely associated with the traumatic event(s).

D) Negative alterations in cognitions and mood associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

- Inability to remember an important aspect of the traumatic event(s) (typically due to dissociative amnesia and not to other factors such as head injury, alcohol, or drugs).
- Persistent and exaggerated negative beliefs or expectations about oneself, others, or the world (e.g., "I am bad," "No one can be trusted," "The world is completely dangerous," "My whole nervous system is permanently ruined").
- Persistent, distorted cognitions about the cause or consequences of the traumatic event(s) that lead the individual to blame himself/herself or others.
- Persistent negative emotional state (e.g., fear, horror, anger, guilt, or shame).
- Markedly diminished interest or participation in significant activities.
- Feelings of detachment or estrangement from others.
- Persistent inability to experience positive emotions (e.g., inability to experience happiness, satisfaction, or loving feelings).

E) Marked alterations in arousal and reactivity associated with the traumatic event(s), beginning or worsening after the traumatic event(s) occurred, as evidenced by two (or more) of the following:

- Irritable behavior and angry outbursts (with little or no provocation) typically expressed as verbal or physical aggression toward people or objects.
- Reckless or self-destructive behavior.
- Hypervigilance.
- Exaggerated startle response.
- Problems with concentration.
- Sleep disturbance (e.g., difficulty falling or staying asleep or restless sleep).

Duration of the disturbance (Criteria B, C, D, and E) is more than one month.

Prevalence (12 month prevalence):

Child:	None quoted	
Adolescent:	5% Lifetime	
Adult:	3.5%	
Male:Female	1:2 (Adult)	1:4 (Adolescent)

Child/Adolescent Rating Scale:

Child PTSD Symptom Scale (CPSS); (ages 8-18 years)
Trauma Symptom Check List (TSCC); copyright PAR requires purchase (8-16 years)

Adult Rating Scale:

Primary Care PTSD Screen (PC-PTSD)

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CLINICAL IMAGES

Roseola

Elise Hyser OMS IV & Lindsay Tjiattas-Saleski DO, MBA, FACOEP

[1]Edward Via College of Osteopathic Medicine – Carolinas
[2]Palmetto Health Tuomey Medical Center, Sumter, South Carolina

A 13-month-old black female presented to her family practice office with a four-day history of fever, nasal congestion, and loose stools. Her maximum temperature was 103.9 degrees Fahrenheit, obtained rectally. Symptoms accompanying the fever included diminished appetite, irritability, and malaise. The child is otherwise healthy and up-to-date on immunizations. She resides at home with her parents and was enrolled in daycare one month ago. On the fifth day of the illness, the child's fever subsided. She then developed an erythematous maculopapular rash restricted to the trunk, sparing the palms, soles, and oral mucosa.

FIGURE 1:
Frontal abdominal view

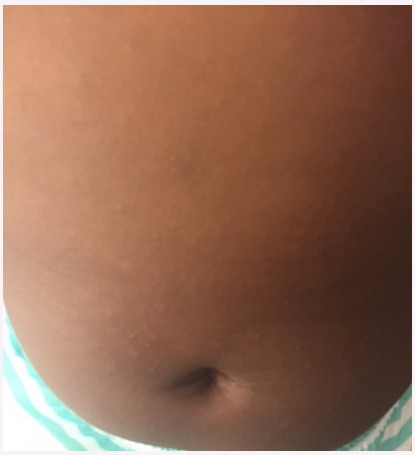
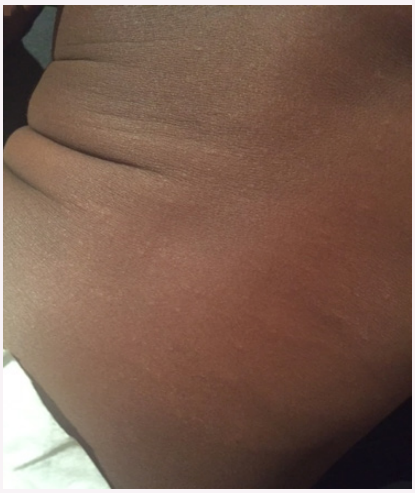


FIGURE 2:
Lateral flank view



QUESTIONS:

What is the most likely diagnosis?

a) Kawasaki disease
b) Measles
c) Roseola
d) Rubella
e) Scarlet fever

What is the primary viral culprit of roseola?

a) HSV-1
b) HHV-3
c) HHV-4
d) HHV-5
e) HHV-6

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ANSWERS

What is the most likely diagnosis?

