Redacted to protect patient privacy

Case Report

Authored by:

Godoy Medical Forensics

www.GodoyMedical.net

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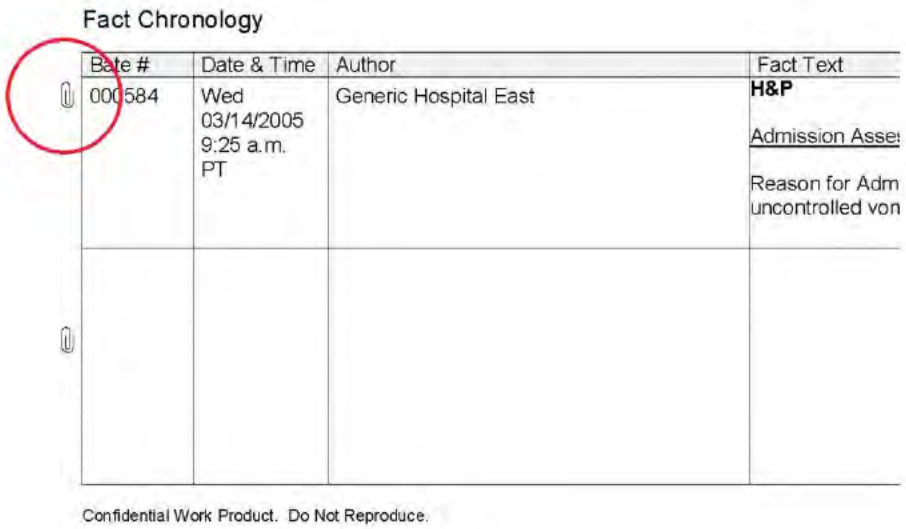
ReportBook Contents

Fact Chronology Instructions:
How to Use this Report

This PDF contains the set of organized records and research pertaining to the case. These files are embedded, and can be accessed through links.

To access the files, click the paperclip icons found in the left hand column of the report:



Fact Chronology

Bate #	Date & Time	Author	Fact Text
000584	Wed 03/14/2005 9:25 a.m. PT	Generic Hospital East	**H&P** Admission Asse Reason for Adm uncontrolled von

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This function allows you to access linked files throughout the report, as well as the complete set of organized records pertaining to the case, found at the bookmark labeled “Organized Records”:

Documents

Bate #	Date	Author(s)	Full Name	Type +	Subtype
000001 - 000002	To Be Determined	Generic Hospital East	ED Record [000001 - 000002]	Medical Record	ED
000003 - 000008	Wed 01/02/2008	Generic Hospital East	Admin [000003 - 000008]	Medical Record	ED
000009 - 000010	Wed 01/02/2008	Generic Hospital East	H&P [000009 - 000010]	Medical Record	ED
000011 - 000012	Wed 01/02/2008	Generic Hospital East	Doctors Orders [000011 - 000012]	Medical Record	ED

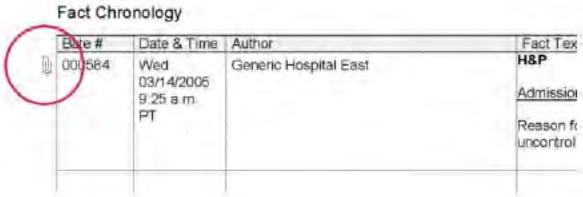
The cited reference material is also embedded and can be accessed through the “Works Cited” section:

