AGENDA

THE MEETING OF THE

DISABILITY PROCEDURES AND SERVICES COMMITTEE
and
BOARD OF RETIREMENT*

LOS ANGELES COUNTY EMPLOYEES RETIREMENT ASSOCIATION

300 NORTH LAKE AVENUE, SUITE 810
PASADENA, CA 91101

9:00 A.M., WEDNESDAY, July 6, 2016 **

The Committee may take action on any item on the agenda, and agenda items may be taken out of order.

COMMITTEE MEMBERS:

Vivian H. Gray, Chair
William de la Garza, Vice Chair
Yves Chery
Les Robbins
David Muir, Alternate

I. APPROVAL OF THE MINUTES

A. Approval of the minutes of the regular meeting of June 1, 2016.

II. PUBLIC COMMENT

III. ACTION ITEMS

A. Consider application of Michael M. Bronshvag, M.D., as a LACERA Panel Physician.

IV. FOR INFORMATION

V. GOOD OF THE ORDER

(For information purposes only)

VI. ADJOURNMENT

***The Board of Retirement has adopted a policy permitting any member of the Board to attend a standing committee meeting open to the public. In the event five (5) or more members of the Board of Retirement (including members appointed to the Committee) are in attendance, the meeting shall constitute a joint meeting of the Committee and the Board of Retirement. Members of the Board of Retirement who are not members of the Committee may attend and participate in a meeting of a Board Committee but may not vote on any matter discussed at the meeting. The only action the Committee may take at the meeting is approval of a recommendation to take further action at a subsequent meeting of the Board.**

****Although the meeting is scheduled for 9:00 a.m., it can start anytime thereafter, depending on the length of the Board of Retirement meeting. Please be on call.**

Assistive Listening Devices are available upon request. American Sign Language (ASL) Interpreters are available with at least three (3) business days notice before the meeting date.

Any documents subject to public disclosure that relate to an agenda item for an open session of the Committee, that are distributed to members of the Committee less than 72 hours prior to the meeting, will be available for public inspection at the time they are distributed to a majority of the Committee, at LACERA's offices at 300 North Lake Avenue, suite 820, Pasadena, California during normal business hours from 9:00 a.m. to 5:00 p.m. Monday through Friday.

Persons requiring an alternative format of this agenda pursuant to Section 202 of the Americans with Disabilities Act of 1990 may request one by calling the Disability Retirement Services Division at 626-564-2419 from 7:30 a.m. to 5:00 p.m. Monday through Friday, but no later than 48 hours prior to the time the meeting is to commence.

MINUTES OF THE MEETING OF THE

DISABILITY PROCEDURES AND SERVICES COMMITTEE
and
Board of Retirement**

LOS ANGELES COUNTY EMPLOYEES RETIREMENT ASSOCIATION

GATEWAY PLAZA - 300 N. LAKE AVENUE, SUITE 810, PASADENA, CA 91101

Wednesday, June 1, 2016, 10:31 A.M. – 10:41 A.M.

COMMITTEE MEMBERS

PRESENT:	Vivian H. Gray, Chair William de la Garza, Vice Chair Yves Chery Les Robbins David Muir, Alternate
ABSENT:	None

ALSO ATTENDING:

BOARD MEMBERS AT LARGE

Anthony Bravo
Shawn R. Kehoe
William Pryor
Vito M. Campese, M.D.

STAFF, ADVISORS, PARTICIPANTS

Gregg Rademacher	Ricki Contreras	Darren Huey
JJ Popowich	Vickie Neely	Shamila Freeman
Steven Rice	Tamara Caldwell	Hernan Barrientos
Vincent Lim	Anna Kwan	Ricardo Salinas
Eugenia Der	James Pu	Ruby Minjares
Allison E. Barrett	Debbie Semnanian	Nichelle Porter
Frank Boyd	Mario Garrido	Danny Hang
Sandra Cortez	Debra Martin	Robert Hill
Angie Guererro	Marco Legaspi	Mike Herrera
Maria Muro	Marilu Bretado	Barbara Tuncay
Maisha Coulter	Karla Sarni	
Michelle Yanes	Thomas Wicke	

ATTORNEYS
Thomas J. Wicke

GUEST SPEAKER
None

The meeting was called to order by Chair Gray at 10:31 a.m.

I. APPROVAL OF THE MINUTES

A. Approval of minutes of the regular meeting of May 5, 2016

Mr. Chery made a motion, Mr. de la Garza seconded, to approve the minutes of the regular meeting of May 5, 2016. The motion passed unanimously.

II. PUBLIC COMMENT

III. ACTION ITEMS

A. Consider application of Noam Drazin, M.D., as a LACERA Panel Physician.

Mr. de la Garza made a motion, Mr. Chery seconded, to approve to accept staff's recommendation and submit the application of Noam Drazin, M.D. to the Board of Retirement for approval to the LACERA Panel of Examining Physicians. The motion passed unanimously.

B. Consider application of Jonathan T. Nassos, M.D. as a LACERA Panel Physician.

Mr. Chery made a motion, Mr. Robbins seconded, to approve to accept staff's recommendation and submit the application of Jonathan T. Nassos, M.D. to the Board of Retirement for approval to the LACERA Panel of Examining Physicians. The motion passed unanimously.

Going forward, Mr. Kehoe requested the Physician's Specialty be included in the cover memo addressed to the Disability Procedures and Services Committee. Ms. Gray concurred with Mr. Kehoe on the request. Staff acknowledged the request.

IV. FOR INFORMATION

V. GOOD OF THE ORDER

Mr. Robbins wanted to commend staff for their hard work regarding a LACERA member who called Mr. Robbins regarding an issue with his Disability Retirement. Mr. Robbins stated that the issue had nothing to do with LACERA's procedures but rather the law and staff handled it well. Ms. Gray also thanked staff for handling this well.

Mr. Muir asked if Dr. Hannani should be reviewed as a Panel Physician due to concerns with his reports. Mr. Boyd responded by saying that he did have a conversation with Dr. Hannani regarding some concerns and Dr. Hannani agreed to comply with the needs of staff and to turn in more detailed reports.

VI. ADJOURNMENT

With no further business to come before the Disability Procedures and Services Committee, the meeting was adjourned at 10:41 a.m.

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June 20, 2016

TO: Disability Procedures & Services Committee
Vivian H. Gray, Chair
William de la Garza, Vice Chair
Yves Chery
Les Robbins
David Muir, Alternate

FROM: Ricki Contreras, Manager
Disability Retirement Services

FOR: July 6, 2016, Disability Procedures and Services Committee Meeting

SUBJECT: **CONSIDER APPLICATION OF INTERNIST/NEUROLOGIST, MICHAEL M. BRONSHVAG, M.D., AS A LACERA PANEL PHYSICIAN**

On June 7, 2016, Debbie Semnanian interviewed Michael M. Bronshvag, M.D., a physician seeking appointment to the LACERA Panel of Examining Physicians.

Attached for your review and consideration are:
- Staff's Interview Summary and Recommendation
- Panel Physician Application
- Curriculum Vitae
- Sample Report(s).

IT IS THEREFORE RECOMMENDED THAT THE COMMITTEE accept the staff recommendation to submit the application of Michael M. Bronshvag, M.D., to the Board of Retirement for approval to the LACERA Panel of Examining Physicians.

Attachments

JJ:RC/mb

NOTED AND REVIEWED:

JJ Popowich, Assistant Executive Officer

Date: 6/24/16

June 21, 2016

TO: **Ricki Contreras, Manager**
Disability Retirement Services

FROM: **Debbie Semnanian** DS
Disability Retirement Specialist Supervisor

Debbie Semnanian, WCCP
Supervising Disability Retirement Specialist

SUBJECT: **INTERVIEW OF INTERNIST/NEUROLOGIST APPLYING FOR LACERA'S PHYSICIAN'S PANEL**

On June 7, 2016, I interviewed **Michael M. Bronshvag, M.D.** at his office location at 15350 Sherman Way, Suite 250, Van Nuys, CA 91406. The office space is located in a well-maintained five-story building with patient paid parking on the ground floor of the building.

Dr. Bronshvag is Board Certified in both internal medicine and neurology, and has been in private practice for forty years. Dr. Bronshvag's office has three examination rooms. He estimates that 5 percent of his practice is devoted to patient treatment, while the other 95 percent of his time is devoted to IME evaluations for other retirement systems and for QTC (Quality Timely Cost-Effective Medical Examinations – a private provider of government occupational health and disability examination services) for federal issues.

As referenced in his Curriculum Vitae, Dr. Bronshvag graduated from Columbia University College of Physicians and Surgeons in New York, with his Medical Degree in 1964. He completed a residency in internal medicine at Northwestern University Medical Center, and a residency in neurology at Letterman General Hospital in San Francisco. Dr. Bronshvag served in the U.S. Army, where he held the position of US-IRR (Colonel) from 1987-1996, and retired in 2000. Dr. Bronshvag advised staff that, throughout his practice, fifty percent of his examinations have been for patients with internal conditions and 50% for patients with neurologic conditions.

The office was clean with ample seating. A handicap accessible restroom is located within the office. There is a staff of two office personnel.

Staff reviewed the LACERA Disability Retirement procedures and expectations in its evaluation of County Employees applying for both service connected and non-service connected disability retirements. The importance of preparing impartial and non-discriminatory reports that are clear and concise and address issues of

causation and incapacity were discussed with the doctor. He understood that he would adhere strictly to the HIPAA laws that would also apply for LACERA reports. Staff reviewed with Dr. Bronshvag the Panel Physician Guidelines for evaluating LACERA applicants and defined the relationship between workers' compensation and disability retirement. Staff discussed the need to rely on his own objective and subjective findings rather than the opinions of previous physician reports and/or comments.

Dr. Bronshvag agreed to adhere to LACERA's standard of having his evaluation reports sent to us within 30 days of examination. Staff confirmed that Dr. Bronshvag is agreeable with accepting payment pursuant to LACERA's contract and billing procedures. Dr. Bronshvag was informed that if he is approved by the Board to be on our panel of physicians, he is required to contact the specialist assigned to the case for approval of any special tests or extraordinary charges. He has also been advised of the requirement to immediately notify LACERA if any license, Board certification, or insurance coverage is lapsed, suspended or revoked. He was informed that a Quality Control Questionnaire is sent to each applicant regarding their visit.

RECOMMENDATION

Based on our interview and the need for his specialties, staff recommends Dr. Bronshvag's application be presented to the Board for approval as a LACERA Panel Physician.

LACERA

Los Angeles County Employees Retirement Association

300 N. Lake Ave., Pasadena, CA 91101 ■ Mail to : PO Box 7060, Pasadena, CA 91109-706 626/564-2419 • 800/786-6464

GENERAL INFORMATION	Date
Group Name: ExamWorks	Physician Name: Michael M. Bronshvag, M.D.
I. Primary Address: 11010 White Rock Road Suite 120, Rancho Cordova, CA 95670	
Contact Person Kristina Lewis	Title General Manager
Telephone: 916 403 1769	Fax 916 920 2515
II. Secondary Address 11010 White Rock Road Road Suite 110, Rancho Cordova, CA 95670	
Contact Person Shawnette Davis	Title Lead Scheduler
Telephone 916 403 1784	Fax 916 920 2515

PHYSICIAN BACKGROUND

Field of Specialty Internal Medicine and Neurology	Subspecialty Board Certified Internal Med and Neurology	04/30/18
Board Certification [X] Yes [ ] No	License # G015805-CA	Expiration Date ~~04/30/2018~~

EXPERIENCE
Indicate the number of years experience that you have in each category.

Evaluation Type

I. Workers' Compensation Evaluations

- [X] Defense How Long? 40 years
- [X] Applicant How Long? 40 years
- [X] AME How Long? 40 years
- [X] IME How Long? 40 years
- [X] QME How Long? 25 years - since QME program began

II. [X] Disability Evaluations How Long? 40 Years

For What Public or Private Organizations? SSA, CALPERS, DOL, LAPFP

Currently Treating? [X] Yes [ ] No

Time Devoted to: Treatment 5 % Evaluations 95 %

Estimated Time from Appointment to Examination

- [X] 2 weeks (if needed)
- [X] 3-4 Weeks (Usually)
- [X] Over a month (rarely)

Able to Submit a Final Report in 30 days?

[X] Yes [ ] No
(more quickly if needed)

LACERA's Fee Schedule

Examination and Initial Report by Physician	$1,500.00 flat fee
Review of Records by Physician	$350.00/hour
Review of Records by Registered Nurse	$75.00/hour
Supplemental Report	$350.00/hour

-OVER-

Other Fees	
Physician's testimony at Administrative Hearing (includes travel & wait time)	$350.00/hour
Deposition Fee at Physician's office	$350.00/hour
Preparation for Expert Testimony at administrative Hearing	$350.00/hour
Expert Witness Fees in Superior or Appellate Court	$3,500.00 half day $7,000 full day

Physician agrees with LACERA's fee schedule? Yes No

Comments

40 year interest in musculo-skeletal injuries, infections, metabolic disorders, hernias

Name of person completing this form:

Michael M. Bronshvag, M.D.
(Please Print Name)

Title: M.D.

Physician Signature: [signature]

Date: 16 MAR 16

FOR OFFICE USE ONLY Physician Interview and Sight Inspection Schedule	
Interview Date: 6/7/16	Interview Time: 9:30 a.m
Interviewer: Debbie [signature]	

CURRICULUM VITAE

MICHAEL M. BRONSHVAG, M.D.

EDUCATION

1956 - 1960	New York University - A.B. 1960; Phi Beta Kappa
1960 - 1964	Columbia University College of Physicians and Surgeons - M.D., 1964

INTERNSHIP AND RESIDENCIES

1964 - 1965	Passavant Memorial Hospital (Northwestern University Affiliated Hospital) Mixed Medical Internship
1965 - 1968	Northwestern University Medical Center, Residency in Internal Medicine
1971	Board Certified in Internal Medicine, American Board of Internal Medicine
1968 - 1971	Letterman General Hospital, San Francisco, California Residency in Neurology
1974	Board Certified in Neurology, American Board of Psychiatry and Neurology

LICENSURE - Current

California License #G15805
Qualified Medical Evaluator, State of California

TEACHING APPOINTMENT -Current

University of California, San Francisco, in Neurology
(Assistant Clinical Professor of Neurology)

MILITARY SERVICE

1968 - 1980	ACTIVE DUTY - U.S. ARMY
1968 - 1980	Letterman Army Medical Center and Letterman Army Institute of Research (Colonel, Medical Corps)
1980 - 1981	ACTIVE DUTY - United States Public Health Service Hospital, San Francisco (0-6)
1982 - 2000	ACTIVE RESERVES - U.S. ARMY 6253 G.H. (Lt. Col., United States Army Reserve) Hamilton Field, Novato, California
1986 - 1987	146 CSH, California Army National Guard (Colonel)
1987 - 1996	USAR-IRR (Colonel)
1996 - 1999	6250 USAH, Ft. Lewis, Washington

1999 - 2000	2290 USAH, WRAMC, Washington, D.C.
2000-	RETIRED - 31 YEARS - U.S. ARMY Colonel, Retired Reserves, U.S. Army

EMPLOYMENT - PRIVATE PRACTICE

1981 - 1985	Ross Valley Medical Clinic, 1350 South Eliseo, Greenbrae
1982 - 1991	350 Parnassus Avenue, San Francisco
1991 - Present	3000 "L" Street, Suite 308, Sacramento and 10 Commercial Boulevard, Suite 108, Novato, CA 94949

RESEARCH INTERESTS

CHILD AND ADULT DEVELOPMENT, NEUROPSYCHIATRIC AND PSYCHOLOGICAL EVALUATION

1979 "Ontogenesis of Human Brainstem Evoked Potential Amplitude," by A. Salamy, C. Birtley-Fenn, and Michael M. Bronshvag in ***Developmental Psychobiology***

DISEASES OF PERIPHERAL NERVES

February 1978 "Spectrum of Gustatory Sweating with Especial Reference to its Presence in Diabetics with Autonomic Neuropathy," by Michael M. Bronshvag, ***American Journal of Clinical Nutrition*** Volume XXI, pages 307-309

"Treatment of Painful Diabetic Neuropathy with Hydroxocobolamin--Clinical Observations, Electrophysiologic Measurements. and Measurement of Urinary Myoinositol Levels," by Michael M. Bronshvag, Valerie Coppes and R.H. Herman.