The correct answer is:

e) Roseola

Roseola is a benign, self-limited virus caused by HHV-6 that typically presents as fevers between 102 and 105°F (38.9–40.6°C) followed by defervescence and subsequent development of a diffuse maculopapular rash.[1,2] The differential diagnosis of roseola consists of measles, rubella, scarlet fever, enteroviruses, adenoviruses, Epstein-Barr virus, and Kawasaki disease.[1,2] The measles virus is characterized by fever, cough, coryza, conjunctivitis, Koplik's spots (white spots on the buccal mucosa) and an erythematous maculopapular rash that classically starts on the face and spreads to the trunk, arms, and legs.[3] Rubella presents with a macular rash spreading from the head to the trunk, arms, and legs, accompanied by mild fever, malaise, posterior lymphadenopathy, and arthralgia.[4] Kawasaki disease is a common pediatric vasculitis characterized by a fever of at least five days plus four of the following: bilateral conjunctival injection, oropharyngeal erythema with fissuring of the lips and strawberry tongue, erythema and edema of the hands and feet with periungual desquamation, an erythematous rash, and cervical lymph node enlargement to >1.5 cm in diameter.[5] Scarlet fever is a viral exanthem that follows infection with streptococcal pyrogenic exotoxins from tonsillitis or pharyngitis.[2] A sore throat, high fever, headache, malaise, chills, and anorexia precedes the development of a sandpaper-like rash by 12-48 hours that spreads from the neck, chest, and axillae down to the trunk and extremities.[2]

What is the primary viral culprit of roseola?

The correct answer is:

e) HHV-6

HSV-1, Herpes Simplex Virus 1, causes an orolabial herpetic infection, otherwise known as cold sores.[1,2] HHV-3, Varicella Zoster Virus (a vesicular viral exanthem), causes chickenpox, and when reactivated, Herpes Zoster.[2,6] HHV-4, Epstein-Barr Virus, causes infectious mononucleosis.[2,7] It is also associated with lymphoproliferative disorders.[2,7] HHV-5, cytomegalovirus, is a member of the group of TORCH infections and can cause congenital deafness and mental retardation.[2,6] HHV-6 is the virus that causes roseola.

DISCUSSION

Roseola infantum, also referred to as exanthema subitum and sixth disease, is a clinical syndrome detected in early infancy and childhood primarily caused by infection with Human herpes virus 6 of the subfamily betaherpesvirinae and genus Roseolovirus.[1,2] While HHV6 is the most common cause, in 1994 HHV7 was also discovered to be a second culprit.8 Both infect lymphocytes, have a predilection for T cells and can establish lifelong latency in these cells. The virus commonly infects infants and children between the ages of six months and three years and targets males and females equally.[9,10] Epidemics tend to have an incubation period of 9-10 days and the transmission of roseola occurs via infectious respiratory secretions, such as saliva.[2,5,8]

The diagnosis of roseola is predominantly clinical, revealed by the classic pattern of a 3-5 day fever, defervescence and subsequent development of a viral exanthem in a well appearing child.[1,7,9] The rash, which typically originates on the trunk and spreads peripherally to the extremities, is small, blanchable, generally non-pruritic rose-pink macules and maculopapules that are 2-5 mm in diameter with a peripheral halo of vasoconstriction.[1,2,5,7,9,11] Periorbital edema, cervical lymphadenopathy, and upper respiratory tract infection, when present, often mark the preexanthematous stage.[1,5,7] The exanthem itself appears asymptomatically and either lasts 1-2 days or occasionally persists for 2-4 hours.[1,7,12] Mild coryza, headache, and abdominal pain may also be present in the clinical history.[1,7,12] As the disease progresses, two-thirds of patients may develop Nagayama spots, erythematous papules localized to the mucosa of the soft palate and uvula.[1,2,7] One study examined 176 infants with an established diagnosis of exanthem subitum and confirmed infection with HHV-6.12 that 98 % of subjects had a fever, 98 % had a macular or papular rash, 68 % had mild diarrhea, 30 % had edematous eyelids, 65 % showed erythematous papules in the pharynx, 50 % had a cough, 31 % developed cervical lymph node swelling, 26 % showed swelling of the anterior fontanelle, and 8% had convulsions.[12]

While lab values are not typically used for the diagnosis of roseola, the gold standard for diagnosis of an HHV-6 infection is laboratory evidence of seroconversion in paired sera with a rise in anti-HHV IgM demonstrated in serum samples during the first week and conversion to anti-HHV IgG two weeks later.[2,8]

The differential diagnosis for roseola includes, but is not limited to measles, scarlet fever, Epstein-Barr virus, rubella, and Kawasaki disease.[1,2] Measles rash typically begins on the face or mouth (in the form of Koplik spots) and spreads cephalocaudally or centrifugally to reach the neck, trunk and extremities.[11] Children with measles appear more ill than those with roseola.[11] Scarlet fever is a condition diagnosed in 10% of cases of streptococcal tonsillopharyngitis marked by fever and sore throat, preceding a rash of sandpaper-like papules that starts on the trunk and spreads throughout the body.[11] In scarlet fever the palms and soles are spared, a circumoral pallor is observed, and the rash develops slowly.[11] Pastia lines, red non-blanching linear rash that appears in skinfolds, distinguish it from other rashes.[11] EBV-induced mononucleosis can also produce a rash, but it is typically associated with exudative tonsillitis, a hyperemic oropharynx, and palatal petechiae.[2] While it may resemble other exanthems, the rash associated with EBV often follows the ingestion of amoxicillin in a patient suspected of having strep throat.[13] The macular rash associated with rubella spreads caudally and involves lymphadenopathy of the posterior cervical, suboccipital, and posterior auricular lymph nodes.[2] Kawasaki disease, a rare cause of pediatric rash, presents as a polymorphic exanthem coupled with mucocutaneous findings and is a clinical diagnosis.[2] Diagnostic criteria for Kawasaki disease include fever for a minimum of five days in addition to four of the following characteristic features: bilateral conjunctival injection, a polymorphous rash, changes in the mucous membranes of the oral cavity (such as fissuring of the lips or strawberry tongue), changes in the peripheral extremities, cervical lymphadenopathy of greater than or equal to 1.5 cm in diameter, and exclusion of diseases that present with similar manifestations.[4]