Works Cited

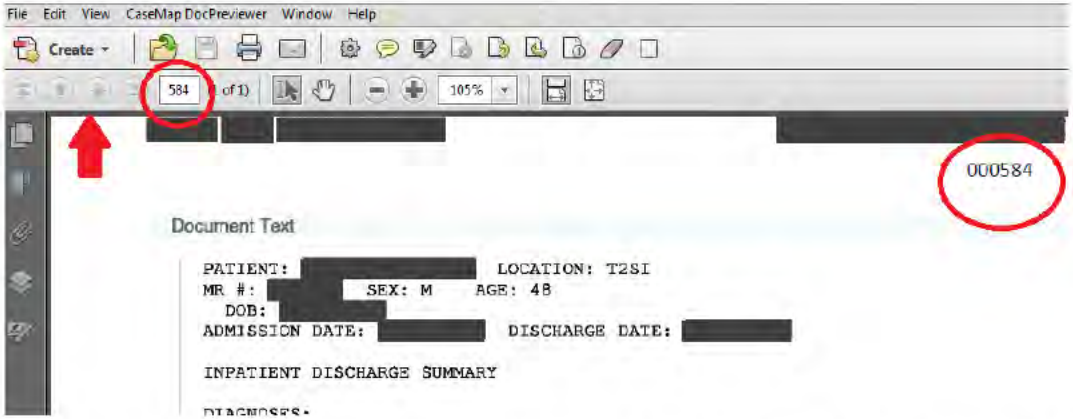
Authority Name	Description	Extract Text	Notes	Linked Issues
Alcohol Health and Research World	Maher, J. (1997). Exploring Alcohol's effects on liver function. Alcohol Health and Research World, 21:1, pp 5-12.	***A large proportion of heavy drinkers develop serious alcoholic liver disease. Susceptibility to alcoholic hepatitis and Cirrhosis appears to be influenced by heredity, gender, diet, and co-occurring liver illness. Most alcoholic liver damage is attributed to Alcohol metabolism. Liver injury may be caused by direct toxicity of metabolic by-products of Alcohol as well as by***	This most advanced form of liver disease is diagnosed in 15 to 30 percent of heavy drinkers.	Liver Disease

Finding fact details in the Fact Chronology:

1. Click the paperclip icon next to the fact you want to view, and the document will open.



2. To find the page you are looking for, use the up and down arrows to navigate to the page number that corresponds to the bate number of the fact. The fact will be on that page.



*To jump directly to the page you are seeking, type the corresponding bate number (without the preceding zeros) into the box next to the arrows.

All names have been changed for privacy.

Instructions for the Expert:

Thank you for working with us on this case. What you will find enclosed is a precise fact chronology transcribed directly from the medical records. A Registered Nurse or Nurse Practitioner has reviewed the records and determined what excerpts have bearing on the case for the purposes of review by a medical expert. There are a few sections, and please use the bookmarks to the left to find them quickly:

Instructions:
(Current Page) Instructions on how to manage the report book and medical record portfolio.

Fact Chronology:
This is all the pertinent records, the fact chronology in full.

Issue Summaries:
If there are issues within the case that warrant their own, smaller fact chronology, you will find them here. The issue summary chronologies consist of facts filtered from the fact chronology that are deemed to hold bearing in that particular issue. **There are no new facts in the issue summaries that are not already present in the full fact chronology.**

Medical Records:
While you are reading the records within the fact chronology, please feel free to reference the organized set of medical records that came with this report. They should be easily navigated, and the *Bate Number* within the fact chronology is a direct reference to the Bate Numbers within the medical records. The medical records are also book marked for quick reference to specific documents or dates within the file.

If you have any questions, please don't hesitate to contact myself or one of the nurses at Godoy Medical Forensics, Inc.