HEADACHE

1978 "Vascular Headaches in Mixed Connective Tissue Disease," by Michael M. Bronshvag, Steven D. Prystowsky, and Daniel C. Traviesa, ***Headache*** Volume XVIII, pages 154-169

ANOXIC ISCHEMIC CEREBRAL PATHOLOGY AND INJURY

July 1978 "Cellular Basis of Anoxic Ischemic Brain Injury," by Michael M. Bronshvag, ***Western Journal of Medicine*** Volume CXXIX, pages 8-18

"Clinical Electroencephalographic Effects of Subacute Hemorrhagic Shock in Sleep," by Michael M. Bronshvag, ***Military Medicine***

"Cerebral Pathophysiology in Hemorrhagic Shock, Nuclide Scan, Fluorescence Microscopy, and Anatomic Correlations," by Michael M. Bronshvag--accepted for publication, ***STROKE***

"Cerebral Pathophysiology in Hemorrhagic Shock, Effects of Hemorrhagic Shock Upon Visual Evoked Potentials"

DIAGNOSIS AND EVALUATION OF HEAD INJURY

"Evaluation and Detection of Superior Sagittal Sinus Occlusion in the Sheep Employing Electroencephalography and Radio-Labeled Fibrinogen"

ELECTROENCEPHALOGRAPHY

"EEG Study of Normal Volunteers Before, During, and After Pyridoxine Deficient Diet"

PUBLICATIONS

BRONSHVAG, M., "Cellular Basis of Anoxic-Ischemic Brain Injury. Present Concepts in Internal Medicine," ***Neurology Symposium*** 1975, pages 112-122

BRONSHVAG, M., "Cellular Basis of Anoxic-Ischemic Brain Injury," ***Western Journal of Medicine*** Volume CXXIX, pages 8-18, 1978

BRONSHVAG, M., PRYSTOWSKY, S.D., AND TRAVIESA, D.C., "Vascular Headaches in Mixed Connective Tissue Disease," ***HEADACHE*** Volume XVIII, Pages 154-160, 1978

BRONSHVAG, M., "Spectrum of Gustatory Sweating with Especial Reference to Its Presence In Diabetics with Autonomic Neuropathy," ***American Journal of Clinical Nutrition,*** Volume XXXI, pages 307-309, 1978

SALAMY, A., FENN, C.B., and BRONSHVAG, M., "Ontogenesis of Human Brainstem Evoked Potential Amplitude," ***Developmental Psychology*** Volume XII, pages 519-526, 1979

BRONSHVAG, M., "Cerebral Pathophysiology in Hemorrhagic Shock, Nuclide Scan Data, Fluorescence Microscopy, and Anatomic Correlations," ***Stroke*** Volume XI, pages 50-59, 1980

BRONSHVAG, M., "Clinical and Electroencephalographic Effects of Subacute Hemorrhagic Shock in Sheep," ***Military Medicine*** Volume CXXXXV, pages 277-281, 1980

BRONSHVAG, M., COPPES, V.G., and BROOKES, E.D., "Acute Reactive Mitrochondrial Changes in Incremental Hemorrhagic Shock in Sheep," ***Neurological Research***, Volume III, pages 381-391, 1981

PRESENTATIONS

BRONSHVAG, M., and HERMAN, R., "New Concepts of Old Diseases: Treatment of Painful Diabetic Neuropathy with Hydroxo B12, Preliminary Observations," American College of Physicians Course, January 1977

BRONSHVAG, M.M., MD, INC. - QME courses (each year)

CURRENT INTERESTS

Electrodiagnosis
Neuroanatomy
Work Injuries and Occupational Health
Dynamics of Medical Instruction

Sample Report #1

MICHAEL M. BRONSHVAG, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM

[redacted]

XXXXXXXX XXXXXXX, [redacted]
XXXXX XXXXXXXXXXXX XXXXXXXXX XXXX
XXXX XXX XXXXX
XXXXXX XXXX, XX XXXXX-XXXX

XXXXXXXXXXX X. XXXXXX, Esq.
XXXXX, XXXXXXXX, XXXXXXX, XXXXXX, XXXX, XXXXXXXX, XXXXXXX & XXXX, LLP
XXXX XXXXXX XXXXXX, XXXXX XXXX
XXX XXXXXXXXX, XX XXXXX

AGREED MEDICAL EVALUATION

Re:	**XXXXXXXXXX, XXXXX**
Date of Injury:	[redacted]
Employer:	**XXXXXXXXXX XXXXXXX XXXXX**
WCAB Case No.:	**XXXXXXXXX**
Claim No.:	**XXXXXXXX**

Dear All:

Mr. XXXXX XXXXXXXXXX presented for an Agreed Medical Examination in [redacted] on the [redacted]. This is my report of my efforts.

The claimant, Mr. XXXXX XXXXXXXXXX, is [redacted] years old and resides in XXXXX XXXX, XXXXXXXXXX.

Under penalty of perjury, this report is submitted pursuant to 8 Cal. Code Regs. Section 9795(b) & (c) as a ***ML-104****-94 Comprehensive Medical Legal Evaluation Involving Extraordinary Circumstances and meets the requirement of four complexity factors. These include: (5) 6+ hours spent on any combination of three of the complexity factors (1)-(3) which shall count as 3*

complexity factors; (6) Addressing the issue of medical causation, which shall count as one complexity factor;

Time spent in direct, face to face contact was .50 hour. Time spent reviewing records required 3 hours. Time spent on research was 3 hours. Time spent preparing the report was 1.50 hours. Total time spent on this case was ***8 hours****.*

INITIAL COMMENT – I am consulted relevant to the specific problem of left basilica vein thrombosis. This apparently resulted from insertion-removal of a PICC vein line in [redacted]. Head blows are noted in [redacted] and then [redacted] (or [redacted]). Migraine, neck trauma and seizure versus non-seizure (?psyche?) problems are weighed.

The discussion to date does not weigh the genetically-determined issues of Migraine with Basilar Aura (MBA – also called Basilar Migraine) and Familial Hemiplegic Migraine (FHM). FHM-BMA have similar dominant genetic drivers. The two (MBA-FHM) may cause syncope/coma/psychosis episodes resembling seizure or psyche events. In view of the positive family history and the history of migraine since age seven, FHM/MBA may explain the 'seizure/pseudoseizure issue. Also, the literature until[redacted] stated that DHE was contraindicated for MBA/FHM, although the[redacted] literature (now) says that DHE is safely given in FBM-BMA

HISTORY

The claimant tells of interest in agribusiness and that he had worked for over [redacted] years as a [redacted] – XXX until [redacted] sustained head trauma (two specific injuries)([redacted] and then [redacted]), and also has had a history of back-spine trauma of a cumulative nature. He has been evaluated in detail by medical neurologist XXXXXX.

The claimant tells me his neck difficulties resulted from airplane turbulence injuries. His job was a XXX[redacted] and the plane he was in – doing surveillance – jolted (in [redacted] and then [redacted]) while he was wearing a five-pound helmet - he struck the ceiling of the plane with his head and he had the onset of left-sided neck pain and left-sided posterior head pain, apparently largely in the distribution of the left greater occipital nerve relevant to the [redacted] injury.

He was referred, apparently at the suggestion of Dr. XXXXXX, to a XXXX headache protocol which involved the infusion of DHE (dihydroergotamine) and this was by "PICC" line

(peripherally inserted central catheter). He developed phlebitis of the left arm, in what is described as the distribution of the left basilic vein in the upper medial part of the arm (largely proximal to the left elbow). He was thus then a candidate for anticoagulation, and Coumadin was chosen because of his previous history of gastroesophageal issues (rather than one of the Factor X inhibitors). He tells me that an left arm ultrasound performed more recently ([redacted] suggested that the clot was "dissipating" and thus the claimant's ongoing left upper arm symptoms are perhaps either due to vein occlusion and perhaps due to vein inflammation.

Accordingly, the sequence of events is of exposure to airplane turbulence in his job for the XXX (officer), diagnosis of left-sided neck symptoms and left-sided posterior head symptoms. His migraine history was noted and resulted in a diagnosis of "migraine plus trauma" and he was entered into a XXXX-DHE protocol. As a result of that DHE protocol (PICC line), he developed phlebitis as noted and the claimant has been on Coumadin therapy. His symptoms in the left proximal arm – medially – persist but are perhaps improving-improved.

The claimant still has the basis issues of

a) Low back – cumulative and

b) Left-sided neck difficulties and left posterior head difficulties (greater occipital neuralgia, migraine, cervical degenerative joint and disc disease, ?combination?, seizure v.s. other).

At this point I turn my attention to the letter from Attorney XXXXXX. Attorney XXXXXX takes note of a blood clot in the left arm believed to be a compensable consequence by Attorney XXXXXX. The injuries in question to head, neck and left arm occurred in [redacted] and [redacted] (or [redacted]

It is stated that the claimant was evaluated by Drs. XXXXXX (neurology), XXXXXXXXXX (neuropsychologist), and XXXXXXX (orthopedist). Hearing loss was noted and low back cumulative trauma noted. The claimant's head pain difficulties since [redacted] are noted and the left arm blood clot issue of [redacted] is noted. The possibility of low back surgery was mentioned as well.

I review the AME letter from Claims Representative XXXXXXX as well. Issues involve left shoulder, upper back, internal organs, abdomen-groin, CHP. The issues of ongoing problems, back surgery and blood thinners are raised by Representative Valenti. It is stated that the claimant was medically retired in [redacted] and had been off work for several months prior to his retirement.

The more recent head blow occurred in [redacted] (Dr. XXXXXXXX) or [redacted]

MEDICAL REVIEW OF SYSTEMS

The claimant gives a history of gastroesophageal symptoms which interact with his Coumadin issues. He is being treated by lisinopril for blood pressure issues and nortriptyline for pain and sleep issues. He is being treated with Coumadin.

CURRENT COMPLAINTS

As noted above, the claimant continues to have symptoms (improved) relevant to the medial aspect of the left upper arm (basilic vein).

He has difficulty with prolonged lying down, sitting, standing and walking, and increased problems with climbing, lifting, bending, reaching, crouching, stooping, kneeling and balancing.

He is unable to work at the present time because of his migraines which he describes as debilitating, his head-neck pain, his low back pain, and also his left medial upper arm symptoms.

PREVIOUS MEDICAL HISTORY

As noted – above – prior to the [redacted] head and neck event, the claimant did have lower back symptoms as well.

JOB DESCRIPTION

The claimant has been a [redacted], including flight details, during his [redacted] years of service. He notes the heavy helmet.

PERSONAL AND SOCIAL HISTORY

The claimant is [redacted] with a weight of [redacted] pounds. He is under the care of Dr. XXXXXXXX of XXXXXX, Dr. XXXXXXX of XXXX, and Dr. XXXXX of XXXXXX, plus treatment for the blood clot. He lists his airplane as a [redacted]. The claimant was hospitalized for what is described as "seizure"-like activity thought not to be actual seizures ([redacted]).

The family history is positive for diabetes. The mother has migraine headaches. He is a nonsmoker, nondrinker, and he denies drug misuse. He is a life-long Californian - [redacted]. He is

continuing studies at XXXXXX XXXXX. He is able to read and write well. He had been a XXX [redacted] from [redacted] including flight duties. He has a driver's license, is right-handed, has difficulty walking a block, climbing more than a few steps or lifting more than a few pounds.

Thus the claimant, who is receiving pain relief treatment (nortriptyline), psychological treatment (Lexapro), blood pressure treatment (lisinopril), and remains on Coumadin, has a history of

a) The left upper arm medial vein problem,

b) Neck and left side of head difficulties described as migraine (?greater occipital neuralgia?),

c) Low back issues, and

d) History of gastrointestinal issues as well.

e) Seizures versus psychological issues

MEDICAL RECORDS REVIEW

At this point I turn my attention to the (big stack of) medical records kindly provided at this time. These records are contained in a sizable stack.

I note that Dr. XXXXXXXX – medical neurologist – in [redacted] described a history of an injury in [redacted] and a further event in [redacted]). Dr. XXXXXXXX diagnosed chronic neck pain with musculoskeletal headaches, work-related, and he thought that there might be a brain tumor or intracranial cause. MRI of the head was thus planned by medical neurologist Cantrell.

The MRI of the head – [redacted] – took note of right maxillary sinusitis and no intracranial abnormalities. Dr. XXXXXXXX thus, after that negative study, stated that a cervical and root problem might be present. Neck abnormalities on the neck MRI were noted. Left-sided changes were prominent. Electrodiagnostics at that time were compatible with left carpal tunnel syndrome and no other overt abnormalities.

Dr. XXXXX had noted in [redacted] that the claimant had cervical radiculitis, neck pain, tension headache, myofascial syndrome, myopathic pain, chronic pain-related insomnia, and chronic pain syndrome.

Imaging studies documented normal carotid in [redacted].

Dr. XXXXX had noted acute right facial droop, with altered speech, and intractable headache (?migraine?).

Dr. XXXX had recommended DHE treatment in[redacted].

Dr. XXXXXXX had described the claimant's set of difficulties, noted that neurological issues were prominent, and that these all seemed to be related to his work efforts (turbulence – head bump). The possibility of seizure episode was mentioned in [redacted]. The issue of psychogenic causes for the seizure-like episodes was mentioned. PTSD was mentioned as well. Dr. XXXXXXX in [redacted] mentioned the possibility of cervical epidural steroid injection, occipital nerve block, and medications. He did not urge trigger point injections and did not urge medical foods or creams. Epidural steroid injection was contemplated.

In [redacted], Dr. XXXXXXXXXX thought that there was a cognitive disorder – mild – post concussion.

EEG in [redacted] was normal.

It was stated that the claimant had a "known history of seizure disorder" (Dr. XXXXXXXXX) and has presented in status epilepticus, with a seizure disorder, controlled hypertension, hypertensive heart disease with left ventricular hypertrophy and chronic neck pain. CT of the brain was described as normal. The EEG brain wave test was normal.

Dr. XXXXX had diagnosed occipital neuralgia, psychogenic nonepileptic seizures, and a mood disorder.

Dr. XXXXXX memorialized the vectors and stated in [redacted] that the claimant had a constant, dull, baseball-bat pain in the cervical occipital junction, a ram-horn distribution head pain, with migrainous "characteristics" and nausea, and a spike headache that appeared to be sexually related. The fabric of the four episodes was analyzed. Dr. XXXXXX did not come to closure but mentioned vascular seizure, periodic, and movement issues. The spectre of nonorganic disease was raised.

Dr. XXXXX noted lumbar puncture brain pressure measurement. He thought the most reasonable diagnosis was occipital neuralgia, left-sided. It was noted that EEG monitoring observation in [redacted] documented a typical episode, with a normal EEG.

The MRI of the low back dated [redacted] documented modest changes at L4-L5 and L5-S1 levels.

The [redacted] note of Dr. XXXXXXXXXX stated that a seizure was not demonstrated and that psychogenic reasons were suspected by Dr. XXXXXXXXXX did not think that there was a clear-cut psychological problem.

Neurosurgeon XXXXXXXXXXX of Los Angeles had stated in [redacted] the claimant had normal neurodiagnostics and the diagnoses were occipital neuralgia, lump at the left occipital, cervical region, seizures vs pseudo-seizures, cervical radiculopathy, lumbar radiculopathy, shoulder pain, cognitive impairment, emotional distress and sleep disturbance. The doctor was curious about the lump at the back of his neck. EEG was advised. The electrodiagnostics were noted.

The imaging study of [redacted] described two soft tissue densities within the subcutaneous fat overlying the occiput in the region of clinical concern. [redacted]).

FCE evaluation is noted.

Clinical psychologist XXXX noted cognitive issues.

The psychological evaluation of Dr. XXXXXXXXXX in [redacted] again took note of cognitive issues.

Dr. XXXXXXX continued to take note of neck and also low back issues.

Dr. XXXXXX had, in [redacted], advised of the complexity of the issues and suggested he see neurologist XXXXXXX, MD, headache specialist referral.

Dr. XXXXXX had noted in [redacted] that further data were provided. Head pain since age of eight was described, with worsening relative to work injuries. Family history of headache was noted as well. CSF protein was 55 in [redacted]. Diagnosis of Dr. XXXXXXX was chronic migraines with medication overuse and primary stabbing headaches, and likely migrainous vertigo. The issue of the claimant's motion sensitivity was noted. Dr. XXXXXX noted that the claimant should have the DHE protocol and then return to Dr. XXXXXX for followup. Opiate withdrawal was advised.

Headache service efforts were noted in [redacted]. Occasional headaches at age 8-9. Constant headaches were described in [redacted]. Left-sided photophobia, phonophobia and nausea, extreme movement sensitivity, neck stiffness, blurring of vision, lightheadedness, allodynia, difficulty concentrating, and mood changes were mentioned. Kidney stones, right inguinal hernia

repair, vasectomy, sinus surgery, head trauma x2, degenerative joint disease of the neck, and upper and lower endoscopy – relevant to GI bleed – and proctitis was thought to be relevant to NSAID usage. Medications included gabapentin, Norco, Lexapro, lisinopril, and Botox. The discharge diagnosis of [redacted] – Dr. XXXXX and Dr. XXX – was chronic migraine.

PM&R specialist XXXXXXXX stated in [redacted] the claimant had a history of traumatic brain injury, head pain and neck pain.

After the [redacted] and [redacted] ([redacted]) head bump events, he noted twisting difficulties in [redacted]. Loss of consciousness followed by headache was mentioned. Episodes of vertigo were noted. Rehab efforts were noted.

The UCSF notes from [redacted] describe IR PICC line placement. Successful midline placement was described, under ultrasound and fluoroscopic guidance. DHE administration was noted. Enoxaparin treatment was mentioned.

The efforts of Dr. Riggins are noted. The Midline (?Medline) was then pulled. The ED note – [redacted] – is noted. Left arm difficulty was noted. Numbness and swelling of the fingers were mentioned.

The note of Dr. XXXXXX of [redacted] is mentioned. Dr. XXXXXX had taken note of the [redacted] left arm phlebitis event. Dr. XXXXXX's rating formula is noted.

Dr. XXXXXXX took note of the claimant's situation in note of [redacted] Improvement of headaches was noted. Botox treatments were commented upon. Proctitis diagnosis was noted.

Dr. XXX was treating the claimant in the "local hematology office" for left upper extremity incompletely occlusive thrombus throughout the visible portion of the left basilic vein with Coumadin treatment.

Dr. XXXX – XXXXXXXXXX XXXXXXXX & XXXXXXX XXXXXX – XXXXXX – reported in [redacted].

Dr. XXXXXX had stated that the claimant cracked his flight helmet in [redacted] and had injury in [redacted]. Diagnostic audiometry mentioned bilateral high-frequency sensorineural hearing loss, high-frequency "mild to severe," was mentioned. Tinnitus, occipital neuralgia type dizziness, vestibular concussion, vestibular response impairment were also noted.

The possibility of acoustic neuroma was weighed by Dr. XXXXXX and thought to be unlikely.

MRI and MRA in [redacted] and [redacted] were unremarkable. EEG video telemetry was normal. EEG at Pacific was negative. CT scan of the head in [redacted] showed the subcutaneous densities. Lumbar puncture in [redacted] described opening pressure of 28. Scintigraphy in [redacted] was described as normal. Coenzyme Q10 therapy was suggested.