Roseola is generally a benign, self-limited illness with an uncomplicated course, requiring symptomatic treatment.[10] Antipyretics such as acetaminophen or ibuprofen can be administered to relieve the fever.[3,5,10] Immunosuppressed patients are more susceptible than the general population to reactivation of HHV-6 and HHV-7, the major viruses that cause roseola.[8] A more severe clinical course can follow viral reactivation, which may be marked by rare, yet serious sequelae such as encephalitis, meningitis, multiple sclerosis, myocarditis, chronic fatigue syndrome, giant cell hepatitis, and pneumonitis.[8] Immunocompromised patients may be treated with antiviral therapy to avoid a more deleterious clinical course, but no randomized clinical trials exist to validate a recommendation on this.[2,14] Since the viral sensitivity profile of HHV-6 bears striking similarity to that of CMV, ganciclovir and foscarnet can be used for such cases, but specific treatments are still being studied.[7,14] Febrile seizures, which constitute the most prevalent complication of roseola, occur 10-15 percent of the time.[2,5,7]

This patient's symptoms resolved after several days. Her four-day fever followed by a maculopapular rash migrating from the trunk to the proximal extremities is consistent with a typical clinical presentation of roseola. She was treated symptomatically. The patient did not suffer any known complications and had no scarring from the rash. When considering roseola as a diagnosis, the clinician should always maintain a high clinical suspicion for other pediatric rashes of more serious etiologies. The challenge faced by physicians in the future will be to engage in discussion with patients about the viral etiology of roseola and to curb misuse of antibiotics.

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MAY / JUNE 2017 ANSWERS: 1. C 2. A 3. D 4. B 5. C 6. D 7. B 8. B 9. A 10. C

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SKIN CANCER

Peter Zajac, DO, FACOFP, Author

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Skin cancer is the most common form of cancer in the United States. There are two main kinds of skin cancer: Melanoma (deadliest type of skin cancer) and Non-Melanoma (e.g. Basal Cell and Squamous Cell). Risk factors for skin cancer include a family or personal history of skin cancer, tendency to freckle or burn easily, many sunburns as a child, a lot of sun exposure throughout your life, exposure to certain chemicals, Human papilloma virus (HPV), chronic wounds, radiation therapy, or use of drugs that affect the immune system. Performing monthly full-body skin self-exams in front of a full-length mirror is the best way to detect possible skin cancer. You should check for unusual skin colors, abnormal appearing moles, or other suspicious skin changes. Ask your spouse or a family member to check hard to see areas.

PREVENTATIVE MEASURES INCLUDE:

- Apply a sunscreen to dry skin 15 minutes before going outdoors. Water-resistant sunscreens with a Sun Protection Factor (SPF) rating of 30 or greater help to protect the skin from sunburn, early skin aging, and skin cancer.
- Reapply a sunscreen every two hours or after swimming or sweating heavily to all exposed skin.
- Wear sun protective clothing and avoid sun exposure from 10 AM to 3 PM year around when rays are strongest.
- Exercise extra caution near sand, snow, or water as they reflect the damaging rays of the sun that can increase the chance of sunburn. Even on cloudy days, up to 80% of the sun's harmful ultraviolet (UV) rays can enter the skin.
- Avoid tanning beds. UV light from tanning beds also can cause skin cancer and wrinkling. If you want to look tan, consider using a self-tanning product along with a sunscreen.
- Skin cancer can form on the lips. Use a lip balm that contains sunscreen with a SPF of 15 or higher.
- Avoid sun exposure and do not use sunscreens on infants younger than 6 months of age.
- Use UV-protective sunglasses and select hats with front and back flaps.
- Seek shady areas to provide protection from the sun's rays as well.
- If you are taking antibiotics or other medications, ask your doctor if it may increase your skin's sensitivity to the sun.

MEDICAL CARE & TREATMENT OPTIONS:

If you develop sunburn or notice anything changing, growing, or bleeding on the skin please call your Osteopathic Family Physician. Skin cancer is treatable when caught early. In case of any emergency, you should call your doctor or 911 right away.

SOURCE(S): ***American Academy of Dermatology, Centers for Disease Control and Prevention, and Skin Cancer.gov.***

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