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Fact Chronology

Fact Chronology

Bate #	Date & Time	Author	Fact Text
000703 - 000704	To Be Determined	MD	**Other Postmortem Reports** Dr. - Autopsy Eyes ...Gross: Right eye: The specimen is received fixed in formalin in a container labeled with the patient's name and "OD" and consists of a right eye of moderately firm consistency measuring 18.0 x 19.0 x 18.0 mm with 15.0 mm of attached optic nerve. The cornea is clear and measures 11 .0 *x* 10.0 mm. The round pupil measures 4.0 mm in diameter. A full thickness scleral discontinuity is identified that measures 1.0 mm in diameter and is located 9.0 mm posterior to the corneoscleral limbus and 12.0 mm anterior to the optic nerve at 12 o 'clock (p resumed aspiration site). A horizontal cut is made with a superior cap removed. On sectioning, the anterior chamber angle is open. The iris, ciliary body and crystalline lens are intact. Retinal hemorrhages are identified at the ora serrata, midperiphery, equator and posterior pole. Hemorrhage covers approximately 80% of the retinal surface (gross estimate). Some of the retinal hemorrhages contain white centers. A moderate amount of gray-tan material is present in a subretinal space (presumed subretinal flu id). A chorioretinal defect is identified that corresponds to the aforementioned scleral discontinuity (presumed aspiration artifact). The optic nerve head margins appear blurred. An overlying whitish tissue is present obscuring the view of the optic nerve head . Apparent optic nerve sheath hemorrhage is identified. Representative sections are submitted in two cassettes labeled PO and optic nerve. Left eye: The specimen is received fixed in formalin in a container labeled with the patient's name and consists of a left eye of moderately firm consistency measuring 23.0 x 22.0 x 23.0 mm with 16.0 mm of attached optic nerve. The cornea is clear and measures 11.0 x 10.0 mm. The round pupil measures 4.0 mm in diameter. A full thickness scleral discontinuity is identified that measures 1.0 mm in diameter and is located 9.0 mm posterior to the corneoscleral limbus and 12.0 mm anterior to the optic nerve at 12 o 'clock (presumed aspiration site) . A horizontal cut is made with a superior cap removed. On sectioning , the anterior chamber angle is open . The iris, ciliary body and crystalline lens are intact. Retinal hemorrhages are identified at the ora serrata , midperiphery, equator and posterior pole . Hemorrhage covers approximately 85% of the retinal surface (gross estimate). Some of the re tina l hemorrhages contain white centers . A partial circinateretinal fold is identified centered on the fovea extending temporally from the optic nerve from 12 to 6 o 'clock and measures 7.0 mm in diameter. A chorioretinal defect is identified that corresponds to the aforementioned sclera l discontinuity (p resumed aspiration artifact). The optic nerve head

Fact Chronology

Bate #	Date & Time	Author	Fact Text
**	**	**	margins appear blurred . An overlying whitish tissue is present obscuring the view of the optic nerve head. Apparent optic nerve sheath hemorrhage is identified. Representative sections are submitted in two cassettes labeled PO and optic nerve. Microscopic: Right eye: Examination discloses an intact corneal epithelium. Bowman's layer, the corneal stroma and Descemet's membrane are intact and unremarkable . The endothelium is unremarkable with 30 nuclei per high power field. The anterior chamber angle is open and unremarkable . The iris, ciliary body and crystalline lens are intact and unremarkable. The vitreous is clear and unremarkable. The retina contains hemorrhages identified at the ora serrata, midperiphery, equator and posterior pole. The hemorrhages are present in all retinal layers including the nerve fiber and ganglion cell layers, inner and outer nuclear layers, and inner and outer plexiform layers .The retinal pigment epithelium and choroid are in tact and unremarkable. The sclera is intact and unremarkable. Longitudinal and cross sections of the optic nerve disclose optic nerve sheath hemorrhage. Iron stains of the globe and optic nerve are positive within cells located with in the optic nerve sheath and within the neural retina. Left eye : Examination discloses an intact corneal epithelium. Bowman's layer, the corneal stroma and Descemet's membrane are intact and unremarkable. The endothelium is unremarkable with 31 nuclei per high power field. The anterior chamber angle is open and unremarkable. The iris, ciliary body and crystalline lens are intact and unremarkable. The vitreous is clear and unremarkable. The retina contains hemorrhages identified at the ora serrata, midperiphery, equator and posterior pole. The retinal hemorrhages are located in the inner and outer nuclear layers, and inner and outer plexiform layers and nerve fiber and ganglion cell layers. The retinal pigment epithelium and choroid are intact and unremarkable. The sclera is intact and unremarkable. Longitudinal and cross sections of the optic nerve disclose optic nerve sheath hemorrhage. Iron stains of the globe and optic nerve are positive within the neural retina and optic nerve sheath. Impression: Eyes, autopsy: Medical Examiner's Office. Eye, right: Retina: Retinal hemorrhage. Positive iron stain. Optic nerve: Optic nerve sheath hemorrhage. Positive iron stain.