The claimant added that the helmets that he had been employing when he was flying were very heavy ones (about 5 pounds) and more recently light-weight helmets have been substituted. It is the opinion of the claimant that the heavy helmets increased his difficulties relevant to skull-cervical spinal cord issues when head trauma was involved. (Narrator's comment: That matches up with the current thinking about heavy football helmets for high school football players.)

Thus the issues challenging the claimant – old and new – include gastroesophageal issues (nonsteroidal-related – proctitis), Coumadin therapy for his left basilic vein area phlebitis, low back pain, and left-sided head issues, and overall musculoskeletal issues.

PHYSICAL EXAMINATION

At the present time, my physical examination of the claimant does not demonstrate any overt cognitive, memory or language deficits. The claimant apparently is continuing in studies at [redacted].

He described to me no current overt psychological symptoms – nor were any evident to me. Vision and hearing were grossly normal (however, high-tone hearing loss has been demonstrated (?noise-related?). The blood pressure is measured at 136/83, the pulse is 101, and no overt dyspnea, cyanosis, edema or cough are noted. Eyes, funduscopic exam, and cranial nerves are normal. ENT exam is normal. Neck shows no abnormality of trachea, veins, or thyroid. Lungs, heart, and pulses are normal. The abdominal and skin examinations are normal.

No arterial pulse deficits are noted.

The craniocervical junction is of note in that there is tenderness to palpation in the left craniocervical junction area over the course of the left greater occipital nerve.

(There is a greater occipital nerve – there is a third occipital nerve – there is a lesser occipital nerve – there is no "occipital nerve.")

The claimant complained of discomfort in the left occipital region of the scalp in the distribution of the left greater occipital nerve.

Range of motion of neck was slightly limited. There was tenderness to palpation in the left paracervical region. Shoulder, elbows, wrists, arms and hands were unremarkable. Midback was normal. There is tenderness to palpation over the low back and range of motion of low back was limited to 40 degrees of forward flexion, -5 degrees of extension, and 10 degrees bilaterally of lateral flexion. Hip rotation and straight leg raising were not limited.

The balance of the musculoskeletal examination was normal.

Examination of the left "upper" arm demonstrated residual tenderness in the distribution of the left basilic vein. I did not demonstrate gross stasis-venous in the upper arms, forearms or hands.

No overt sensory or motor neurological deficits are noted and the tendon reflexes are 1+.

DIAGNOSES

Accordingly, the appropriate diagnoses are:

1 Complex headache issue with positive family history of migraine; headaches with motion sensitivity dating back to youth; head injuries, most probably involving craniocervical junction and the left greater occipital nerve, with associated cervical issues as well; the combination of migrainous issues, and greater occipital nerve neuralgia issues are noted. Migraine with basilar aura (MBA) and variant Familial Hemiplegic Migraine (FHM) should be considered as MBA often causes peculiar episodes of loss of consciousness

2 Neck strain difficulties, cervical degenerative joint and disc disease - ? nature of lump in left occipital area ?.

3 Low back difficulties, low back degenerative joint and disc disease.

4 History of kidney stones.

5 Episodes of altered awareness and neurological difficulties, suspected to be seizures; seizures not demonstrated; the possibility of an ischemic problem or a migraine-related difficulty, possibility of ?familial hemiplegic migraine? ?migraine with basilar aura? noted at this time.

5. Occlusive phlebitis, left basilic vein – improving – treated.

INITIAL DISCUSSION

The primary issue I am challenged with is the left basilic vein and the left upper arm and to what extent the claimant will or will not require ongoing anticoagulation. As a rule of thumb (see references), a problem with phlebitis may require up to six months of therapy in the absence of ongoing phlebitis. Episodes of pulmonary embolism with obvious phlebitis are usually treated for six months. Episodes of pulmonary embolism in the absence of known phlebitis are often treated for a longer period of time (a year).

The diagnosis of "DVT" ("deep") has been offered but perhaps it is better to regard the basilic vein as a superficial ("S") vein. I do not observe any current documentation of spread of the process into the axillary area (issue of effort thrombosis – axillary vein) or more proximally (Lemierre's syndrome).

Accordingly,

Temporary total disability – This claimant has not worked in 2+ years. He apparently is not felt able to go back to his job as a[redacted] and[redacted]

His episode of left basilic vein thrombosis is almost certainly caused by the PICC line. If it is concluded that his headaches have an occupational component, and referral to UCSF was an occupational issue, then this phlebitis event is undoubtedly work-caused. The claimant did continue with his studies and examinations.

If this episode of basilic vein thrombosis had occurred while the claimant was actively working as a[redacted] and[redacted], a period of TTD would have been appropriate.

Permanent partial disability-MMI – Occlusive phlebitis disease of this type often remits entirely (sometimes it doesn't). Sometimes the periphlebitis (inflammatory issue) persists and gives ongoing symptoms even though flow has been regained. The claimant has been told that the basilic vein is partially functional at this time (see discussion below). Spread of the clot problem proximally into the chest is possible but unlikely.

AMA Guides Impairment Rating – I take note of the efforts of Drs. XXXXXXXXX, XXXXXX and XXXXXXX.

It is my understanding that I am being asked to comment only relevant to this claimant's left basilic vein area. It is possible that this area will "heal up entirely." I want to make sure that we are not dealing with anything that might lead to a troublesome vein problem more proximally

(see discussion below). Therefore, relevant to AMA Guides rating, I will hold off at this time. I take note of the fact that Dr. XXXXXX has provided ratable language in other areas.

Subjective Symptoms – Relevant to the claimant's left upper arm, he was acutely symptomatic in [redacted], with a swollen arm. His findings currently are much more benign and it is possible that they will entire remit (see discussion below).

Objective factors – At the present time there is tenderness over the course of the left basilic vein without other striking abnormalities.

Over and above that, the claimant quite obviously has other issues relevant to spine and head.

Causation – As noted above, assuming that the claimant's head pain is at least partially work-caused (family history was noted), then his admission to XXXX in [redacted] was for work-related issues, and the removal of the PICC line and the onset of phlebitis would be a work-caused compensable issue.

Apportionment – Pending. It is possible and hoped for that there will be no residual ratable disability and therefore apportionment might not need to be contemplated relevant to the basilic vein.

Return-to-work issues – The claimant is a college student. He is probably going to be employable. Return to [redacted] work seems unlikely.

Treatment efforts – The claimant is on Coumadin. His doctors are tentative or uncomfortable with the newer Factor X inhibitors, because if they do cause bleeding, the bleeding might not be easily stopped (if a patient on Coumadin bleeds, you can give him or her a shot of vitamin K) (or have him eat a few Brussels sprouts).

Studies requested – The claimant requires a current MR–V study of the left arm and the left thoracic outlet area to make sure that the clot has not spread proximally.

Ultrasound is a reasonable modality for the basilic vein, but since the issue is not just the basilic vein but the more proximal areas (axillary-subclavian-vertebral), the MR "V" (high field 3 tesla) is appropriate. I am going to order up a collateral-companion blood count, chemistry panel, ESR and CRP relevant to the issue of an inflammatory disorder.

Research performed – Please refer to the refereed-open source articles I have selected relevant to familial hemiplegic migraine, greater occipital neuralgia, effort thrombosis (axillary vein), basilic vein, Lemierre's syndrome, and all anticoagulants (Coumadin versus Factor X inhibitors).

I request the privilege of seeing the claimant back again in two to four months to make sure that no worrisome residual vein problems are present.

If questions remain or arise, kindly write me back and I will respond immediately. As you will note, it is my assumption that Drs. XXXXXXXXXX, XXXXXXX and XXXXXX are providing the ratings to areas "other than" the claimant's left upper extremity. However, if further input is required on any of those "other" issues, let me know. As you will also note, I am looking forward to seeing the claimant back again to make sure that his venous system is 'okay'.

As you will also note, I have commented upon the possibility of MBA-FHM, and the pertinent QMEs and treaters may choose to weigh those comments.

I certify that I took the complete history from the claimant, conducted the examination, reviewed all available medical records, and composed and drafted the conclusions of this report. The conclusions and opinions within this report are solely mine. I declare under penalty of perjury that the information contained in this report and its attachments, if any, is true and correct to the best of my knowledge and belief, except as to information that I have indicated I received from others. As to that information, I declare under penalty of perjury that the information accurately describes the information provided to me and, except as noted herein, that I believe it to be true. In accordance with Labor Code Section 57039(a), there has not been a violation of Labor Code Section 139.3, and the contents of the report are true and correct to the best of my knowledge. This statement is made under penalty of perjury.

Pursuant to 8 Cal. Code Regs Section 49.2-49.9, I have complied with the requirement for face-to-face time with the claimant in this evaluation. If necessary, I have discussed apportionment in the body of this report. If I have assigned disability caused by factors other than industrial injury, that level of disability constitutes the apportionment. The ratio of non-industrial disability, if any, to all described disability represents my best medical judgment of the percentage of disability caused by the industrial injury and the percentage of disability caused by other factors, as defined in Labor Code Sections 4663 and 4664.

Respectfully,

Michael M. Bronshvag, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM
Date of Report: [redacted]

PubMed

Abstract

Full text links

Angiology. 2000 Feb;51(2):173-7.

Internal jugular vein thrombosis, Lemierre's syndrome; oropharyngeal infection with antibiotic and anticoagulation therapy--a case report.

Nakamura S[1], Sadoshima S, Doi Y, Yoshioka M, Yamashita S, Gotoh H, Onoyama K.

Author information

Abstract

The authors present a case of Lemierre's **syndrome** that is an uncommon septic thrombophlebitis of the internal jugular **vein**. A 31-year-old man developed pharyngeal pain one month before hospital admission when he suffered from a severe headache and painful swelling of the left side of his neck. He was diagnosed with tonsillitis. Contrast-enhanced computed tomography and magnetic resonance imaging of the neck revealed the presence of an occlusive thrombosis of the left internal jugular **vein** and an inflamed mesopharynx. His symptoms and the jugular **vein** thrombus showed remarkable improvement after administration of antibiotic and anticoagulation therapy. No pulmonary embolism or other metastatic infection were observed. It was suggested that accurate diagnosis during early treatment is essential to obtain a successful prognosis for Lemierre's **syndrome**.

PMID: 10701727 [PubMed - indexed for MEDLINE]

Publication Types, MeSH Terms

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Abstract

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FULL-TEXT ARTICLE

Thromb Res. 2015 Sep 1. pii: S0049-3848(15)30103-1. doi: 10.1016/j.thromres.2015.08.020. [Epub ahead of print]

Systematic review of anticoagulant treatment of catheter-related thrombosis.

Baumann Kreuziger L[1], Onwuemene O[2], Kolesar E[3], Crowther M[4], Lim W[5].

Author information

Abstract

Central venous catheter-related thrombosis (CRT) is a complication seen in patients requiring long-term intravenous access. Treatment of CRT is not standardized and international guidelines for treatment are based on extrapolation of evidence from lower extremity thrombosis. We performed a systematic review of the literature to evaluate if duration of anticoagulation affects the risk of recurrent venous thrombosis, post-thrombotic **syndrome**, or major hemorrhage. We searched PubMed, Embase, Medline, CINAHL, Cochrane, and ACP Journal club for studies of CRT treated with anticoagulation. Of 1648 titles and abstracts, 23 studies met our inclusion criteria. No randomized trials were identified. Duration of anticoagulation varied from 8days to more than 6months. Outcomes of patients with upper extremity thrombosis due to CRT or other etiologies were often combined. The incidence of post-thrombotic **syndrome** varied between 0 and 75% depending on the definition used. Seven percent of patients with upper extremity thrombosis treated with anticoagulation experienced recurrent **deep vein** thrombosis and 2.8% experienced pulmonary embolism. Major hemorrhage was reported in 2.8-4.9% of anticoagulated patients. Prospective studies evaluating the optimal duration of anticoagulation in patients with CRT are needed.

PMID: 26342400 [PubMed - as supplied by publisher]

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Objective factors – At the present time there is tenderness over the course of the left basilic vein without other striking abnormalities.

Over and above that, the claimant quite obviously has other issues relevant to spine and head.

Causation – As noted above, assuming that the claimant's head pain is at least partially work-caused (family history was noted), then his admission to UCSF in December 2014 was for work-related issues, and the removal of the PICC line and the onset of phlebitis would be a work-caused compensable issue.

Apportionment – Pending. It is possible and hoed for that there will be no residual ratable disability and therefore apportionment might not need to be contemplated.

Return-to-work issues – The claimant is a college student. He is probably going to be employable. Return to CHP – TSO work seems unlikely.

Treatment efforts – The claimant is on Coumadin. His doctors are tentative or uncomfortable with the newer Factor X inhibitors, because if they do cause bleeding, the bleeding might not be easily stopped (if a patient on Coumadin bleeds, you can give him or her a shot of vitamin K) (or have him eat a few Brussels sprouts).

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If questions remain or arise, kindly write me back and I will respond immediately. As you will note, it is my assumption that Drs. Hutchinson, Mandell and Kasman are providing the ratings to areas "other than" the claimant's left upper extremity. However, if further input is required on

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FULL TEXT ARTICLE

J Formos Med Assoc. 2006 Feb;105(2):182-6.

Effort thrombosis of the upper extremities related to an arm stretching exercise.

Liang HW[1], Su TC, Hwang BS, Hung MH.

Author information

Abstract

"Effort" axillary-subclavian vein thrombosis (Paget-Schroetter syndrome) is an uncommon deep venous thrombosis due to repetitive activity of the upper limbs. Most cases of this condition are related to strenuous or prolonged sport or occupational activities, while others are associated with endogenous or exogenous risk factors. We report the case of a 43-year-old, previously healthy, male who developed right axillary-subclavian venous thrombosis, which was possibly associated with an exercise involving arm extension and shaking in a posture of shoulder abduction and outstretched for 10 minutes on 2 consecutive days. The condition improved but returned with increased severity when he resumed the exercise after a 2-day break, when he presented with a swollen and bluish arm at the emergency department. Sonographic examination showed moderate thrombotic stenosis of the right axillary vein. Effort thrombosis was diagnosed after ruling out associated coagulopathy or concomitant malignancy. External compression of the accessory ribs or lymph nodes were not detected. He was treated with low molecular weight heparin, followed by oral anticoagulant therapy for 6 months. Only partial resolution of thrombosis was achieved after 6 months of anticoagulant therapy, but pulmonary embolism did not occur during 18 months of follow-up. This case illustrates that, although unusual, Paget-Schroetter syndrome can occur in a healthy patient as a result of mild to moderate exercise.

PMID: 16477342 [PubMed - indexed for MEDLINE]

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Patient information: Deep vein thrombosis (DVT) (Beyond the Basics)

Authors
Menaka Pai, MD, FRCPC
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Lawrence LK Leung, MD

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Geraldine Finlay, MD

DEEP VEIN THROMBOSIS OVERVIEW

Venous thrombosis is a condition in which a blood clot (thrombus) forms in a vein. This clot can limit blood flow through the vein, causing swelling and pain. Most commonly, venous thrombosis occurs in the "deep veins" in the legs, thighs, or pelvis (figure 1); this is called a deep vein thrombosis, or DVT.

DVT is the most common type of venous thrombosis. However, a thrombus can form anywhere in the venous system. If a part or all of the blood clot in the vein breaks off from the site where it is formed, it can travel through the venous system; this is called an embolus. If the embolus lodges in the lung, it is called pulmonary embolism (PE), a serious condition that leads to over 50,000 deaths a year in the United States. In most cases, PE is caused when part of a DVT breaks off and lodges in the lung. The term "venous thromboembolism" is sometimes used when discussing both DVT and PE.

This topic review discusses the risk factors, signs and symptoms, diagnostic process, and treatment of a deep vein thrombosis. The diagnosis and treatment of pulmonary embolisms are discussed separately. (See "Patient information: Pulmonary embolism (Beyond the Basics)".)

DEEP VEIN THROMBOSIS RISK FACTORS

There are a number of factors that increase a person's risk of developing a deep vein thrombosis.

If a person is found to have a DVT and there is no known medical condition or recent surgery that could have caused the DVT, it is possible that an inherited condition is the cause. This is especially true in people with a family member who has also experienced a DVT or pulmonary embolism. In these cases, testing for an inherited thrombophilia may be recommended. However, finding an inherited thrombophilia does not change the way that doctors treat the venous thromboembolism, and may not increase the chance of the blood clot coming back. (See 'Finding the cause of venous thrombosis' below.)

Medical conditions or medications — Some medical conditions and medications increase a person's risk of developing a blood clot:

- Pregnancy
- Obesity
- Smoking
- Heart failure
- Previous DVT or pulmonary embolism (PE)
- Increased age
- Cancer – Some cancers increase substances in the blood that cause blood to clot.

Anticoagulation in Deep Vein Thrombosis

• Author: Donald Schreiber, MD, CM; Chief Editor: Barry E Brenner, MD, PhD, FACEP more...

Updated: Jan 14, 2015

Advantages of Anticoagulant Therapy

Anticoagulant therapy remains the mainstay of medical therapy for deep venous thrombosis (DVT) because it is noninvasive, it treats most patients (approximately 90%) with no immediate demonstrable physical sequelae of DVT, it has a low risk of complications, and its outcome data demonstrate an improvement in morbidity and mortality. Meta-analyses of randomized trials of unfractionated heparin (UFH) and low-molecular-weight heparin (LMWH) showed that they were similar, with risk of recurrent DVT of 4%, a risk of pulmonary embolism (PE) of 2%, and a risk of major bleeding of 3%.[1, 2]

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Disclosure: Nothing to disclose.

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Headache. 2010 Jun;50(6):1057-9. doi: 10.1111/j.1526-4610.2010.01682.x. Epub 2010 May 7.

Acute treatment of basilar-type migraine with greater occipital nerve blockade.

Baron EP[1], Tepper SJ, Mays M, Cherian N.