Fact Chronology

Bate #	Date & Time	Author	Fact Text
**	**	**	Left eye: Retina: Retinal hemorrhage. Positive iron stain. Optic nerve: Optic nerve sheath hemorrhage. Positive iron stain.
000417	Wed 02/21/2007		**Consultations** Consultation - Neurology ...Physical Examination: ...HEENT: Does not open his eyes to stimulation, but withdraws and has a strong cry. When the eyelids are open the gaze is dysconjugate. Pupils are pinpoint. Fundus was not visualized. Extraocular movements were full. No horizontal plane. The face is symmetric. Gag is intact. He moves his extremities in response to local noxious stimuli.
000042	Wed 02/21/2007		**EEG** Electroencephalogram ...Impression: This EEG is within normal limits during predominantly restless wakefulness. The patient is seen to raise his legs and manifest other motor activity without evidence of underlying seizure activity on the EEG. Clinical correlation is needed .
000043	Wed 02/21/2007		**Radiology** CT Brain, without Contrast ...No intracranial hemorrhage noted. There is an area of abnormal hypodensity measuring approximately 2.6 x 1 cm in size in the area of the left external capsule and basal ganglia. There is some mass effect on the left lateral ventricle frontal horn. Lesion is compatible with possible mass versus CVA. There appears to be some edema in the left posterior temporal lobe and parietal region posterior to the lesion as well. No significant shifts or herniation identified. MRI with contrast is recommended for further evaluation of these findings. ...Impression: 1. Abnormal brain CT as described, with mass versus stroke left cerebral hemisphere. MRI with contrast recommended for further evaluation.

Fact Chronology

Bate #	Date & Time	Author	Fact Text
000024	Wed 02/21/2007 12:30 a.m. PT		**Nurse Assessments** LII Evaluation Reason for Evaluation Babe having 1 episode of arching back, rolling eyes in back of head, clinching fists and turning feet inward. No classic symptoms of seizures. No duskiness with this episode. RN reported that babe did have circumoral cyanosis @ shift change and mom reports babe rolls eyes back frequently. RN called physician. Order to keep in nursery and monitor for seizure activity and to try Enfamil A.R. S/S baby is exhibiting Other Annotation: possible seizure activity or reflux with arching of back, straightening legs and clinching fists and rolling eyes back with diaphoresis.
000036	Wed 02/21/2007 7:40 a.m. PT		**Labs** Microbiology Blood Culture-Routine @ Source: Blood Acc/#: 07-052-00907 Pedi Received: 02/21/07 1700 Final Report Culture: Final 02/26/07 1700 No organisms isolated. @ - Bld
000036	Wed 02/21/2007 11:45 a.m. PT		**Labs** Microbiology Smear Report Gram Stain 02/21/07 2348 Cytospin prepared smear results: WBC's seen RBC's seem

Fact Chronology

Bate #	Date & Time	Author	Fact Text
**	**	**	No organisms seen Final Report Culture Final 02/25/07 1122 No Aerobic Growth @ = CSF Cult/GS Performed at
000267 - 000268	Wed 02/21/2007 4:34 p.m. PT		**Radiology** Outside CT Scan of Brain . ...Impression: 1. Infarction In the head of the left caudate nucleus and putamen, consistent with disease in the region of the A1 and M1 segments with additional infarction identified more posteriorly and superficially in the left temporal cortex. 2. Mild mass effect. Findings: ...Axial. scans show no fractures. Evaluation of the soft tissue images demonstrates a focal area of abnormal signal intensity which is lentiform in shape corresponding to the putamen and extending into the head of the left caudate nucleus. This well circumscribed appearance is typical of a basal ganglia infarct in the left M1 territory. There is also mildly decreased density in the temporal lobe consistent with additional infarction in the left middle cerebral artery territory temporal branches. No hemorrhage is identified. There is very mild mass effect with some compression of the left lateral ventricle compared to the right, but none of the ventricles appear dilated. No obvious hemorrhage is evident.
000120	Thu 02/22/2007		**Progress Notes** Neonatal Progress Note ...Infectious Disease: The infant's CBC this morning showed a white count of 17.39, along with a platelet count of 331. His differential showed segment count of 73, along with 6 bands, 16 lymphocytes, and 5 monocytes. This infant has had a blood culture upon his admission at Hospital prior to his transfer here. We will obtain those results as soon as possible. and ampicillin. Our plan for today is to continue monitoring his CBC, and for clinical signs and symptoms of sepsis and we will also consider discontinuing ampicillin and gentamicin with a negative 48-hour blood culture.