Author information

Abstract

Basilar-type migraine (BTM) precludes use of migraine-specific medications such as triptans and ergots based on concerns originating from the vascular theory of migraine, although data supporting this contraindication are lacking. Availability of effective treatments for acute BTM is limited. We report a case of BTM aborted with greater occipital nerve (GON) blockade given in the setting of prominent suboccipital tenderness. GON blockade may provide an additional option in acute management of BTM. It may be particularly useful when associated with prominent ipsilateral suboccipital tenderness.

PMID: 20487035 [PubMed - indexed for MEDLINE]

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Migraine with Brainstem Aura

Migraine with brainstem aura (MBA) is characterized by recurring attacks of certain temporary symptoms that are believed to originate in the brainstem. The brainstem is located at the base of the brain and connects to the top of the spinal cord; its function is to help regulate the two-way channel of communication between the brain and the body. Until 2013, MBA was known as "basilar-type migraine." In the updated version of the International Headache Society's guidelines – The International Classification of Headache Disorders, 3rd Edition (ICHD-III) – it was renamed "migraine with brainstem aura" because research now suggests that the basilar artery in the brain is not involved in causing its symptoms, as had previously been thought.[1]

The ICHD-III classifies MBA as a sub-type of the broader category of migraine with aura, which was also revised in the newest guidelines to include MBA and three other sub-types: migraine with typical aura (MTA), hemiplegic migraine, and retinal migraine. MTA is the most common of those four, with MBA and the others being relatively rare. While researchers are unsure about precisely how many people have MBA worldwide, a 2006 study of 362 patients with MTA in Denmark reported that about 1 in 10 of those patients also had MBA.[2] Another study in China in 2011 found that 6.6% (23/348) of MTA patients had also experienced MBA attacks.[3] In both studies, there were about 4 times as many women with MBA than men. Most people are initially diagnosed with MBA as teenagers or young adults.

Symptoms of migraine with brainstem aura

The ICHD-III defines a specific set of brainstem aura that are experienced as symptoms during MBA attacks:

- Vertigo is a type of dizziness described as the feeling that the body or surrounding environment is spinning or tilting, even though both are actually stationary.
- Dysarthria refers to difficulty controlling the speech-producing muscles (mouth, tongue, larynx, etc) resulting in slurring, mumbling, and general difficulty in producing sounds and

IHS Classification ICHD-II

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Subcommittees and working groups

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Part I: The primary headaches

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Part III: Cranial neuralgias, facial pain and other headaches

Appendix

Definition of Terms

Home » Basilar-type migraine |1.2.6|G43.103

IHS	Diagnosis	ICD-10
1.2.6	**Basilar-type migraine**	**G43.103**
Previously used terms	Basilar artery migraine, basilar migraine	

Description:
Migraine with aura symptoms clearly originating from the brainstem and/or from both hemispheres simultaneously affected, but no motor weakness.

Diagnostic criteria:

A. At least 2 attacks fulfilling criteria B-D

B. Aura consisting of at least two of the following fully reversible symptoms, but no motor weakness:
   1. dysarthria
   2. vertigo
   3. tinnitus
   4. hypacusia
   5. diplopia
   6. visual symptoms simultaneously in both temporal and nasal fields of both eyes
   7. ataxia

Consult the Sitemap to learn more about the structure of the classification and its main chapters.

More

To facilitate headache diagnosis in daily practice, the classification provides the corresponding WHO ICD-10NA codes for each IHS code.

More

The Classification Subcommittee prepares and revises the International Classification of Headache Disorders.

More

ICHD-III Beta
The International Classification of Headache Disorders, 3rd edition, beta version

ICHD-III Beta

Extend your electronic library with important IHS publications. All documents may be downloaded free of charge.

More

Cephalalgia is the official journal of the IHS. It contains original papers on all aspects of headache. The journal provides an international forum for original research papers, review articles and short communications.

http://cep.sagepub.com/

Ask questions and share information about the 2nd edition of the Headache Classification in one of our

Occipital neuralgia

From Wikipedia, the free encyclopedia

Occipital neuralgia, also known as **C2 neuralgia**, or (rarely) **Arnold's neuralgia**, is a medical condition characterized by chronic pain in the upper neck, back of the head and behind the eyes. These areas correspond to the locations of the lesser and greater occipital nerves. The greater occipital nerve also has an artery that supplies blood that is wrapped around it - the occipital artery - that can contribute to the neuralgia. This condition is also sometimes characterized by diminished sensation in the affected area as well.

Occipital neuralgia

Classification and external resources

ICD-10	G52.8 (http://apps.who.int/classifications/icd10/browse/2015/en#/G52.8),[1] R51 (http://apps.who.int/classifications/icd10/browse/2015/en#/R51),[2] G44.847
ICD-9-CM	723.8 (http://www.icd9data.com/getICD9Code.ashx?icd9=723.8)

Contents

Causes

Occipital neuralgia is caused by damage to these nerves. Ways in which they can be damaged include trauma (usually concussive), physical stress on the nerve, repetitive neck contraction, flexion or extension, and as a result of medical complications (such as *osteochondroma*, a benign tumour of the bone). Another rare but possible cause is CSF leaks.[3] Yet another cause is from radio frequency nerve ablation. Rarely, occipital neuralgia may be a symptom of metastasis of certain cancers to the spine.[4] There are several areas that have potential to cause injury from compression:

1. The space between the vertebral bones of C1 and C2
2. The atlantoaxial ligament as the dorsal ramus emerges
3. The deep to superficial turn around the inferiolateral border of the obliquus capitis inferior muscle and its tight investing fascia
4. The deep side of semispinalis capitis, where initial piercing can involve entrapment in either the muscle itself or surrounding fascia
5. The superficial side of semispinalis capitis, where completion of nerve piercing muscle and its fascia again poses risk
6. The deep side of the trapezius as the nerve enters the muscle
7. The tendinous insertion of the trapezius at the superior nuchal line
8. The neurovascular intertwining of the GON and the occipital artery

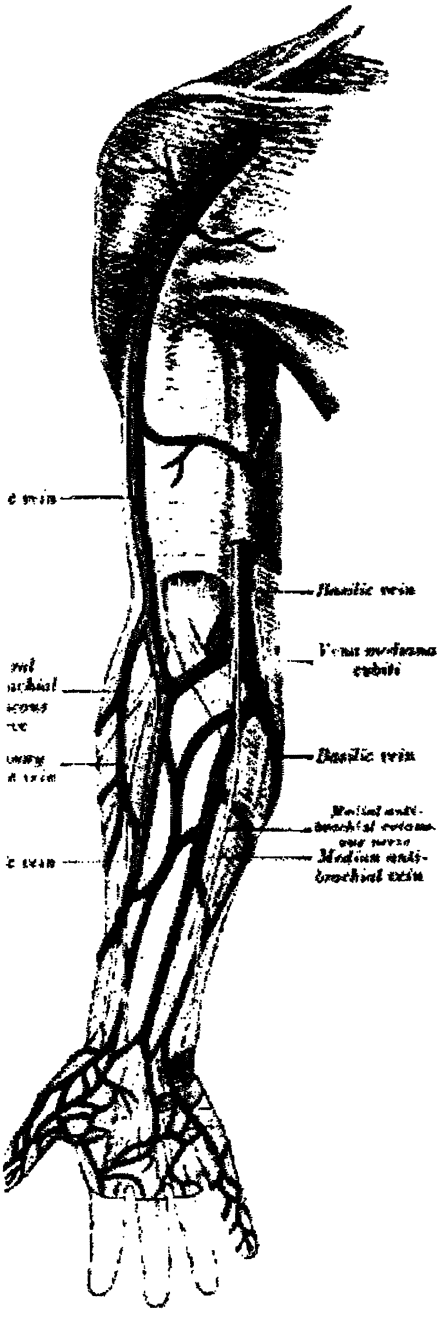
Symptoms

The main symptom of this condition is chronic headache. The pain is commonly localized in the back and around or over the top of the head, sometimes up to the eyebrow or behind the eye. Because chronic headaches are a common symptom of numerous conditions, occipital neuralgia is often misdiagnosed at first, most commonly as tension

Basilic vein

From Wikipedia, the free encyclopedia

Basilic vein



uperficial veins of the upper limb.

Migraine with brainstem aura (basilar-type migraine)

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All topics are updated as new evidence becomes available and our peer review process is complete.
Literature review current through: Aug 2015. | **This topic last updated:** Feb 10, 2015.

INTRODUCTION AND DEFINITION — Migraine with brainstem aura (MBA), previously called basilar-type migraine, is a rare form of migraine with aura wherein the primary signs and symptoms seem to originate from the brainstem, without evidence of weakness. Originally described by Bickerstaff in 1961 as a distinct clinical entity [1], previous descriptions consistent with MBA were given by Aretaeus in ancient Greece and by Gowers in 1907 [1-3].

The terminology used to describe what some have called "Bickerstaff syndrome" has evolved over time; "basilar artery migraine" was replaced by "basilar migraine" and then by "basilar-type migraine." The disorder is now called migraine with brainstem aura [4]. Each subsequent term attempted to maintain the identity of the disorder, while weakening the association with the basilar artery. This evolution has occurred because there is no evidence that the basilar artery is involved, and because some of the symptoms may localize outside the territory of the basilar artery.

This topic will review the clinical manifestations, diagnosis, and treatment of MBA. Other aspects of migraine are discussed separately. (See "Pathophysiology, clinical manifestations, and diagnosis of migraine in adults".)

PATHOPHYSIOLOGY — Most experts now consider MBA as a subset of migraine with aura, and its etiology rests in the theory that cortical spreading depression produces the aura. Cortical spreading depression is a self propagating wave of neuronal and glial depolarization that spreads across the cerebral cortex (see "Pathophysiology, clinical manifestations, and diagnosis of migraine in adults", section on 'Cortical spreading depression'). The difference between MBA and migraine with typical aura is that the location of the aura symptoms in MBA primarily involves the brainstem or the bilateral occipital hemispheres, whereas in typical migraine the aura symptoms are mainly restricted to a unilateral hemisphere. However, cortical spreading depression as the cause of altered local blood flow and metabolism in the brainstem has only been proven in animals [5].

Bickerstaff invoked the vascular hypothesis, the prevailing theory at the time, to explain the symptoms of "basilar artery migraine" that were referable to either the brainstem or the bioccipital hemispheres [1]. In a later publication, he acknowledged that he had "rather loosely termed" this condition basilar artery migraine [6]. In truth, there is no evidence that the basilar artery is involved in the etiology of MBA, and abnormal flow in the basilar artery has never been proven in MBA. Only two cases, one with familial hemiplegic migraine with MBA-like symptoms, and one with MBA, have shown ictal spasm of the basilar artery on angiography [7,8]. Another case was reported with reduced mean flow velocity in both posterior cerebral arteries during a single MBA episode with resolution after the aura [9]. Despite these reports, it is unlikely that reversible ischemia is the source of the prolonged symptoms that occur with MBA.

Genetics — Data are limited regarding the genetic basis of MBA. A mutation in the ATP1A2 gene was found in three first-degree relatives with MBA [10], and another study reported that a single patient suffering from attacks of both MBA and episodic ataxia type 2 had a novel nonsense mutation in the CACNA1A gene, which has been frequently implicated in patients with familial hemiplegic migraine (FHM) [11]. These findings suggest a possible shared pathogenetic mechanism between MBA and FHM (see "Hemiplegic migraine", section on 'Pathophysiology and genetics'). However, in a population-based study from Denmark, no causative mutation

Supplemental Report
Sample #1

MICHAEL M. BRONSHVAG, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM

[redacted]

XXXXXXXX XXXXXXX, [redacted]
XXXXX XXXXXXXXXXX XXXXXXXXX XXXX
XXXX XXX XXXX
XXXXXX XXXX, XX XXXXX-XXXX

XXXXXXXXXXX X XXXXXX, Esq.
XXXXX, XXXXXXXX, XXXXXXX, XXXXXX, XXXX, XXXXXXXX, XXXXXXXX & XXXX, LLP
XXXX XXXXXX XXXXXX, XXXXX XXXX
XXX XXXXXXXXX, XX XXXXX

AGREED MEDICAL REEVALUATION

Re: **XXXXXXXXXX, X**
Date of Injury: [redacted]
Employer: **XXXXXXXXXX XXXXXXX XXXXXX**
WCAB Case No.: **XXXXXXXXXX**
Claim No.: **XXXXXXXX**

Dear All:

XXXXX XXXXXXXXXX was seen for an Agreed Medical Reevaluation on [redacted] at XXXX XXXXXX XXXXXX, XXXXX XXX, XXXXXX, XX XXXXX.

Pursuant to Title 8 Cal Code of Regulations, Section 9795 (b) and (c), this report is submitted as a basic comprehensive medical-legal evaluation, ***ML 101****-94. Time spent face-to-face with the claimant was 45 minutes, record review required 120 minutes and report preparation required 90 minutes. Total time spent on this case was* ***255 minutes.***

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I have seen the claimant, Mr. XXXXX XXXXXXXXXX, previously – [redacted]. At that time I took note of his left upper arm basilic vein thrombosis – apparently improving. I also took note of his complex set of difficulties – head pain, loss of consciousness-issues and also a history of low back issues, kidney stones, and the possibility of seizures, psychological problem, but I had suggested migraine with basilar aura (Bickerstaff's syndrome).

At this time the claimant returns.

I had commented upon greater occipital neuralgia.

The basic issue – however – was the claimant's left upper arm-basilic vein.

At this time I review the current cover letters from Attorney XXXXXX and Specialist XXXXXXX, XXXX. Of note, it is also mentioned that the claimant suffered injury on [redacted] (left shoulder, upper back, internal organs, abdomen and groin). Attorney Dehner asks about doing further testing relevant to the phlebitis issue.

As you will note, I got the history that the claimant had been working as a [redacted] until [redacted]. He described to me two specific injuries ([redacted] and then he referred to a [redacted] injury) and he had told me he had a history of back-spine trauma of a cumulative nature.

The efforts of medical neurologist XXXXXX were noted.

The claimant states that his neck difficulties appear to have resulted from several episodes of airplane turbulence while he was using a heavy (? too heavy ?) helmet (surveillance duties). As a result of the [redacted] event, relevant to airplane turbulence, the claimant had worsening of his left-sided neck symptoms and left-sided posterior head (greater occipital nerve) symptoms.

The diagnosis of "migraine plus trauma" was advanced. A DHE protocol was employed, as described. As a result of the DHE protocol, he developed phlebitis of the left basilic vein and has been placed upon Coumadin therapy.

Of note, the claimant has a history of hemorrhagic-type gastroesophageal difficulties and nonsteroidals are not thought to be a reasonable risk. Coumadin therapy was given and it was thought that the Factor X blocker anticoagulants were too much of a risk.

The basic issues thus were

1 Low back – cumulative.
2 Left-sided neck difficulties.
3 Left posterior head difficulties (greater occipital neuralgia).
4 Recurrent symptoms of a migraine nature.
5 Cervical degenerative joint and disc disease.
6 The issue of seizure versus psychosis or psychological issue (as an explanation for what appear to be syncopal episodes).
7 The ongoing current problem of thrombophlebitis in the left basilic vein.

Accordingly, there are two different areas that I am being tasked to comment upon at this time:

1 The left upper arm phlebitis (per Attorney XXXXXX).
2 Musculoskeletal difficulties involving neck and left arm (XXXX).

The claimant tells me that he is being followed by a hematologist and the most recent studies (which I don't have yet) suggest recurrence of the phlebitis problem-left basilic vein (rather than the hoped-for resolution).

The claimant continues to complain of pain and episodic swelling of the left upper arm in the medial area (where the basilic vein is). Initially the claimant's left hand had been swollen and purplish but this is not evident at this time.

Thus the claimant tells me at this time that as a result of injuries, including [redacted], he has head and neck symptoms, migraine headaches, and low back difficulties. Coumadin therapy is being continues. The claimant has not returned to work and it appears that he will not ever return to his usual and customary job ([redacted]).

TREATMENT EFFORTS

The claimant is being followed by Dr. XXXXXXXX (XXXXXX), Dr. XXXXXXX (neurology – headache – UCSF), and Dr. XXXXX (XXXXXX), plus a hematology specialist. Imaging studies have been done recently.

JOB DESCRIPTION

As noted – above – TSO until [redacted] – no work since, although the claimant is a college student now.

MEDICATIONS

The claimant is being treated with amitriptyline and Coumadin. He avoids nonsteroidals and is not a candidate for the "Factor X inhibitor" anticoagulants. He has been told that he needs to continue on Coumadin because of the apparent residual or recurrent clot activity in the left basilic vein (left upper arm).

COMMENT

As you will note, my previous effort provided a picture of the left basilic vein and discussion of the entity of migraine with basilar aura (migraine with basilar onset, Bickerstaff's syndrome).

It is my understanding that the detailed workup I had commented upon previously did not provide any primary evidence for either a seizure disorder or a psychological-psychiatric problem.

REVIEW OF SYSTEMS

The height is [redacted]. The weight is [redacted] pounds.

The claimant denies other primary evidence of lung or heart disease, high blood pressure or diabetes. There is no history of kidney or liver disease, gastrointestinal problems, weight loss or anemia. The family history is positive for diabetes – mother's side.

PERSONAL AND SOCIAL HISTORY

The claimant is [redacted] with a weight of [redacted] pounds. He does not smoke or drink. He denies drug misuse. He is a life-long Californian, [redacted] and is a senior at [redacted]. He is able to read and write well. He is unable to return to his [redacted]. He does not employ crutches, a cane or a

brace. He has a driver's license, is right-handed, can walk a block, climb a flight of steps, and lift a few pounds.

Accordingly, as I approach the claimant today (repeat evaluation), the problems are

1 Left basilic vein – status – thrombophlebitis.

2 Head pain with migraine features, greater occipital neuralgia features, and the question of seizure versus psychological issue versus migraine with basilar aura (or combination or other).

3 The musculoskeletal issues relative to the [redacted] injury, and the claimant adds that injury had also occurred in [redacted] and cumulatively, with
   a) Neck symptoms
   b) Left shoulder area symptoms (before the basilic vein problem)
   c) Low back symptoms.

The claimant denies bladder, bowel, or erectile deficits.

RECORDS REVIEW

Treating Doctor XXXXXXXX took note of the claimant's head pain, treatment by XXXX – Dr. XXXXXXX, nortriptyline and tapering the gabapentin. Neck range of motion was abnormal. Impression included traumatic brain injury with sequelae, headaches, and chronic neck pain. Improvement with Botox was noted.

Input of Dr. XXXXXXXXXX is noted.

Dr. XXXXXXX - XXXX - thought the claimant had "trauma - head pain." Video EEG of [redacted] was noted. Numbness in fourth and fifth fingers – left hand – was noted. Neck-spine symptoms were mentioned. Treatment efforts of Dr. XXXXX are noted. Dr. XXXXXXX noted candesartan therapy in [redacted]. Dr. XXXXXXX noted the UCSF evaluation of [redacted] for left basilic vein thrombosis. Nonocclusive thrombus was recanalized. Venous flow was described with no evidence of thrombus in the left basilic vein at that time. It was thought that there was evidence for previous thrombus in the left subclavian vein.

The efforts of hematologist Rao are noted. The claimant had described the DHE treatment as otherwise reasonably successful and ultrasound of the left upper extremity had been planned for [redacted] It was thought that NSAID therapy caused the proctitis.

Dr. XXXXXX had stated that the claimant could not take NSAIDs.

The [redacted] imaging study – CT – showed soft tissue densities in the occipital area.

The cisternography of [redacted] was physiologic, and the CTA head study in [redacted] was normal. The brain MRI of [redacted] was described as normal. Review of that study in [redacted] did not document left occipital abnormality.

Dr. XXXXXXX, as of [redacted], diagnosed posttraumatic chronic migraine with improvement with DHE treatment. Nortriptyline was offered. Botox was mentioned and treatment with candesartan. The possible significance of injections causing swelling in [redacted] was noted. Numbness of the left fourth and fifth fingers was noted.

PHYSICAL EXAMINATION

My physical examination of the claimant performed at this time demonstrated no evidence of memory, language or cognitive deficits. Overt anxiety and depression were not documented at this time. The vision and hearing were grossly normal. Blood pressure was measured at 135/80 with a pulse of 80, and no dyspnea, cyanosis, edema, or cough were noted. The eyes, funduscopic exam, and cranial nerves are normal.

There is tenderness to palpation in the left occipital skull area and slight sensory diminution in the distribution of the greater occipital nerve. Overt lumps or bumps are not palpated or appreciated at this time.

ENT exam is otherwise normal. Neck shows no abnormality of trachea, veins, or thyroid. Lungs, heart, and pulses are normal. The abdominal and skin examinations are normal.

The musculoskeletal examination is of note.

To begin with, temporomandibular joints are normal. Neck range of motion shows slight limitation to full range of motion, with comfortable flexion allowed, forward flexion to plus 30 degrees, extension to minus 10 degrees, and lateral flexion bilaterally allowed to plus 10 degrees.

The right shoulder shows a full range of motion. Abduction of the left shoulder past 160 degrees causes symptoms.

Midback is normal. There is complaint of pain on range of motion of low back. Hip rotation is full and straight leg raising in the seated position is full. The balance of the musculoskeletal examination of the spine and legs is normal.

Right arm is normal. There is residual tenderness to palpation in the right medial upper arm area. I do not demonstrate gross or overt phlebitis changes at this time but the claimant notices symptoms in the area.

No sensory or motor neurological deficits are noted. Tone, stance and speech are normal, and the tendon reflexes are 1+. The balance of the neurological examination is unremarkable.

Accordingly, the claimant has been told by his hematologist (Dr. XXX) that there continues to be phlebitis activity in the left basilic area and mention is made of previous involvement of the left subclavian vein area (?).

INITIAL COMMENT

There are records from Dr. XXX (hematologist) and Dr. XXXXXX (gastroenterologist) that would be helpful at this time.

I have been given two different tasks.

1 Evaluate the claimant's left upper arm basilic vein problem (Attorney [redacted]

2 Evaluate this claimant's musculoskeletal-neuromusculoskeletal issues (SCIF – Specialist Valenti).

DIAGNOSTIC IMPRESSION

1 Occlusive phlebitis, left basilic vein – related to DHE procedure (catheter tube in left basilic vein) - ? current status – see discussion.
2 Complex history of headache, dizziness (? syncope ?), seizure, suspected psychological issue (not demonstrated), left greater occipital neuralgia.
3 Musculoskeletal issues involving neck, left shoulder, and low back, without striking neurological deficit.

DISCUSSION

The up-to-date data from Dr. XXX and Dr. XXXXXX are needed. I would prefer to get their data rather than order some tests that they probably have already been done.

Relevant to the claimant's musculoskeletal difficulties, neck, left shoulder, and low back, these are more straightforward. The physical findings are positive (but modest rather than severe). There is some question as to whether these are entirely secondary to the [redacted] injury, or are there contributions from the [redacted] injury, cumulative occupational microtrauma, and anything else (?). If the claimant indeed has active phlebitis in the left basilic vein and possible involvement of the left subclavian vein, these neck, left shoulder, low back issues are in "second position" (less important).

Temporary total disability – The claimant has been off work since the difficulties with the DHE infusion ([redacted]).

Of note, the DHE apparently really helped relevant to the head pain issues. There is discussion of giving DHE again with a catheter placed more centrally (PICC line) (?).

Permanent partial disability-MMI – As noted above, the claimant is most probably permanent, stationary, and ratable – MMI – relevant to primarily neck, left shoulder, and low back issues. I am going to ask for the privilege of having an orthopedist or physical medicine specialist QME evaluate the claimant as well, so my ratings will be as accurate as possible relevant to levels of impairment and the causation-apportionment issues. As noted above, the findings are definite but are not severe and are about ready to rate.

AMA Guides Impairment Rating – This is a complex issue, and some of the questions have not been posed directly to me. It is not clear to me to what extent this claimant's left upper arm-basilic vein-issues are ready to rate (? or not yet ?). His musculoskeletal difficulties (neck, left shoulder and low back) are in line for an AMA Guides rating.

To what extent this claimant's symptoms in the distribution of the left greater occipital nerve can be related to his multiple episodes of head trauma (the bulky helmet – hitting his head against the roof of the plane) needs to be clarified relevant to a rating for headache issues. I do note the claimant has a positive family history of migraine.

Subjective symptoms-complaints – Left upper arm pain, symptoms in neck, left shoulder and low back, head pain.

Objective findings – Impaired range of motion – modest – of neck, left shoulder, and low back. Discomfort and sensory diminution in left occipital area.

Causation – The causation of the claimant's left basilic vein is entirely the DHE procedure, which is a work-related treatment for a work-caused problem. Accordingly, the claimant's basilic vein problem is entirely work-related, without apportionment.

The claimant's neck, left shoulder and low back issues are related to a set of work-related issues (definitely [redacted], perhaps [redacted], and probably cumulative occupational microtrauma). Orthopedic QME input is needed.

The claimant's headaches are a complex combination of the multitrauma described by the claimant, and migraine susceptibility, plus objective findings (post trauma) in the distribution of the left greater occipital nerve.

Apportionment – As noted above, apportionment for anything other than the DHE treatment for the left basilic vein problem is not indicated.

As noted above, the claimant's neck, left shoulder, and low back difficulties are apportionable to the [redacted] injury (on the job) and further clarification is required relevant to the potential role of the [redacted] injury and cumulative efforts and events.

Studies reviewed – Stack of medical records provided at this time (please note I need the newer records) ([redacted]).

Studies performed time – Nothing further at this time.

Studies requested – The newer records, especially those of Dr. XXX and Dr. XXXXXX, and those of Dr. XXXXXXXX and anybody else (I think I have the up-to-date records from [redacted]eadache neurology data).

Research performed at this time – None.

Return to work – Relevant to the [redacted] job, probably not. The claimant is finishing up a college education and this will probably lead to opportunities.

Treatment recommended – This is a complex issue. To begin with, I need to know what Dr. XXX thinks should and shouldn't happen relevant to this claimant's basilic vein and related areas.

Conservative treatment is indicated relevant to neck, left shoulder, and low back (findings are definite but not severe, and I do await orthopedic or physical medicine input). Relevant to the headache issue, and noting that the claimant is being treated in the Fresno area and also at UCSF, I refer and defer at this time. Relevant to the neck, low back and left shoulder area, I again await some further orthopedic input.

Further comment – As noted above, I am requesting the privilege of having the claimant seen by a QME orthopedist or physical medicine specialist. I am requesting any further and newer records. I would like to see the claimant back again in about three to six months. At that time hopefully his left arm vein problem will be cleared up, his head pain issue will be more clarified, and his neck, left shoulder and low back issues will be rated by me.

If questions remain or arise, kindly write me back and I will respond immediately.

I certify that I took the complete history from the claimant, conducted the examination, reviewed all available medical records, and composed and drafted the conclusions of this report. The conclusions and opinions within this report are solely mine. I declare under penalty of perjury that the information contained in this report and its attachments, if any, is true and correct to the best of my knowledge and belief, except as to information that I have indicated I received from others. As to that information, I declare under penalty of perjury that the information

accurately describes the information provided to me and, except as noted herein, that I believe it to be true. In accordance with Labor Code Section 5703(a) (2), there has not been a violation of Labor Code Section 139.3, and the contents of the report are true and correct to the best of my knowledge. This statement is made under penalty of perjury.

Pursuant to 8 Cal. Code Regs. Section 49.2-49.9, I have complied with the requirement for face-to-face time with the client in this evaluation. If necessary, I have discussed apportionment in the body of this report. If I have assigned disability caused by factors other than the industrial injury, that level of disability constitutes the apportionment. The ratio of nonindustrial disability, if any, to all described disability represents my best medical judgment of the percentage of disability caused by the industrial injury and the percentage of disability caused by other factors, as defined in Labor Code Sections 4663 and 4664.

Thanks again, and respectfully,

Michael M. Bronshvag, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM
Date of Report: [redacted]

Sample Report #2

MICHAEL M. BRONSHVAG, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM

[redacted]

XXXXX XXXXXX, [redacted]
XXXXXXXX XXXXXXXX XXXX XXXXXXXXXXXXXX
XXXX XXX XXXX
XXXXXX, XX XXXXX

XXXXXXX XXXXXXX
XXXX XXXXX XXX XXX XXXXXX
XXXXXXX, XX XXXXX

Disability Evaluation Unit
Office of Benefit Determinations
Division of Workers' Compensation
[redacted]

QUALIFIED MEDICAL EVALUATION
ELECTROENCEPHALOGRAM
Panel #1634221

Re	**XXXXXXX, XXXXXXX**
Date of Injury:	[redacted]
Employer:	**XXXXXX XX XXXXXX**
WCAB Case No.:	**Unknown**
Claim No.:	**XXXXXXXX**

Dear Concerned Parties:

XXXXXXX XXXXXXX was seen for a Qualified Medical Evaluation on [redacted], at [redacted]

Pursuant to 8 Cal. Code of Regulations, Section 9795(b) and (c), this report is submitted as an ***ML 104-95*** *Comprehensive Medical-Legal Evaluation involving Extraordinary Circumstances and meets the requirement of four complexity factors. These factors include: (4) 4.50 hours spent on any combination of three of the complexity factors (1)-(3) which shall count as two complexity factors; (6) Addressing the issue of medical causation, which shall count as one complexity factor;(7) Addressing the issue of apportionment, which shall count as one complexity factor.*

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Phone: (800) 458-1261 Fax: (916) 920-2515

*Time spent in direct face-to-face contact with the claimant was .50 hour. Time spent reviewing records required 2 hours. Time spent on research was 2 hours. Time spent preparing the report was 1.50 hours. Total time spent on this case was **6 hours** plus electroencephalogram.*

PROBLEM STATEMENT

XXXXXXX XXXXXXX is [redacted] years old, lives in XXXXXXX, and worked for the [redacted] department - XXXXXX XXXXXX. He had a stroke in [redacted]. The claimant asks me to comment on whether he can go back to his full job or not.

HISTORY of the PRESENT ILLNESS

He is currently doing light rather than full duty (he does phone work and reports). He tells me that he had the onset of difficulties in [redacted]. He noted he was having right eye area pain for several days before noting a problem after going off of work on the day in question (of [redacted]).

He states he had worked for 9 p.m. to 7:30 a.m., and then was noticing in his driveway, difficulties with the left arm (paralyzed). This problem seemed to remit but he noted that he was having recurrent symptoms daily for a week or so. Symptoms in addition to the left arm problems included fatigue, stuttering, and word finding problems.

Accordingly, this claimant's presentation of difficulties circa [redacted] included initially headache behind the right eye for several days, an episode of difficulty in his driveway on [redacted] and recurring symptoms for about a week further. The claimant, himself, tells me that he notes that he was in a high stress work environment and he was handling heavy equipment and was required to do long hours of work. He currently denies any overt psychological - psychiatric difficulties.

He is unaware of similar previous or subsequent stroke - like issues and he is unaware of having had a convulsion.

CURRENT COMPLAINTS

The claimant's current status is not alarming. He notes minimal residual symptoms, of this [redacted] set of difficulties, and is on the job at modified duties. He does not employ crutches, a cane, or a brace.

He can walk 1 to 4 blocks, climb a few steps, and lift 40 pounds. He has had to curtail job and hobbies because of his residual symptoms.

CURRENT TREATMENT

Plavix, Lipitor, and aspirin. He is under the care of Dr. XXXXX and cardiologist Dr. XXXXXX and neurologist XXXXXXX.

MEDICAL REVIEW OF SYSTEMS

The claimant did not provide a history of previous high blood pressure, diabetes, heart difficulties, or similar types of risk factors. The claimant described to me only minimal smoking and drinking.

Previous surgical history - the claimant had stomach surgery (fundoplication - for GERD symptoms in [redacted] He was hospitalized for pneumonia in [redacted]

The claimant has a positive family history of heart disease and hypertension. He, himself, is allergic to penicillin and Bactroban.

JOB DESCRIPTION / EMPLOYMENT HISTORY

As noted above, law enforcement, XXXXXX XXXXXX [redacted] [redacted] - currently doing lighter - desk - telephone work since the events of [redacted]

SOCIAL HISTORY

He is right-handed. He smokes one or no cigars per day, and has a drink no more often than once every two weeks. He denies drug misuse. He is a lifelong California resident, [redacted]nd has BA and further training. He is able to read and write well. He is skilled in law enforcement. He is on the job (telephone reports at this time). He has a driver's license. He is right-handed.

INITIAL COMMENT

Thus, the claimant's initial presenting symptom, while in his car, was left arm difficulty, which has not persisted, but dizziness, confusion, facial numbness, and problems speaking persisted for at least a week. Also, the claimant had described to me previous right eye pain for days (? significance ?). At this point, I turn my attention to the medical records kindly provided.

RECORDS REVIEW

I review the job description ([redacted]). I review the [redacted] occupational data. Dr. XXXXXXX noted the onset of difficulties in the left hand, but he felt that the claimant had

recovered. The imaging study of this claimant (enhanced CT scan) showed multifocal areas of stenosis, especially in the middle cerebral arteries (right greater than left). Patchy - scattered hypo-dense areas were felt to be indicative of infarctions. No evidence of hemorrhagic transformation was described [redacted] Dr. XXXXXXXX had gotten the history of right-sided head pain. The difficulties when he came home from work are noted. The brain MRI [redacted] showed two right temporal lobe - parietal lobe abnormalities. The MRA study showed severe right M1 segment stenosis. The medical records previously do not document clear-cut preexisting vascular abnormalities.

PHYSICAL EXAMINATION

My physical examination of this claimant, performed at this time, demonstrates no overt deficits of memory, language function, or overall intelligence, except that the claimant speaks slowly and hesitantly. I do not document, at this time, overt depressive or anxiety data.

The blood pressure is measured at 129/78, and the pulse (fast) is 111. The height is [redacted] and the weight is [redacted] pounds in a right-handed person. Vision and hearing are grossly normal. Eyes, funduscopic exam, and cranial nerves are normal. ENT exam is normal. Neck shows no abnormality of trachea, veins, or thyroid. Lungs, heart, and pulses are normal. Abdominal and skin examinations are normal.

The musculoskeletal examination - performed at this time - demonstrates no gross or overt derangements.

Relevant to the neurological examination, I do not currently demonstrate clear-cut lateralized sensory or motor deficits at this time. Tone, stance, and speech are normal, and the tendon reflexes are 1+.

Because of actual or possible parenchymal or neurocognitive difficulties, EEG brain wave test is done at this time.

ELECTROENCEPHALOGRAM STUDIES

Please note that I had the benefit of help from technologist XXXXXXX, who did the actual study and provided me with the data.

EEG - The claimant's basic waking clinical rhythm is a low-voltage fast activity. This is usually a normal (variant) rhythm, but sometimes indicates anxiety. The claimant's basic rhythm is interrupted episodically by slowing into the theta range, which does "not" have a lateralized or

paroxysmal flavor. This accordingly, is a borderline abnormal comment nonspecific basic rhythm.

Hyperventilation and photic stimulation did not demonstrate abnormalities. No clear-cut seizure - dysrhythmic abnormalities are noted. Accordingly, this is a borderline EEG of uncertain cause. Clinical correlation is required.

DIAGNOSTIC IMPRESSION

1. Encephalopathy, most probably ischemic, with description of at least two right-sided lesions compatible with ischemic disease (stroke) - no evidence of hemorrhage. Assuming no other evidence is demonstrated of medical - metabolic - ischemic disease, the possibility exists that the problem is autoimmune or inflammatory, however, the most likely possibility is ischemic disease caused by cerebrovascular arteriosclerotic disease.