Issue Summaries

Issue Summaries

Issue: 1. Intracranial hemorrhage

Facts bearing on Intracranial hemorrhage:

Volume	Bate #	Date & Time	Source(s)	Fact Text
0	000419 - 000420	Sat 02/24/2007	Consultations [%LF%|3] Page 419	**Consultation** Genetic Consultation Reason for Consultation: Ischemic CNS changes with prothrombin gene mutation. ...Laboratory / Radiologic Studies: ...There is mild swelling and also petechial hemorrhage, but no gross hemorrhage. A thrombophilic workup has already been done and some results are already available. His PT, PTT, fibrinogen, and antithrombin Ill levels show a normal PT 15.1, PTT 30.2, fibrinogen 166, and antithrombin Ill 57. CBC has shown a consistently normal platelet count with a platelet count today of 333,000. Homocysteine levels are pending. Anticardiolipin antibodies are pending. His molecular genetic studies show a homozygous normal MTHFR, but a heterozygous abnormal specimen for prothrombin gene mutation. Protein C and protein S levels have been done which are pending. Lupus anticoagulant is pending. ...Assessment: ...To complete the workup, recommendation is to obtain a family history, which was not available in the chart in terms of family history of strokes, deep venous thromboses, pulmonary embolism, etc. as well as draw lipoprotein A level. In terms of therapy, the patient, despite the finding of a heterozygous prothrombin gene study, should not be anticoagulated in view of several things: 1. The risk of recurrence in neonates is very low. 2. The fact that this patient has a non-cardioembolic stroke being the only absolute indication for anticoagulation in neonatal central nervous system ischemia. 3. And the fact that this patient has already evidence of central nervous system bleeding even though not major
0	000447 - 000449	Tue 03/27/2007	Doctor Letter [%LF%|1] Page 447	**Doctor Letter** Dear Dr. ...He was not anticoagulated since his echocardiography did not show the presence of a cardioembolic stroke. He had an EEG early on that showed frequent spikes.

Continued: Facts bearing on Intracranial hemorrhage:

Volume	Bate #	Date & Time	Source(s)	Fact Text
**	**	**	**	cerebral dysfunction as well as epileptiform activity from the left, however, repeat EEG done 6 days later was normal. was eventually discharged from the hospital on March 5, 2007. He was sent home on his anticonvulsants. According to the parents who accompany him, he has done quite well at home. They do not think that he is having any seizures, however sometimes he will shake his tight side and they wonder whether those movements could represent seizures. ...He has not had any intercurrent illnesses. ...Family History: ...The mother has 1 first cousin that was on Coumadin for a couple of years secondary to a clot of the arm. There are no other family members with a history of thrombosis or death or recurrent miscarriages. ...Laboratory Workup: Protein C normal at 35, protein S normal at 41, antithrombin III normal at 57, factor VIII APC resistance 2.4, prothrombin gene mutation heterozygous abnormal, homocysteine 9.2. PT and PTT 15.1 and 30.2 respectively. Fibrinogen 166. Phospholipid antibody panel negative. Cardiolipin screen negative. MTHFR homozygous normal. ...Summary: ...history of perinatal stroke diagnosed a few hours after birth, who was found to have a heterozygous mutation of the prothrombin gene. ...this is the second most common inherited abnormality that will increase the risk for the development of thrombosis. As you know the etiology of strokes in the neonatal period is not always found. A percentage of those patients are going to end up being diagnosed as having cardioembolic strokes. Of those without any cardioembolic problems, a fraction is eventually found to have either an inherited or acquired thrombophilic states. ...This is felt to be a mild prothrombotic state, and usually other risk factors are felt to be needed for thrombosis development. Redacted to pro does not need any treatment at this point since neonatal patients without cardioembolic complications are not felt to be at high risk of stroke redevelopment.
0	000514	Wed 04/11/2007 4:22 p.m. PT	Call Records [%LF%|0] Page 514	**Call Records** Telephone Message