2. Status post surgery - fundal - plication for gastroesophageal.

DISCUSSION

This claimant has clear-cut evidence of brain parenchymal difficulty. He is currently on limited duty, and his long-term prognosis is not yet established.

The possibility exists that something other than arteriosclerosis is present.

At the present time, I do not have evidence for heart or great vessel disease.

FURTHER STUDIES

The claimant requires at this time:

1. Current MRA - MRI of brain.
2. He requires EKG, chest x-ray, and a set of chemistries and immunochemistries. A serum homocysteine will be added.

I would like to have the privilege of seeing the claimant back again in two to four months, to try and comment further and fully relevant to the important questions, which include:

A. What is his prognosis?
B. What is the causation - apportionment conclusion?

On written approval from the carrier, we will schedule these diagnostic tests.

TEMPORARY TOTAL DISABILITY

The claimant clearly has had some brain impairment, which he appears to be recovering from. It is my understanding that actual work loss has been minimal. The claimant is no longer off of work, but he is doing lighter duties. At the present time, the period of temporary total disability in relationship to the stroke was caused by the stroke.

PERMANENT DISABILITY

Follow up is required

SUBJECTIVE COMPLAINTS

The claimant apparently only has minimal residual difficulty relevant to his [redacted] stroke or stroke-like episode.

OBJECTIVE FACTORS

The claimant has apparently largely, if not entirely, improved. However, the abnormal studies from [redacted] are of note and the slightly abnormal EEG brain wave test at this time is of note as well. Therefore, the objective findings are, at this time, minimal, but still of some concern.

WORK RESTRICTIONS

The claimant has currently been placed on desk duty. Based on the recent history [redacted], and the residual physical findings, this seems reasonable, and I agree with it. I do not at this time feel that the claimant could be returned with safety to his full-time job as a [redacted]

AMA IMPAIRMENT RATING

Pending work up.

CAUSATION

As I understand the process and the presumption, the claimant does not, at this time, have evidence of heart disease. He describes long hours and a stressful job. His brain vascular problem has not yet been fully diagnosed (although arteriosclerosis - typical type - seems most likely). I will report further in this regard.

APPORTIONMENT

Apportionment correlates with causation. The issue of causation has not yet been fully determined, and therefore, apportionment is best described - currently - as pending work up.

TREATMENT RECOMMENDATIONS

The claimant is on medications for secondary stroke prophylaxis, and I agree with this choice. I have not found any other or newer diagnosis and therefore, at this time, do not have any other treatment recommendations.

RETURN TO WORK

The claimant is returned to work at [redacted]. Whether he will be able to return to the full range of his [redacted] or not is a question I do not yet have the answer to.

RESEARCH STUDIES SELECTED

Please note that I have reviewed data on ischemic stroke and inflammatory disorders - see appended articles.

The claimant would also benefit from a lumbar puncture for clarification of inflammatory and infectious issues. Please note that although arteriosclerosis of the vessels serving the brain is common and everything else is rare it is a little unusual for somebody of this young age to be presenting with ischemic disease inside the brain (cerebral vessels) without evidence elsewhere.

I certify that I took the complete history from the patient, conducted the examination, reviewed all available medical records, and composed and drafted the conclusions of this report. The conclusions and opinions within this report are solely mine. I declare under penalty of perjury that the information contained in this report and its attachments, if any, is true and correct to the best of my knowledge and belief, except as to information that I have indicated I received from others. As to that information, I declare under penalty of perjury that the information accurately describes the information provided to me and, except as noted herein, that I believe it to be true. In accordance with Labor Code Section 5703(a) (2), there has not been a violation of Labor Code Section 139.3, and the contents of the report are true and correct to the best of my knowledge. This statement is made under penalty of perjury.

Pursuant to 8 Cal. Code Regs. Section 49.2-49.9, I have complied with the requirement for face-to-face time with the patient in this evaluation. If necessary, I have discussed apportionment in the body of this report. If I have assigned disability caused by factors other than the industrial injury, that level of disability constitutes the apportionment. The ratio of nonindustrial disability, if any, to all described disability represents my best medical judgment of the percentage of disability caused by the industrial injury and the percentage of disability caused by other factors, as defined in Labor Code Section 4663 and 4664.
I further declare under penalty of perjury that the name and qualifications of each person who performed any services in connection with the report, including diagnostic studies, other than clerical preparation, are as follows:

Name	Qualifications
XXXXX XXXXXXX	R.NCS.T (Registered Nerve Conduction Studies Technologist)

Respectfully,

Michael M. Bronshvag, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM

Date of Report: [redacted]
Signed this [redacted] *in* [redacted]

RESEARCH PERFORMED

Please refer to refereed - open source articles I have selected relevant to isolated middle cerebral artery disease, and position dependent cerebral ischemia, and internal carotid - external carotid bypass therapy - therapeutic.

ARTICLES CITED AS RESEARCH FOR THE EVALUATION

J Neurosci Rural Pract. 2012 May;3(2):204-6. doi: 10.4103/0976-3147.98250.

Cerebrogenic tachyarrhythmia in acute stroke.

Kumar AP[1], Babu E, Subrahmanyam D.

Author information

Abstract

The electrocardiac abnormalities following acute stroke are frequent and seen in both ischemic and hemorrhagic stroke. The changes seen in electrocardiogram (ECG) consist of repolarization abnormalities such as ST elevation, ST depression, negative T waves, and QT prolongation. Among tachyarrhythmias, atrial fibrillation is the most common and occurrence of focal atrial tachycardia is very rare though any cardiac arrhythmias can follow acute stroke. We report a case of focal atrial tachycardia following acute ischemic stroke in 50-year-old female without structural heart disease, and their mechanisms and clinical implications.

KEYWORDS:

Atrial tachycardia; atrial fibrillation; insula; ischemic stroke; repolarization abnormality

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When Your Heart Rhythm Isn't Normal

In this article

"Arrhythmia" means your heartbeat is irregular. It doesn't necessarily mean your heart is beating too fast or too slow. It just means it's out of its normal rhythm.

It may feel like your heart skipped a beat, added a beat, is "fluttering," or is beating too fast (which doctors call tachycardia) or too slow (called bradycardia). Or, you might not notice anything, since some arrhythmias are "silent."

Arrhythmias can be an emergency, or they may be harmless. If you feel something unusual happening with your heartbeat, call 911 so doctors can find out why it's happening and what you need to do about it.

Causes and Types of of Arrhythmias

You could have an arrhythmia even if your heart is healthy. Or it could happen because you have:

- Heart disease

- The wrong balance of electrolytes (such as sodium or potassium) in your blood
- Changes in your heart muscle
- Injury from a heart attack
- Healing process after heart surgery

The many types of arrhythmias include:

Premature atrial contractions. These are early extra beats that start in the heart's upper chambers, called the atria. They are harmless and generally don't need treatment.

Premature ventricular contractions (PVCs). These are among the most common arrhythmias. They're the "skipped heartbeat" we all occasionally feel. They can be related to stress or too much caffeine or nicotine. But sometimes, PVCs can be caused by heart disease or electrolyte imbalance. If you have a lot of PVCs, or symptoms linked to them, see a heart doctor (cardiologist).

Atrial fibrillation. This common irregular heart rhythm causes the upper chambers of the heart to contract abnormally.

Atrial flutter. This is an arrhythmia that's usually more organized and regular than atrial fibrillation. It happens most often in people with heart disease and in the first week after heart surgery. It often changes to atrial fibrillation.

Paroxysmal supraventricular tachycardia (PSVT). A rapid heart rate, usually with a regular rhythm, starting from above the heart's lower chambers, or ventricles. PSVT begins and ends suddenly.

Accessory pathway tachycardias. You can get a rapid heart rate because there is an extra pathway between the heart's upper and lower chambers. It's just like if there was an extra road on your way home as well as your usual route, so cars can move around faster. When that happens in your heart, it can cause a fast heart rhythm, which doctors call tachycardia. The impulses that control your heart rhythm travel around the heart very quickly, making it beat unusually fast.

Causes and Types of of Arrhythmias continued...

AV nodal reentrant tachycardia. This is another type of fast heartbeat. It's caused by there being an extra pathway through a part of the heart called the AV node. It can cause heart palpitations, fainting, or heart failure. In some cases, you can stop it simply by breathing in and bearing down. Some drugs can also stop this heart rhythm.

Ventricular tachycardia (V-tach). A rapid heart rhythm starting from the heart's lower chambers. Because the heart is beating too fast, it can't fill up with enough blood. This can be a serious arrhythmia -- especially in people with heart disease -- and it may be linked to other symptoms.

Ventricular fibrillation. This happens when the heart's lower chambers quiver and can't contract or pump blood to the body. This is a medical emergency that must be treated with CPR and defibrillation as soon as possible.

Long QT syndrome. This may cause potentially dangerous arrhythmias and sudden death. Doctors can treat it with medications or devices called defibrillators.

Bradyarrhythmias. These are slow heart rhythms, which may be due to disease in the heart's electrical system. If you experience this, call your doctor.

Sinus node dysfunction. This slow heart rhythm is due to a problem with the heart's sinus node. Some people with this type of arrhythmia need a pacemaker.

Heart block. There is a delay or a complete block of the electrical impulse as it travels from the heart's sinus node to its lower chambers. The heart may beat irregularly and, often, more slowly. In serious cases, you'd get a pacemaker.

Symptoms of Arrhythmias

An arrhythmia can be silent, meaning you don't notice any symptoms. A doctor can find an irregular heartbeat during a physical exam by taking your pulse or through an electrocardiogram (ECG or EKG).

If you do have symptoms, they may include:

- Palpitations (a feeling of skipped heart beats, fluttering or "flip-flops")
- Pounding in your chest
- Dizziness or feeling light-headed
- Fainting
- Shortness of breath
- Chest pain or tightness
- Weakness or fatigue (feeling very tired)

Diagnosis of Arrhythmias

To diagnose an arrhythmia or find its cause, doctors use tests including:

Electrocardiogram -- Also called an EKG or ECG, this test records the electrical activity of your heart. You wear small electrode patches on your chest, arms, and legs for the quick, painless test, which you take in your doctor's office.

Holter monitor -- This is a portable EKG that you'll use for 1 to 2 days. You'll have electrodes taped to yourskin. It's painless and you can do everything but shower while wearing the electrodes.

Event monitor -- If your symptoms don't happen often, your doctor may suggest you wear one of these, usually for about a month. This is a device that, when you push a button, will record and store your heart's electrical activity for a few minutes. Each time you notice symptoms, you should try to get a reading on the monitor. Your doctor will interpret the results.

Stress test -- There are different kinds of stress tests. The goal is to check how much stress your heart can manage before having a heart rhythm problem or not getting enough blood flow to the heart. For the most common type of stress test, you'll walk on a treadmill or pedal a stationary bike at increasing levels of difficulty while you're getting an EKG and getting your heart rate and blood pressure monitored.

Echocardiogram -- This test uses ultrasound to evaluate heart muscle and heart valves.

Cardiac catheterization -- Your doctor will insert a long, thin tube, called a catheter, into a blood vessel in your arm or leg. She will guide it to your heart with help from a special X-ray machine. Then she'll inject dye through the catheter to help make X-ray videos of your heart's valves, coronary arteries, and chambers.

Electrophysiology study -- This test records your heart's electrical activities and pathways. It can help find out what's causing heart rhythm problems and find the best treatment for you. During the test, your doctor will safely reproduce your abnormal heart rhythm and then may give you different medications to see which controls it best, or to see what procedure or device you need to treat it.

Head-up tilt table test -- Doctors use this test to find out what's causing fainting spells. It measures the difference in heart rate and blood pressure when you're standing up or lying down. You'll get this test in a lab. You'll lie on a stretcher, tilted at different angles while you're getting an EKG and specialists are checking your blood pressure and oxygen level.

Treatment of Arrhythmias

Treatment depends on the type and seriousness of your arrhythmia. Some people with arrhythmias don't need treatment. Others may need medication, making lifestyle changes, and surgery.

Drugs that treat arrhythmias include:

Antiarrhythmic drugs. These drugs control heart rhythm and include beta-blockers and calcium channel blockers.

Anticoagulant or antiplatelet therapy. These drugs lower the risk of blood clots and stroke. These include the blood thinner warfarin (Coumadin) or aspirin. Other blood thinners called apixaban (Eliquis), dabigatran (Pradaxa), edoxaban (Savaysa), and rivaroxaban (Xarelto) have been approved to prevent stroke in people with atrial fibrillation.

Everyone is different. Finding the medicine and dose that works best for you may take some time.

- If you notice that your arrhythmia happens more often with certain activities, stop doing them.
- If you smoke, stop.
- Limit alcohol to no more than one drink a day for women, and two drinks a day for men.
- Limit or stop using caffeine.
- Don't use cough and cold medications that have stimulants. Read the label and ask your doctor or pharmacist what medication would be best for you.

What Is Electrical Cardioversion?

If drugs can't control a persistent irregular heart rhythm (such as atrial fibrillation), you might needcardioversion. For this, doctors, give you a short-acting anesthesia, then deliver an electrical shock to your chest wall to allow the normal rhythm to restart.

What Is a Pacemaker?

This device sends small electrical impulses to the heart muscle to keep a safe heart rate. The pacemaker has a pulse generator (which houses the battery and a tiny computer) and wires that send impulses from the pulse generator to the heart muscle.

What Is an Implantable Cardioverter Defibrillator (ICD)?

Doctors mainly use ICDs to treat ventricular tachycardia and ventricular fibrillation, two life-threatening heart rhythms.

The ICD constantly tracks the heart rhythm. When it detects a very fast, abnormal heart rhythm, it delivers an electric shock to the heart muscle to cause the heart to beat in a normal rhythm again. There are several ways the ICD can be used to restore normal heart rhythm. They include:

- **Anti-tachycardia pacing (ATP).** When the heart beats too fast, you get a series of small electrical impulses to the heart muscle to restore a normal heart rate and rhythm.
- **Cardioversion.** You may get a low-energy shock at the same time as the heart beats to restore normal heart rhythm.
- **Defibrillation.** When the heart is beating dangerously fast or irregularly, the heart muscle gets a higher-energy shock to restore a normal rhythm.
- **Anti-bradycardia pacing.** Many ICDs provide back-up pacing to maintain heart rhythm if it slows too much.

What Is Catheter Ablation?

You can think of this procedure as rewiring to fix an electrical problem within the heart.

The doctor will insert a catheter through the leg. The catheter delivers high-frequency electrical energy to a small area inside the heart that causes the abnormal heart rhythm. This energy "disconnects" the pathway of the abnormal rhythm.

Doctors use ablation to treat most PSVTs, atrial flutter, atrial fibrillation, and some atrial and ventricular tachycardias. Some people also need other procedures.

Heart Surgery for Arrhythmias

The Maze procedure is a type of surgery used to correct atrial fibrillation. During this procedure, the surgeon makes a series, or "maze," of cuts in the heart's upper chambers. The goal is to keep the heart's electrical impulses only on certain pathways. Some people may need a pacemaker afterward.

WebMD Medical Reference

The Demise of EC-IC Bypass Surgery

Seemant Chaturvedi, MD reviewing Powers WJ et al. JAMA 2011 Nov 9.

Extracranial-intracranial bypass plus medical therapy for symptomatic carotid occlusion was no more effective than medical therapy alone at preventing stroke and early mortality in a randomized trial.

During the 1980s, a large trial showed that extracranial-to-intracranial (EC-IC) arterial bypass surgery was ineffective for stroke prevention. A lingering question was whether patients with objective evidence of hemispheric hypoperfusion would benefit from surgery. The Carotid Occlusion Surgery Study (COSS) was undertaken to study patients with symptomatic carotid occlusion and evidence on positron emission tomography of increased oxygen extraction ipsilateral to the occlusion, a marker of hemodynamic cerebral ischemia. Participants were randomized to optimal medical therapy with or without EC-IC surgery. The primary outcome was the composite of stroke or death within 30 days after either surgery or randomization, and ipsilateral stroke within 2 years of randomization.

The study was terminated early for futility, when 195 patients had been randomized. At the time of the study's termination, there was only a 2% chance for surgery to be proven effective if the trial were carried to completion. The primary outcome occurred in 21.0% of the surgical group and 22.7% of the nonsurgical group, a nonsignificant difference. Thirty-day rates of ipsilateral stroke were 14.4% in the surgical group and 2.0% in the nonsurgical group. Graft patency was 98% at 30 days and 96% at last follow-up.

COMMENT

In planning the study, the investigators expected a 40% rate of stroke in the nonsurgical group. The fact that the actual rate was about half that is consistent with a recent study of optimal medical therapy for intracranial stenosis (JW Neurol Sep 20 2011), in which the medically treated group did better than expected.

The current findings reinforce the need for aggressive medical therapy for optimal stroke prevention. Only 71% of the nonsurgical group had an LDL level <100 mg/dL at final follow-up, suggesting that a stroke rate even lower than that observed in these medically treated patients is achievable. This study should also stimulate a new trial to investigate aggressive medical therapy for extracranial carotid stenosis. For EC-IC bypass surgery, the door should be closed, with a padlock this time.

Dr. Chaturvedi is Professor of Neurology and Director, Stroke Program, Wayne State University, Detroit.