Issue Summaries

Issue: 2. Stroke

Facts bearing on Stroke:

Volume	Bate #	Date & Time	Source(s)	Fact Text
0	000417	Wed 02/21/2007	Consultations [%LF%|1] Page 417	**Consultations** Consultation - Neurology ...Physical Examination: ...HEENT: Does not open his eyes to stimulation, but withdraws and has a strong cry. When the eyelids are open the gaze is dysconjugate. Pupils are pinpoint. Fundus was not visualized. Extraocular movements were full. No horizontal plane. The face is symmetric. Gag is intact. He moves his extremities in response to local noxious stimuli.
0	000042	Wed 02/21/2007	EEG [%LF%|0] Page 42	**EEG** Electroencephalogram ...Impression: This EEG is within normal limits during predominantly restless wakefulness. The patient is seen to raise his legs and manifest other motor activity without evidence of underlying seizure activity on the EEG. Clinical correlation is needed .
0	000043	Wed 02/21/2007	Radiology [%LF%|0] Page 43	**Radiology** CT Brain, without Contrast ...No intracranial hemorrhage noted. There is an area of abnormal hypodensity measuring approximately 2.6 x 1 cm in size in the area of the left external capsule and basal ganglia. There is some mass effect on the left lateral ventricle frontal horn. Lesion is compatible with possible mass versus CVA. There appears to be some edema in the left posterior temporal lobe and parietal region posterior to the lesion as well. No significant shifts or herniation identified. MRI with contrast is recommended for further evaluation of these findings. ...Impression: 1. Abnormal brain CT as described, with mass versus stroke left cerebral hemisphere. MRI with contrast recommended for further evaluation.

Continued: Facts bearing on Stroke:

Volume	Bate #	Date & Time	Source(s)	Fact Text
0	000024	Wed 02/21/2007 12:30 a.m. PT	Nurse Assessments [%LF%|0] Page 24	**Nurse Assessments** LII Evaluation Reason for Evaluation Babe having 1 episode of arching back, rolling eyes in back of head, clinching fists and turning feet inward. No classic symptoms of seizures. No duskiness with this episode. RN reported that babe did have circumoral cyanosis @ shift change and mom reports babe rolls eyes back frequently. RN called physician. Order to keep in nursery and monitor for seizure activity and to try Enfamil A.R. S/S baby is exhibiting Other Annotation: possible seizure activity or reflux with arching of back, straightening legs and clinching fists and rolling eyes back with diaphoresis.
0	000267 - 000268	Wed 02/21/2007 4:34 p.m. PT	Radiology [%LF%|0] Page 267	**Radiology** Outside CT Scan of Brain . ...Impression: 1. Infarction In the head of the left caudate nucleus and putamen, consistent with disease in the region of the A1 and M1 segments with additional infarction identified more posteriorly and superficially in the left temporal cortex. 2. Mild mass effect. Findings: ...Axial. scans show no fractures. Evaluation of the soft tissue images demonstrates a focal area of abnormal signal intensity which is lentiform in shape corresponding to the putamen and extending into the head of the left caudate nucleus. This well circumscribed appearance is typical of a basal ganglia infarct in the left M1 territory. There is also mildly decreased density in the temporal lobe consistent with additional infarction in the left middle cerebral artery territory temporal branches. No hemorrhage is identified. There is very mild mass effect with some compression of the left lateral ventricle compared to the right, but none of the ventricles appear dilated. No obvious hemorrhage is evident.
0	000298 -	Thu	EEG [%LF%|0] Page 298	**EEG**

Organized Records

Organized Records

#	Bate #	Year		Full Name	Type +
01	000001a -000002a	2007	Hospital	Medical Timeline	Other
02	000001 - 000008	2007	Hospital	Birth Records	Inpatient
03	000009 - 000019	2007	Hospital	Admin	Inpatient
04	000020 - 000023	2007	Hospital	H&P	Inpatient
05	000024 - 000027	2007	Hospital	Nurse Assessments	Inpatient
06	000028 - 000033	2007	Hospital	Doctors Orders	Inpatient
07	000034 - 000041	2007	Hospital	Labs	Inpatient
08	000042	2007	Hospital	EEG	Inpatient
09	000043 - 000044	2007	Hospital	Radiology	Inpatient
10	000045 - 000051	2007	Hospital	MARs	Inpatient
11	000052 - 000075	2007	Hospital	Flowsheets	Inpatient
12	000076	2007	Hospital	Respiratory Therapy	Inpatient
13	000077 - 000080	2007	Hospital	Transport	Inpatient
14	000081 - 000110	2007	Hospital	Admin	Inpatient
15	000111 - 000116	2007	Hospital	H&P	Inpatient
16	000117 - 000161	2007	Hospital	Progress Notes	Inpatient
17	000162 - 000210	2007	Hospital	Doctors Orders	Inpatient
18	000211 - 000266	2007	Hospital	Labs	Inpatient
19	000267 - 000285	2007	Hospital	Radiology	Inpatient
20	000286 - 000297	2007	Hospital	ECG	Inpatient
21	000298 - 000308	2007	Hospital	EEG	Inpatient
22	000309 - 000312	2007	Hospital	Audiology	Inpatient
23	000313 - 000329	2007	Hospital	MARs	Inpatient
24	000330 - 000415	2007	Hospital	Flowsheets	Inpatient

Organized Records

#	Bate #	Year		Full Name	Type +
25	000416 - 000445	2007	Hospital	Consultations	Inpatient
26	000446 - 000456	2007	Hospital	Doctor Letter	Inpatient
27	000457 - 000460	2007	Hospital	Rerpiratory Therapy	Inpatient
28	000461 - 000462	2007	Hospital	Anesthesia	Inpatient
29	000463 - 000465	2007	Hospital	Social Worker Notes	Inpatient
30	000466 - 000499	2007	Hospital	Discharge	Inpatient
31	000500 - 000513	2007	Health Center	Clinic Visits	Outpatient
32	000514 - 000517	2007	Health Center	Call Records	Outpatient
33	000518 - 000523	2007		Clinic Visit	Outpatient
34	000524 - 000707	2007 - 2009		Autopsy & Other Reports	Police
35	000708 - 001159	2007 - 2011		Interviews	Police
36	001160 - 001211	2007 - 2011	Co. Sheriff's Office	Other Police Documents	Police
37	001212 - 001569	2008 - 2010		Doctor Depositions	Legal
38	001570 - 001578	2007 - 2009		Other Legal Documents	Legal
39	001579 - 001587	n/a		Misc. Documents	Other