Cerebral bypass surgery

Overview

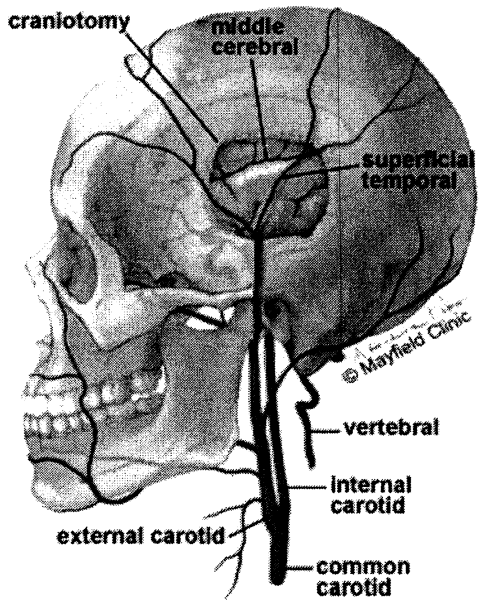
A cerebral bypass is a surgical procedure performed to restore, or "revascularize," blood flow to the brain. A cerebral bypass is the brain's equivalent of a coronary bypass in the heart. The surgery involves connecting a blood vessel from outside the brain to a vessel inside the brain to reroute blood flow around an artery that is narrowed, blocked, or damaged. The main goal of bypass surgery is to restore blood supply to the brain and prevent strokes.

What is cerebral bypass surgery?

Blood carries nutrients and oxygen to the brain through four main arteries: the right and left carotid arteries and the right and left vertebral arteries. Poor delivery of blood flow reduces the brain's ability to function. Called cerebrovascular insufficiency, a lack of blood supply leads to transient ischemic attacks (TIA), stroke, and ultimately brain cell death. In a cerebral artery bypass, the surgeon reroutes blood flow around a blocked or damaged artery to improve or restore blood flow to an oxygen-deprived (ischemic) area of the brain. A cerebral bypass can be performed in a variety of ways depending on where the blockage has occurred, the underlying condition being treated, and the size of the brain area to be revascularized. In general, there are two types of bypasses:

The first type uses a vessel graft – a length of artery or vein harvested from somewhere else in the body. The graft is connected above and below the blocked artery so that blood flow is rerouted (bypassed) through the graft. Common vessels used as a graft are the saphenous vein in the leg or the radial or ulnar arteries in the arm. A separate incision is required to harvest the graft. Next, one end of the graft is connected to the external carotid artery (ECA) in the neck and then tunneled under the skin in front of the ear to the scalp. A hole is cut in the skull through which the graft is passed and connected to an artery in the brain. This method is typically used when a large (high-flow) artery is affected or needs to be sacrificed to treat a tumor or aneurysm.

The other type does not use a vessel graft but a healthy donor artery that flows in the scalp or face. The donor artery is detached from its normal position on one end, redirected to the inside of the skull, and connected to an artery on the surface of the brain (Fig. 1). The scalp artery now supplies blood to the brain and bypasses the blocked or damaged vessel. This method is typically used when a smaller (low-flow) artery has narrowed and is incapable of delivering enough blood to the brain.



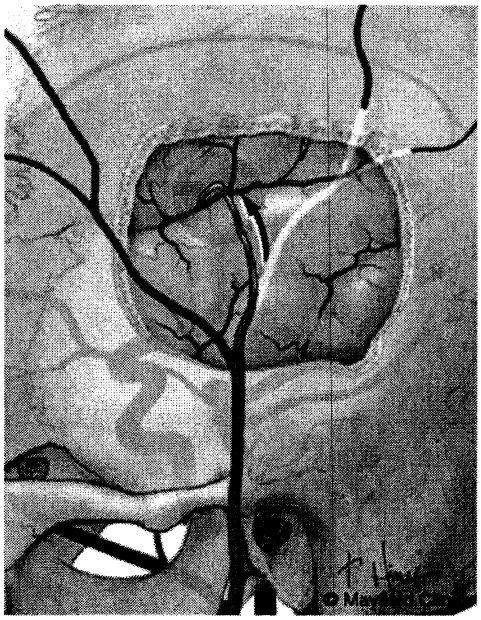


Figure 1. In a cerebral bypass surgery, an artery from outside the skull is connected to an artery inside the skull through a craniotomy. A donor artery, usually the superficial temporal artery (STA), is freed from its normal position on the scalp and connected to the middle cerebral artery (MCA) on the surface of the brain.

The most common type of bypass is the STA-MCA (superficial temporal artery to middle cerebral artery) bypass. The superficial temporal artery (STA) normally provides blood to the face and scalp. You can feel the pulse of the STA in front of your ear. The middle cerebral artery (MCA) normally provides blood to the frontal, temporal and parietal lobes of the brain. Blood flow through the MCA is often reduced when narrowing of the internal carotid artery occurs. In an STA-MCA bypass, the STA (donor vessel) is rerouted from the scalp, passed through a hole in the skull, and connected to the MCA (recipient vessel) above the blockage to restore blood flow to the brain. If the STA is too small or unsuitable, another vessel such as the occipital artery may be used.

Both types of bypasses require creating a hole in the skull to pass the vessel graft or scalp donor artery from outside the skull to the cerebral artery inside the skull. Thus, this surgery is also called an extracranial–intracranial bypass (EC-IC bypass).

Who is a candidate?

You may be a candidate for a cerebral bypass if you have:

- **an aneurysm, tumor, or atherosclerotic plaque that is not treatable by endovascular or other means**
- **failure of medication to control TIA symptoms or stroke**
- **imaging tests (angiogram, CTA, MRA) that show arterial stenosis or occlusion**
- **cerebral blood flow studies (CT perfusion, PET, SPECT) that show arterial stenosis is causing insufficient blood flow to the brain**

Cerebral bypass may be helpful in restoring blood flow and reducing the risk of stroke in conditions such as:

- **Moyamoya disease: a narrowing of the internal carotid arteries at the base of the brain that can cause multiple strokes or hemorrhages. To compensate for the narrowing arteries, the brain creates collateral blood vessels in an attempt to deliver oxygen-rich blood to deprived areas of the brain. A bypass can restore blood flow to the brain and prevent future strokes.**
- **Aneurysm: a bulge or ballooning of an artery wall. Some giant, fusiform, or dissecting aneurysms cannot be treated with surgical clipping or endovascular coiling. In such cases, the parent artery must be sacrificed and the blood flow bypassed for the aneurysm to be effectively treated.**
- **Skull base tumor: a tumor can grow where the major vessels enter the skull and surround or invade the artery. Removing the tumor may require sacrificing the encased artery and bypassing the blood flow.**
- **Carotid artery stenosis or occlusion: a narrowing or blockage of the carotid artery in the neck caused by atherosclerotic plaque deposits in the vessel wall.**
- **Intracranial arterial stenosis: a narrowing or blockage of an artery inside the skull that supplies blood**

Tilt-Table Testing

Author: James V Talano, MD, MBA; Chief Editor: Kartheinz Peter, MD, PhD more...

Updated: Nov 20, 2014

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References

Background

The tilt-table test is a simple, noninvasive, and informative test first described in 1986 as a diagnostic tool for patients with syncope of unknown origin.[1] It is usually performed in hospital electrophysiology departments with the endpoint of reproducing syncope and subsequent appropriate therapy.

The causes of syncope have been divided into 6 major categories, as listed below.[2] After a careful history and physical examination, tilt-table testing is particularly helpful in confirmation of the etiology of syncope dysfunction of the autonomic nervous system, encompassing primary or secondary dysautonomias, postural orthostatic tachycardia syndrome (POTS), and vasodepressor or vasovagal syncope. Other venues of investigation, such as a 12-lead electrocardiogram, orthostatic blood pressure readings, Holter/event recording, serum glucose and electrolytes, echocardiography, and psychiatric and/or neurology consultation should be considered prior to tilt-table testing to rule out malignant dysrhythmic, metabolic, cardiac mechanical, or psychological/neurological etiologies of syncope.[3]

The 6 major categories of syncopal etiologies [2]

Neurological disorders

- Vertebrobasilar transient ischemic attacks
- Subclavian steal syndrome
- Normal pressure hydrocephalus
- Seizure disorders

Metabolic disorders

- Hypoxia
- Hyperventilation
- Hypoglycemia

Mechanical heart disease

- Aortic stenosis
- Mitral stenosis
- Global ischemia
- Aortic dissection
- Pulmonic dissection
- Obstructive cardiomyopathy
- Left atrial myxoma
- Prosthetic valve dysfunction
- Pulmonary embolus
- Pulmonary hypertension

Cardiac arrhythmias/brady arrhythmias/tachyarrhythmias

- Bradycardia/pauses
- Sinus node dysfunction
- AV conduction disease

Psychiatric disorders

- Panic attacks
- Hysteria

Autonomic Nervous System Dysfunction

- Primary and secondary dysautonomias
- Postural orthostatic tachycardia syndrome (POTS)
- Postural orthostatic hypotension
- Vasodepressor or vasovagal syncope

Indications

Consider tilt-table testing in patients with signs or symptoms suggestive of orthostatic hypotension, vasodepressor or vasovagal syncope, postural orthostatic tachycardia, or when other causes of syncope have been eliminated. In general, consider tilt-table testing for patients with the following issues:

- Hypotension (unexplained)
- Tachycardia when standing
- Pallor when upright
- Orthostatic palpitations
- Dizziness (unexplained)
- Lightheadedness
- History of frequent unexplained falls
- History of episodes of fainting or loss of consciousness

The ACC expert consensus document for tilt-table testing including indications was first published in 1996.[4] The European Society of Cardiology formed a taskforce to update guidelines for the diagnosis and management of syncope in 2001, which was revised in 2009.[3] It includes indications for tilt-table testing with classes of recommendation and levels of evidence (see Table 1).

Table 1. European Society of Cardiology 2009 Indications for Tilt-Table Testing[3](Open Table in a new window)

Recommendations	Class	level
Tilt table is indicated in the case of an unexplained single syncopal episode in high-risk settings (eg, occurrence of, or potential risk of physical injury or with occupational implications)or recurrent episodes in the absence of organic heart disease, after cardiac causes of syncope have been excluded	I	B
Tilt testing is indicated when it is of clinical value to demonstrate susceptibility to reflex syncope to the patient	I	C
Tilt testing should be considered to discriminate between reflex and orthostatic hypotensive syncope	IIa	C
Tilt testing may be considered for differentiating syncope with jerking movement from epilepsy	IIb	C
Tilt testing may be indicated for evaluating patients with recurrent unexplained falls	IIb	C
Tilt testing may be indicated for evaluating patients with frequent syncope and psychiatric disease	IIb	C
Tilt testing is not recommended for assessment of treatment	III	B
Isoproterenol tilt testing is contraindicated in patients with ischemic heart disease	III	C

Contraindications

Contraindications to tilt-table testing include the following:[2]

- Coma

- Feeble patient unable to stand
- Lower extremity fractures
- Severe anemia
- Recent stroke (within seven days)
- Recent myocardial infarction
- Severe proximal cerebral or coronary arterial disease
- Critical mitral or aortic stenosis
- Left ventricular outflow tract obstruction
- Hypotensive shock
- Tachyarrhythmias
- Severe metabolic acidosis
- Electrolyte imbalance
- End-stage renal failure
- Severe heart failure

Technical Considerations

Complication Prevention

Avoid invasive intra-arterial blood pressure monitoring during tilt-table testing because catheterization may provoke a vasovagal reaction. Use a manual sphygmomanometer or digital plethysmography.[5]

Outcomes

Generally tilt-table testing is safe, but complications may occur related to decreased perfusion of the heart, including the following:

- Electrocardiographic changes of transient myocardial ischemia with or without angina.
- Vasospasm with isoproterenol administration.[6]
- Occasionally, cardiac arrhythmias result in termination of the test, such as advanced atrioventricular block (second or third degree), severe bradycardia or pauses, atrial fibrillation, or tachyarrhythmias.

Complications may also occur related to decreased perfusion of the brain, including the following :

- Seizures from prolonged hypotension (this is a transient phenomenon and not indicative of a seizure disorder)
- Rarely, transient ischemic attacks or strokes occur.
- Transient mental confusion can occur.
- Patients may also experience nonspecific symptoms such as nausea or anxiety.

Tilt-Table Testing

Author: James V Talano, MD, MBA; Chief Editor: Karlheinz Peter, MD, PhD more...

Updated: Nov 20, 2014

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- Hysteria

Autonomic Nervous System Dysfunction

- Primary and secondary dysautonomias
- Postural orthostatic tachycardia syndrome (POTS)
- Postural orthostatic hypotension
- Vasodepressor or vasovagal syncope

Indications

Consider tilt-table testing in patients with signs or symptoms suggestive of orthostatic hypotension, vasodepressor or vasovagal syncope, postural orthostatic tachycardia, or when other causes of syncope have been eliminated. In general, consider tilt-table testing for patients with the following issues:

- Hypotension (unexplained)
- Tachycardia when standing
- Pallor when upright
- Orthostatic palpitations
- Dizziness (unexplained)
- Lightheadedness
- History of frequent unexplained falls
- History of episodes of fainting or loss of consciousness

The ACC expert consensus document for tilt-table testing including indications was first published in 1996.[4] The European Society of Cardiology formed a taskforce to update guidelines for the diagnosis and management of syncope in 2001, which was revised in 2009.[3] It includes indications for tilt-table testing with classes of recommendation and levels of evidence (see Table 1).

Table 1. European Society of Cardiology 2009 Indications for Tilt-Table Testing[3](Open Table in a new window)

Recommendations	Class	level
Tilt table is indicated in the case of an unexplained single syncopal episode in high-risk settings (eg, occurrence of, or potential risk of physical injury or with occupational implications)or recurrent episodes in the absence of organic heart disease, after cardiac causes of syncope have been excluded	I	B
Tilt testing is indicated when it is of clinical value to demonstrate susceptibility to reflex syncope to the patient	I	C
Tilt testing should be considered to discriminate between reflex and orthostatic hypotensive syncope	IIa	C
Tilt testing may be considered for differentiating syncope with jerking movement from epilepsy	IIb	C
Tilt testing may be indicated for evaluating patients with recurrent unexplained falls	IIb	C
Tilt testing may be indicated for evaluating patients with frequent syncope and psychiatric disease	IIb	C
Tilt testing is not recommended for assessment of treatment	III	B
Isoproterenol tilt testing is contraindicated in patients with ischemic heart disease	III	C

Contraindications

Contraindications to tilt-table testing include the following:[3]

- Coma
- Feeble patient unable to stand
- Lower extremity fractures
- Severe anemia
- Recent stroke (within seven days)
- Recent myocardial infarction
- Severe proximal cerebral or coronary arterial disease
- Critical mitral or aortic stenosis
- Left ventricular outflow tract obstruction
- Hypotensive shock
- Tachyarrhythmias
- Severe metabolic acidosis
- Electrolyte imbalance
- End-stage renal failure
- Severe heart failure

Technical Considerations

Complication Prevention

Avoid invasive intra-arterial blood pressure monitoring during tilt-table testing because catheterization may provoke a vasovagal reaction. Use a manual sphygmomanometer or digital plethysmography.[3]

Outcomes

Generally tilt-table testing is safe, but complications may occur related to decreased perfusion of the heart, including the following:

- Electrocardiographic changes of transient myocardial ischemia with or without angina.
- Vasospasm with isoproterenol administration.[3]
- Occasionally, cardiac arrhythmias result in termination of the test, such as advanced atrioventricular block (second or third degree), severe bradycardia or pauses, atrial fibrillation, or tachyarrhythmias.

Complications may also occur related to decreased perfusion of the brain, including the following :

- Seizures from prolonged hypotension (this is a transient phenomenon and not indicative of a seizure disorder)

- Rarely, transient ischemic attacks or strokes occur.
- Transient mental confusion can occur.
- Patients may also experience nonspecific symptoms such as nausea or anxiety.

- ARTICLES

Atherosclerotic disease of the middle cerebral artery.

1. J Bogousslavsky,
2. H J Barnett,
3. A J Fox,
4. V C Hachinski and
5. W Taylor

Abstract

Three hundred and fifty-two patients with atherosclerotic middle cerebral artery stenosis (MCAS, 53%) or occlusion (MCAO, 47%) have been systematically studied. The study involved all patients entered into the EC/IC Bypass Study with isolated MCA disease or a tandem lesion predominating in the MCA ipsilateral to the ischemic events (18 patients with a tandem lesion of greater magnitude in the internal carotid artery were not included). The Asian patients represented 58% of all Asians entered into the EC/IC Bypass Study, whereas the white patients represented 18% of all whites and the black patients 34% of all blacks. Isolated TIAs were less frequent in MCAO (12%) than in MCAS (34%). Warning TIAs before a stroke occurred in one third of the cases. Presentation with stroke or isolated TIA was not influenced by sex, age, level of MCA obstruction, collateral circulation nor associated carotid disease. In MCAS, no major difference in presentation was found between severe and moderate stenosis. Pure motor hemiparesis occurred in 15% and pure sensory stroke in 2% of the patients with stroke and 30% of the MCA territory infarcts were small and limited to the lenticulocapsular area, confirming that so-called lacunar infarcts may be due to large vessel disease. During follow-up (42 months) of 164 medically-treated patients, further cerebrovascular events (TIA and stroke) occurred in 11.7% of the patients per year. In MCAO the stroke rate was 10.1% per patient-year and the ipsilateral infarct rate was 7.1% per patient-year. In MCAS, the stroke rate was 9.5% per patient-year and the ipsilateral stroke rate was 7.8% per patient-year.(ABSTRACT TRUNCATED AT 250 WORDS)

Neurology. 1985 Jul;35(7):975-82.

Occlusive disease of the middle cerebral artery.

Caplan L, Babikian V, Helgason C, Hier DB, DeWitt D, Patel D, Stein R.

Abstract

We studied 20 patients with severe occlusive disease of the mainstem middle cerebral artery (MCA) or its major division branches, and 25 patients with internal carotid artery (ICA) disease. MCA disease patients were more often black, female, younger, and had fewer TIAs than the ICA disease patients. Neurologic signs in patients with MCA disease evolved progressively during days to weeks, whereas ICA disease patients more often had an acute onset of nonprogressive deficits. CT commonly showed restricted subcortical or wedge-shaped infarcts in MCA disease patients. All MCA disease patients had stroke, but 40% of ICA disease patients had no infarction. MCA lesions usually affected the mainstem MCA or its major superior division. Patients with MCA disease seldom had recurrent ischemia in the same vascular territory as the stroke and had a low incidence of subsequent cardiac death.

PMID:

-

J Neurol Neurosurg Psychiatry. 2004 May; 75(5): 727–732.

doi: 10.1136/jnnp.2003.022574

PMCID: PMC1763587

Isolated middle cerebral artery disease: clinical and neuroradiological features depending on the pathogenesis

P Lee, S Oh, O Bang, I Joo, and K Huh

Author information ▶ Copyright and License information ▶

Supplemental Rpt Sample #2

MICHAEL M. BRONSHVAG, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM

[redacted]

XXXXX XXXXXX, Adjuster
XXXXXX XXXXXX XXXXXXXXXX
XXXX XXX XXXX
XXXXXX, XX XXXXX

XXXXX XXXXXXX, Esq.
Law Offices of XXXXXXXX X. XXXXXXX
XXXX XXXX XXXXX XXXXXX, XXXXX XXX
XXXXX XXX, XX XXXXX-XXXX

XXXXXX X. XXXX, Esq.
XXX XXX XXXXXXX XX XXXXXX X XXXX.
XXXX XXXXXX XXXXXX, XXXXX XXX
XXXXXX, XX XXXXX

QUALIFIED MEDICAL REEVALUATION

Re:	**XXXXXXX, XXXXXXX**
Dates of Injury:	[redacted] **to** [redacted]
Employer:	**XXXXXX XX XXXXXX (XXXXXXX XXXXXXXXXX)**
EAMS #s:	**XXXXXXXXXX, Unassigned**
Claim #s:	**XXXXXXX, XXXXXX**

Dear All:

XXXXXXX XXXXXXX was seen for a Qualified Medical Reevaluation on [redacted] at XXXX XXXXXX XXXXXX XXXXX XXX, XXXXXX, XXXXXXXXXX XXXXX.

Under penalty of perjury, this report is submitted pursuant to 8 Cal. Code Regs. Section 9795(b) & (c) as a ***ML-104****-95 Comprehensive Medical Legal Evaluation Involving Extraordinary Circumstances and meets the requirement of four complexity factors. These include:*
(5) 6+ hours spent on any combination of three of the complexity factors (1)-(3) which shall count as 3 complexity factors;
(6) Addressing the issue of medical causation, which shall count as one complexity factor

---

11010 White Rock Road, Suite 120, Rancho Cordova, CA 95670
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Time spent in direct, face to face contact was 45 minutes. Time spent reviewing records required 240 minutes. Time spent on research was 120 minutes. Time spent preparing the report was 120 minutes. Total time spent on this case was ***525 minutes plus EEG.***

INTRODUCTION

As you will note, I have seen the claimant previously. I, at this time, turn my attention to the [redacted] Joint Panel Qualified Medical Examination Letter - thank you.

As you will note, I had seen the claimant in [redacted] wrote an initial report and then in [redacted] supplemental. Mention is made of two dates of injury ([redacted] stroke, and cumulative issue from [redacted] through [redacted] (stroke, heart trouble, PVC's, headaches, bilateral finger numbness, right wrist pain, upper back pain, lower back pain, bilateral hip pain, bilateral knee pain, ankle pain, GERD, hiatal hernia, bradycardia, and sleep problems).

It is stated that this cumulative trauma claim has been denied. It is stated that the claimant will be evaluated separately for orthopaedic issues.

The claimant tells me that he has not worked since the spring of [redacted] He tells me that he is weak on the left side of his body. He is sensitive to light. He has had palpitations. He sleeps restlessly and has been provided with positive pressure (CPAP), which appeared to help his PVC's.

HISTORY OF PRESENT ILLNESS

The claimant had initially presented for stroke issues with a date of [redacted] He states that his problems included a high-stress work environment, heavy equipment issues, and prolonged work hours.

He notes that a loop recorder for cardiac rhythms was provided and he is not certain as to what it did and did not show. As you will note, when I saw the claimant in [redacted], in [redacted], I took note of his stroke or stroke-like episode of [redacted] I noted that the claimant had been placed on desk duty, and I personally was not enthusiastic as of [redacted] to send him back to his full job as a [redacted]. I took note of secondary stroke prophylaxis. I noted that the initial event of [redacted] was probably an ischemic stroke-like event. The imaging study demonstrated two right sided brain lesions compatible with ischemic disease - stroke, and no evidence of hemorrhage. I felt the most likely possibility was ischemic disease caused by cardiovascular - cerebrovascular arteriosclerotic disease.

It took note of the fundal - plication in [redacted] for treatment of gastroesophageal reflux issues. I had noted that the claimant had clear-cut evidenced of brain parenchymal difficulty. I noted that the MRI of the brain performed without and with contrast in [redacted] demonstrated the right temporal and parietal infarctions - no acute abnormalities. I noted that the EKG was borderline abnormal relevant to sinus bradycardia. His homocysteine level (sometimes a stroke indicator) was high - normal and the ANA was normal. I felt that the claimant should be examined by a QME cardiologist to determine to what extent the next step should be taken. My review of this and my talking to the claimant indicates that a loop device had been provided in [redacted]. I note that he was provided with CPAP therapy in [redacted], which seemed to improve his PVC's (premature ventricular contractions).

CURRENT TREATMENT

The claimant is currently being treated with aspirin and Dexilant, Plavix had been discontinued.

The notes of the claimant's Psychiatric Doctor - XXXXXXXXX - is noted.

MEDICAL REVIEW OF SYSTEMS

Apart from as noted above, the claimant does not provide a positive history of lung or heart disease, high blood pressure, or diabetes. He denies or is unaware of kidney or liver disease, gastrointestinal problems, weight loss or anemia. I, again, took note of the [redacted] fundoplication. The claimant had been treated for pneumonia in [redacted]

Accordingly, as I approach the balance of this evaluation, the issues appear to be the event of [redacted] which certainly sounds like ischemic brain disease, presumably embolic from the heart or involving middle cerebral artery, plus mention of slow pulse and arrhythmia, plus taking note of CPAP treatment for sleep apnea and noting the fundal plication surgery of [redacted]

The medical records state that the actual stroke was in [redacted] but symptoms apparently stuttered in [redacted] The more recent efforts of attorneys Epperly and Lusk are noted.

JOB DESCRIPTION

As noted previously - [redacted] XXXXXX XXXXXX between [redacted] and [redacted] - apparently the last four months at [redacted]

SOCIAL HISTORY

The height is [redacted] The weight is [redacted] pounds. The claimant is under the care of Dr. XXXXX (primary), cardiologist XXXXX, and neurologist XXXXXX (it was XXXXXXX).

The family history is positive for heart disease and hypertension. The claimant is allergic to penicillin and Bactroban. He smokes an occasional cigar. He has an occasional drink. He denies drug misuse. He is a lifelong Californian - is married and has college and advanced studies. He is skilled in [redacted] He feels he could do telephonic reports, and he believes he could do full duty as well.

He does not employ crutches, a cane, or a brace. He has a driver's license. He is right-handed. He can walk one to four blocks but gets tired, and can climb a flight of steps but gets tired. He can lift 40 pounds.

In summary, this claimant, who had what sounds like one or two stroke or stroke-like episodes in [redacted] has not fully clarified cardiovascular difficulties, has sleep apnea, whose treatment improves PVC's and has a history of fundoplication, at this time, feels he could return to full duties as a [redacted] (?). He adds that the [redacted] - [redacted] stroke - stroke-like episode occurred when he was on duty. A full box of medical records is reviewed.

RECORDS REVIEW

It is my understanding that the musculoskeletal - orthopaedic - chiropractic challenges are challenges for other doctors. Chiropractic efforts of Dr. XXXXXXXX in [redacted] are noted. Stomach difficulties are commented upon. The September [redacted] MRI showed the temporal - parietal right-sided lesions. I noted the potential need for a lumbar puncture. Left arm and leg numbness were described in [redacted] - (not [redacted] BIBA - I guess that means brought in by ambulance). With the [redacted] event, right-sided head pain was noted. The [redacted] depo of the claimant is noted. The recorded statement is noted. The [redacted] esophageal studies are noted. Minimal sliding hiatal hernia was mentioned. Moderate intolerance of air distention of the stomach and duodenum was noted. Gastritis was noted, but Helicobacter was not demonstrated. Hemorrhoid surgery is noted - in [redacted] Efforts of Drs. XXXXXX and XXXXX in [redacted] are noted. It was noted that the pH study showed only minimal degree of gastroesophageal reflux and was poor correlation between symptoms of heartburn and the reflux events. Attorney Lusk took note of the input of Drs. XXXXXX, XXXXXXX, and XXXXXXXXX. Dr. [redacted] (American Board of Neurology) had noted that the claimant had a history of a stroke, and some back problems. Hypercoagulable state was mentioned, and the claimant was described to have MTH - FR- gene mutation and hyper - homocystenimea. I reviewed the efforts of Dr. XXXXXXXXX and it appears that the exam was normal, but Dr.

XXXXXXXXX did not provide a conclusionary sentence or paragraph. The [redacted] pneumonia hospitalization is noted. The skin efforts of Dr. XXXXXX are note. Actually, medicine - these symptoms mention that the claimant was evaluated on [redacted] for symptoms that had begun with a right-sided headache one and a half weeks prior. The notes describe right MCA lesion (middle cerebral artery).

(Doctor's Comment - the problems are in the distribution of the right middle cerebral artery, but could represent embolus from a more proximal source such as the heart.)

Palpitations and dizziness were noted in [redacted] Holter monitoring study was advised in [redacted] Further data of Drs. XXXXX and XXXXXX ([redacted] is noted. Abdominal ultrasound in [redacted] was unremarkable. Treadmill test in [redacted] was noted to be negative. Further [redacted] and [redacted] data are noted. The [redacted] surgical procedure takes note of severe refractory gastroesophageal reflux disease and hiatal hernia, and laparoscopic Nissen fundoplication and laparoscopic hiatal hernia repair. The [redacted] data describe the arrhythmias at the time of hospitalization for the stroke symptoms. The bubble echo of [redacted] took note of a family history for coronary artery disease. Neurological deficits had remitted. The rhythm strips demonstrate rare PVC's. Gout was mentioned. The claimant had difficulties with Plavix. The MR A angiogram demonstrated right middle cerebral disease. Left-sided foot drop was mentioned. Mild left atrial enlargement was mentioned. It was felt that the claimant had normal factor to prothrombin genetics. A C6-7 7-T polymorphism at the MTHFR general as felt not to be pertinent. The need for transesophageal echocardiogram was mentioned. The PEE was ultimately done on the [redacted] - normal.

In summary, these records document actual or potential irregular heartbeat dating back perhaps [redacted] years. His gastroesophageal studies and treatments are noted. Apparently, ultimately, the endoscopic data were minimally abnormal at worst and did not correlate well with his symptoms. Cardiac workup including loop studies has been noted. The genetic evaluation apparently demonstrated only rather minimal abnormalities. The most pertinent problem relevant to the claimant's brain was a somewhat unusual finding, namely isolated middle cerebral artery arteriosclerosis or similar derangement.

As noted - see research below - this is somewhat unusual, but does happen.

PHYSICAL EXAMINATION

At this point, I turn my attention to the physical examination.

My physical examination demonstrates a blood pressure of 151/99 (slightly elevated) with a pulse of 65 (relatively normal) - no arrhythmia palpated. No cognitive memory or language deficits are noted at this time. The claimant did not demonstrate overt psychological - psychiatric difficulties. Vision and hearing are grossly normal. Eyes, funduscopic exam, and cranial nerves are normal. ENT exam is normal. Neck shows no abnormality of trachea, veins, or thyroid. Lungs, heart, and pulses are normal. Abdominal and skin examinations are normal.

The musculoskeletal examination does not demonstrate any derangements or rheumatoid findings. The neurological examination - currently - demonstrates no gross or overt abnormal or lateralized findings. Tone, stance, and speech are normal, and the tendon reflexes are 1+.
I did not document any overt neurocognitive deficits.

I now do an EEG with the help of technologist XXXXXXX.

The basic waking rhythm is a combination of 9 Hz. alpha and low voltage fast activity. This is a normal basic-background rhythm. Drowsiness and sleep are not obtained. No abnormal waves, assymetries or paroxysmal changes are seen.

This is a normal EEG.

DIAGNOSTIC IMPRESSION

1. Episode, [redacted] of right-sided brain lesions (two separate lesions) - with occlusive disease of right middle cerebral artery.

(Although everybody talks about middle cerebral artery disease, usually this middle cerebral artery problem is usually instead caused by either a carotid occlusion or an embolus from the heart. However, this claimant is the exception that proves the rule.)

2. Sleep apnea symptoms - CPAP treatment.

3. Irregularity of heartbeat - not fully defined.

4. Gastroesophageal disorder - status post fundus plication - most recent endoscopic findings apparently relatively normal.

INITIAL DISCUSSION

This claimant has occlusive disease of the right middle cerebral artery. His somewhat stuttering course over a week - two weeks in [redacted] matches up with that somewhat unusual anatomic - pathologic finding.

To what extent this correlates with irregular heartbeat is a puzzle. There are some articles (see below) of arrhythmia caused by temporal lobe lesions. His EEG was normal today.

TEMPORARY TOTAL DISABILITY

This claimant had the onset of stroke in [redacted], worked on at [redacted] for four months, but not since. The claimant wants to go back to work. At this point, he remains TTD.

PERMANENT DISABILITY

The claimant is probably MMI relevant to the two strokes on the right side of the brain. However, it is not entirely clear to what extent further treatment is required relevant to his persisting partial occlusion of the right middle cerebral artery - see discussion below.

SUBJECTIVE COMPLAINTS

Gastroesophageal symptoms - see discussion above.

No current stroke-like or seizure-like symptoms. No overt neurological deficits or neurocognitive deficits. The claimant specifically denied neuropsychological - neuropsychiatric issues. He did complain of pain in neck, shoulders, back, and knees.

OBJECTIVE FACTORS

Borderline high blood pressure. The pulse was normal today. Neurological examination was normal today.

AMA IMPAIRMENT RATING

Neurologically speaking, this claimant does not currently have a neurologically proven hemiparetic deficit that is ratable. The issue of his partial vascular occlusive disease of the right middle cerebral artery is not fully clarified - yet.

CAUSATION

Relevant to this claimant's occlusive right middle cerebral artery disease, the claimant describes a stressful environment. I did not do a psychiatric examination of the claimant. I think it would be essentially impossible to prove that occupational causation was not present (presumption). Causation is occupational.

APPORTIONMENT

At the present time, I identify no basis for apportionment.

FUTURE MEDICAL CARE

I have no specific recommendations relevant to medications. It is not clear to me whether some sort of an endoscopic approach to the middle cerebral artery or - alternately - external carotid - internal carotid bypass is a reasonable thought in this young man.

RETURN TO WORK

This claimant feels he can return to work. He has no fixed neurological deficits. It is not - yet - clear to me to what extent aggressive efforts on his part would trigger recurrent and worsened cerebral ischemia (that is why I am suggesting a tilt table EEG study).

STUDIES RECEIVED

Big stack of medical records - four hours.

STUDIES PERFORMED

EEG.

STUDIES REQUESTED

I need an up-to-date cardiological evaluation of this claimant with the latest loop data. It is possible that this claimant's right middle cerebral artery disease is position-dependent and I hope that a copy of this report is provided to Dr. XXXXXX. It is my thinking that a tilt table - EEG study might provide useful information relevant to cerebral ischemia and also relevant to arrhythmia (see discussion below). His basic EEG today was WNL.

On written approval from the carrier, we will schedule these diagnostic tests as possible.

RESEARCH PERFORMED

Please refer to refereed - open source articles I have selected relevant to isolated middle cerebral artery disease, and position dependent cerebral ischemia, and internal carotid - external carotid bypass therapy - therapeutic.

FURTHER COMMENT

The most pertinent abnormality is narrowing of the right middle cerebral artery. To what extent this can be approached endoscopically or by bypass (and whether this should be done) is a pertinent question. The claimant's week and a half of symptoms in [redacted] - [redacted] suggest a waxing and waning course with an area of brain experiencing variable ischemia.

If questions remain or arise that I can answer at this time, kindly write me back and I will respond immediately. Please note that the recent literature on syncope emphasizes tilt table testing.

If questions remain or arise that I can answer at this time - kindly write me back and I will respond immediately. Please note that a crucial issue is whether this claimant can or cannot return to work. I request the privilege of seeing the claimant back again in two - four months, at which time I will provide standard ratable language relevant to this issue, which is going to be about two years old when I see the claimant back again.

I certify that I took the complete history from the claimant, conducted the examination, reviewed all available medical records, and composed and drafted the conclusions of this report. The conclusions and opinions within this report are solely mine. I declare under penalty of perjury that the information contained in this report and its attachments, if any, is true and correct to the best of my knowledge and belief, except as to information that I have indicated I received from others. As to that information, I declare under penalty of perjury that the information accurately describes the information provided to me and, except as noted herein, that I believe it to be true. In accordance with Labor Code Section 5703(a) (2), there has not been a violation of Labor Code Section 139.3, and the contents of the report are true and correct to the best of my knowledge. This statement is made under penalty of perjury.

Pursuant to 8 Cal. Code Regs. Section 49.2-49.9, I have complied with the requirement for face-to-face time with the client in this evaluation. If necessary, I have discussed apportionment in the body of this report. If I have assigned disability caused by factors other than the industrial injury, that level of disability constitutes the apportionment.

The ratio of nonindustrial disability, if any, to all described disability represents my best medical judgment of the percentage of disability caused by the industrial injury and the percentage of disability caused by other factors, as defined in Labor Code Sections 4663 and 4664.

Respectfully,

Michael M. Bronshvag, M.D.
Diplomate in Neurology, ABP&N
Diplomate in Internal Medicine, ABIM
Date of Report: [redacted]
Signed this [redacted]

P.S. Copy to treating neurologist XXXXXX – XXXXXX, XX.