Works Cited

Works Cited

Authority Name	Description	Extract Text
American Heart Association	Ferraresi, P. et al. (1997). T*he Heterozygous 20210 G/A Prothrombin Genotype Is Associated With Early Venous Thrombosis in Inherited Thrombophilias and Is Not Increased in Frequency in Artery Disease. Arteriosclerosis, Thrombosis, and Vascular Biology, 17:2418-2422.* Retrieved from http://atvb.ahajournals.org/cgi/content/full/atvbaha;17/11/2418	Very recently, the A allele of a genetic variation (20210 G/A) in the 3'-untranslated region of the prothrombin mRNA has been found to be associated with an increase in venous thrombosis.20 This variation was also associated with elevated plasma prothrombin levels indicating, as previously observed for other hemostatic genes,21-24 the presence of a genetic component in determining protein levels in plasma...
Annals of Emergency Medicine	Lashutka, M., Chandra, A., Murray, H., Phillips, G., & Hiestand, B. (2004). The Relationship of Intraocular Pressure to Intracranial Pressure. *Annals of Emergency Medicine*, 585-591.	Abnormal intraocular pressure as measured with the handheld tonometer is an excellent indicator of abnormal intracranial pressure in patients with known intracranial pathology.
Circulation: Journal of the American Heart Association	Varga, E.A. & Moll, S. (2004). Prothrombin 20210 Mutation (Factor II Mutation). *Circulation, 110:*e15-e18. doi: 10.1161/01.CIR.0000135582.53444.87	Prothrombin is a protein in the blood that is required for the blood to clot. It is also called factor II. Blood clots are composed of a combination of blood platelets and a meshwork of fibrin. If somebody has too little prothrombin, he or she has a bleeding tendency. If an individual has too much prothrombin, blood clots may form when they shouldn't. What Does It Mean to Have the Prothrombin 20210 Mutation? It was discovered in 1996 that a specific change in the genetic code causes the body to produce too much of the prothrombin protein. Having too much prothrombin makes the blood more likely to clot. People with this condition are said to have a prothrombin mutation, also called the prothrombin variant, prothrombin G20210A, or a factor II mutation.
Gene Reviews	Kujovich, J. (2006). Prothrombin-Related Thrombophilia. *Gene Reviews,* July 25, 2006.	**Cerebral vein thrombosis in children.** Although most thromboses in children occur in the extremities, some evidence suggests that 20210G>A heterozygosity may predispose to central nervous system (CNS) thrombosis. However, the evidence regarding the risk for cerebral vein thrombosis is conflicting. Studies that support an association of 20210G>A heterozygosity with cerebral vein thrombosis in children: - In the largest reported series of 20210G>A heterozygous children, 37% of symptomatic children had a history of arterial or venous CNS thrombosis, accounting for 30% of thromboembolic episodes. Cerebral sinus thrombosis occurred in 13% of

Works Cited

Authority Name	Description	Extract Text
**	**	symptomatic children, all of whom were age two years or older [Young et al 2003]. - Heterozygosity was found in 4%-5% of children with cerebral vein thrombosis compared to 1%-2% of controls, differences that did not achieve statistical significance because of the small number of cases [Bonduel et al 2003, Heller et al 2003]. - Underlying illnesses and/or circumstantial risk factors were present in the the majority of children reported with cerebral vein thrombosis [DeVeber et al 2001, Heller et al 2003, Kenet et al 2004]. The combination of an inherited or acquired thrombophilic disorder (including 20210G>A heterozygosity) and an underlying medical condition conferred a 4-fold increased risk, underscoring the multifactorial etiology of this thrombotic complication [Heller et al 2003]. Studies that do not support an association of 20210G>A heterozygosity with cerebral vein thrombosis in children: - In a small case-control study, the prevalence of 20210G>A heterozygosity was similar in children with cerebral vein thrombosis (2.6%) and a group of control children (3.5%) [Kenet et al 2004]. - Data from a large population-based registry suggest a low prevalence of the 20210G>A allele among children and neonates with cerebral vein thrombosis [DeVeber et al 2001]. - A meta-analysis found a nonsignificant trend toward a 2-fold increased risk for cerebral vein thrombosis in children (pooled OR = 1.95); however, 20210G>A was associated with a significant 2-fold increased risk for the combined outcome of first cerebral vein thrombosis or acute ischemic stroke [Kenet et al 2010]. **Stroke in children.** Arterial ischemic stroke in children usually occurs in the setting of multiple predisposing factors [Barnes & Deveber 2006]. Data on the association of thrombophilia with ischemic stroke are conflicting and mostly limited to case series and case-control studies, many of which lacked statistical power due to small sample size. Stroke accounted for 21% of thrombotic events in a highly selected group of symptomatic children with a 20210G>A allele. Children younger than age two years had a significantly higher rate of arterial thrombosis than older children in whom venous thrombosis was far more common. Stroke accounted for 67% of arterial thrombotic events [Young et al 2003]. An International Pediatric Stroke Study (IPSS) is prospectively evaluating the association between inherited thrombophilia and acute ischemic stroke in neonates and children (see International Paediatric Stroke Study). Studies supporting the association of an *F2* 20210G>A allele with stroke in children: