INCIDENT AND/OR RESPONSE TO RESISTANCE REPORT

Supplemental Pages Attached : ☐

(Please print narrative) PAGE 1 OF 1

On 06-26-09, I was assigned to Second Holding at the Duval County Courthouse. At approximately 1230 hours I observed inmate Merrifield sitting in a wheelchair in Second Holding area. Inmate Merrifield was mumbling and moving his feet back and forth. I questioned the inmate as to if he felt he was able to attend court today. The inmate did not respond in a coherent manner. I immediately contacted the sally port to make arrangement to have the inmate returned to the P.T.D.F for medical examination. The wheelchair van was notified. At approximately 1240 hours Officer Norton arrived at the courthouse sally port to transport the inmate back to the P.T.D.F. Upon attempting to load inmate Merrifield on to the jail van officer Norton noticed the inmate was unresponsive. Rescue was immediately notified and responded to the sally port. Rescue 5 treated and transported the inmate to Shands Hospital for further medical evaluation.

REPORTING EMPLOYEE NAME	REPORTING EMPLOYEE SIGNATURE	ID #	DATE SIGNED
FREDDIE L SANDERS		63431	6/25/09

#58

OFFICE OF THE SHERIFF
CONSOLIDATED CITY OF JACKSONVILLE
DEPARTMENT OF CORRECTIONS

INCIDENT AND/OR RESPONSE TO RESISTANCE SUPPLEMENTAL REPORT

DIVISION: ■ JD ☐ PD ☐ CCD	REPORT DATE: 6-24-2009	WATCH #:II	REPORT #: 14058

On 6-24-2009, I was assigned as the Consolidated Transportation Officer. At approximately 1145 hours Officer Tuten from the court house called the Shands clinic and spoke with Officer Rook, and informed her that the inmates in wheelchairs at the court house were ready to be transported back to PDF. AT 1223 hours, I arrived at the court house to transport the inmates back to PDF. Inmate Merrifeild got down stairs via the elevators in his wheelchair and was escorted into the sally port where my van was located. As inmate Merrifield was being escorted to my van I observed that he was slumped over in his wheelchair and had drool coming from the side of his mouth. Inmate Merrifield appeared to be breathing fine so I attempted to wake him up with a gentle shake. I repeated this three or four times, with no response. I then tried to wake him with a sternum rub and he still did not respond. I immediately instructed Civilian Bailiff Daniels to call Rescue which she did. Inmate Merrifield was transported to Shands Hospital via Rescue #5.

REPORTING EMPLOYEE NAME	REPORTING EMPLOYEE SIGNATURE	ID #	DATE SIGNED
K. E. Norton	K. E. Norton	7421	6-25-2009

P-288A REV. 04/2005

#59

Image Encounter	29 Jun 2009 12:00 AM
Image Encounter	29 Jun 2009 12:00 AM
Nursing Encounter	27 Jun 2009 01:46 AM
Image Encounter	26 Jun 2009 12:00 AM
Nursing Encounter	24 Jun 2009 07:00 AM
HST Encounter	23 Jun 2009 06:45 PM
Chronic Care	23 Jun 2009 01:56 PM
Chronic Care	23 Jun 2009 09:36 AM
Nursing Encounter	23 Jun 2009 04:47 AM
Nursing Encounter	22 Jun 2009 04:57 PM
Nursing Encounter	22 Jun 2009 04:53 AM
HST Encounter	21 Jun 2009 06:58 PM
HST Encounter	21 Jun 2009 04:42 PM
Nursing Encounter	21 Jun 2009 07:31 AM
Nursing Encounter	21 Jun 2009 06:23 AM
HST Encounter	20 Jun 2009 05:13 PM
Provider Encounter	20 Jun 2009 04:37 PM
Nursing Encounter	20 Jun 2009 05:20 AM
HST Encounter	19 Jun 2009 10:50 PM
HST Encounter	19 Jun 2009 09:46 PM
Chronic Care	19 Jun 2009 01:48 PM
Nursing Encounter	19 Jun 2009 04:36 AM
HST Encounter	18 Jun 2009 10:26 PM
HST Encounter	18 Jun 2009 04:07 PM
Provider Encounter	18 Jun 2009 03:49 PM
Nursing Encounter	18 Jun 2009 09:00 AM
Nursing Encounter	18 Jun 2009 05:25 AM
Nursing Encounter	17 Jun 2009 11:22 PM
HST Encounter	17 Jun 2009 10:08 PM
Nursing Encounter	17 Jun 2009 04:57 AM
HST Encounter	16 Jun 2009 11:14 PM
HST Encounter	16 Jun 2009 05:10 PM
Nursing Encounter	16 Jun 2009 04:55 PM
Nursing Encounter	16 Jun 2009 03:00 PM
HST Encounter	16 Jun 2009 08:39 AM
HST Encounter	15 Jun 2009 09:34 PM
Nursing Encounter	15 Jun 2009 05:10 PM
HST Encounter	15 Jun 2009 02:40 PM
Provider Encounter	15 Jun 2009 01:04 PM
HST Encounter	15 Jun 2009 11:04 AM
Nursing Encounter	15 Jun 2009 10:01 AM
Nursing Encounter	15 Jun 2009 05:21 AM
HST Encounter	15 Jun 2009 02:38 AM
Nursing Encounter	15 Jun 2009 02:06 AM

#60

- Lantus 35u - pm
- R-S/S - 3x day / IDDM - X 2000 CP 6/13/09

OOS

BAY STREET HEALTH CARE CENTER
DIABETES MELLITUS FLOW SHEET

ACCU/Bo

Frequency Blood Sugar Levels (Use pencil)
Changed by and date (Use pencil)

DOB	Wt	Ht	BMI
10-30-45			

Date Started:

Date	Time	BS Level	Insulin	Provider
6/11	BB			
	BL			
	BD	284	R3	
6/12	BB	246	R-2	
	BL			
	BD	279	R3	
6/13	BB	237	R-2	
	BL			
	BD	333	20N-R5u lunch	CP
6/14	BB	140	Ø	
	BL			
	BD	312	20N-R5u	CP
6/15	BB	94	L-20	
	BL			
	BD	118	Ø	
6/16	BB	129	L20	
	BL			
	BD	95	Ø	
6/17	BB	Refused to come to MR		
	BL			
	BD	158	Ø	
6/18	BB	215	L20 R2	
	BL			
	BD	221		
6/19	BB	60	Held L20 - call MD	
	BL	163 @ 3:30		
	BD	140	Ø	
6/20/09	BB	160	L20	
	BL			
	BD	114		
6/21/09	BB	128	L20	
	BL			
	BD	190	Ø	
6/22	BB	163	L-20, R-1	
	BL			
	BD	76	Ø, NS	
6/23	BB	80	L-20	
	BL	56	4g/juice tab/6 @ 13:00	
	BD	176	Ø	
6/24	BB	67	Insulin held	
	BL			
	BD	Hosp		

Date	Medication	Freq
	Lantus 20 units AM	
	Sliding scale AM	
	sliding scale 4PM	
6/25	Hosp	D

BB=Before Breakfast BL=Before Lunch BD=Before Dinner. Use R=Regular N=NPH 7/3=70/30 L=Lantus A=Apidra Use full name for other insulins.

Name	Docket #	Location
[illegible]	20090[illegible]	4E03 2W65

HST Encounter	14 Jun 2009 10:03 PM
HST Encounter	14 Jun 2009 09:34 PM
Provider Encounter	14 Jun 2009 09:04 PM
HST Encounter	14 Jun 2009 05:16 PM
Infection Control	14 Jun 2009 02:55 PM
HST Encounter	14 Jun 2009 12:45 PM
Nursing Encounter	14 Jun 2009 04:21 AM
Provider Encounter	14 Jun 2009 04:03 AM
HST Encounter	14 Jun 2009 12:24 AM
HST Encounter	13 Jun 2009 08:48 PM
HST Encounter	13 Jun 2009 03:48 PM
Nursing Encounter	13 Jun 2009 04:08 AM
Provider Encounter	13 Jun 2009 02:57 AM
HST Encounter	12 Jun 2009 10:25 PM
Nursing Encounter	12 Jun 2009 04:57 PM
HST Encounter	12 Jun 2009 10:30 AM
HST Encounter	12 Jun 2009 08:03 AM
Nursing Encounter	12 Jun 2009 07:52 AM
HST Encounter	12 Jun 2009 02:38 AM
HST Encounter	11 Jun 2009 10:03 PM
HST Encounter	11 Jun 2009 05:34 PM
Nursing Encounter	11 Jun 2009 04:48 PM
HST Encounter	11 Jun 2009 02:57 PM
Nursing Encounter	11 Jun 2009 12:55 PM
HST Encounter	11 Jun 2009 10:04 AM
Provider Encounter	11 Jun 2009 07:58 AM
Screening	11 Jun 2009 05:59 AM
Provider Encounter	11 Jun 2009 04:58 AM

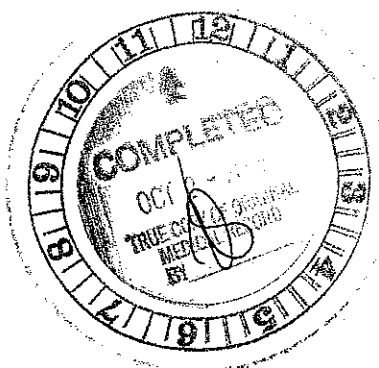

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Image Encounter	29 Jun 2009 12:00 AM
Nursing Encounter	27 Jun 2009 01:46 AM
Image Encounter	26 Jun 2009 12:00 AM
Nursing Encounter	24 Jun 2009 07:00 AM
HST Encounter	23 Jun 2009 06:45 PM
Chronic Care	23 Jun 2009 01:56 PM
Chronic Care	23 Jun 2009 09:36 AM
Nursing Encounter	23 Jun 2009 04:47 AM
Nursing Encounter	22 Jun 2009 04:57 PM
Nursing Encounter	22 Jun 2009 04:53 AM
HST Encounter	21 Jun 2009 06:58 PM
HST Encounter	21 Jun 2009 04:42 PM
Nursing Encounter	21 Jun 2009 07:31 AM
Nursing Encounter	21 Jun 2009 06:23 AM
HST Encounter	20 Jun 2009 05:13 PM
Provider Encounter	20 Jun 2009 04:37 PM
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HST Encounter	19 Jun 2009 10:50 PM
HST Encounter	19 Jun 2009 09:46 PM
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Nursing Encounter	16 Jun 2009 04:55 PM
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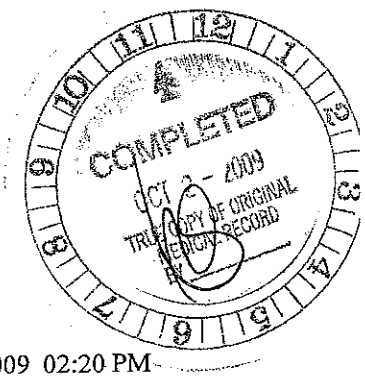
HST Encounter	15 Jun 2009 09:34 PM
Nursing Encounter	15 Jun 2009 05:10 PM
HST Encounter	15 Jun 2009 02:40 PM
Provider Encounter	15 Jun 2009 01:04 PM
HST Encounter	15 Jun 2009 11:04 AM
Nursing Encounter	15 Jun 2009 10:01 AM
Nursing Encounter	15 Jun 2009 05:21 AM
HST Encounter	15 Jun 2009 02:38 AM
Nursing Encounter	15 Jun 2009 02:06 AM



- Lantus 35U - PM
- R-S/S - 3x day / IDDM - x 2000 CP 6/13/09

OOS

BAY STREET HEALTH CARE CENTER
DIABETES MELLITUS FLOW SHEET

Accu-Chek

DOB	Wt	Ht	BMI
12-30-45			

Date Started:

Frequency Blood Sugar Levels (Use pencil)
Changed by and date (Use pencil)

Date	Time	BS Level	Insulin	Provider
6/9	BB			
	BL			
	BD	284	R3	
6/12	BB	216	R-2	
	BL			
	BD	279	R3	
6/13	BB	337	R-2	
	BL			
	BD	333	20N-R5U (typo) CP	
6/14	BB	140	Ø	
	BL			
	BD	312	20N-R5U CP	
6/15	BB	94	L-20	
	BL			
	BD	118	Ø	
6/16	BB	129	L20	
	BL			
	BD	95	Ø	
6/17	BB	Refused to come to MA		
	BL			
	BD	158	Ø	
6/18	BB	215	L20 R2	
	BL			
	BD	221		
6/19	BB	60	Held L20 - call back	
	BL	163 @ 19:30	—	
	BD	160	Ø	
6/20/09	BB	160	L20	
	BL			
	BD	114		CP
6/21/09	BB	128	L20	
	BL			
	BD	190	Ø	CP
6/22	BB	163	L-20, R-1	
	BL			
	BD	76	Ø ins.	BD
6/23	BB	80	L-20	
	BL	56	4g/Inc tab/Ø @ 1310	
	BD	176	Ø	
6/24	BB	67	L insulin held	
	BL			
	BD	Hosp	—	

Date	Medication	Freq
	Lantus 20 units qAM	
	Sliding scale qAM	
	sliding scale qPM	
6/25	Hosp	D

B=Before Breakfast BL=Before Lunch BD=Before Dinner. Use R=Regular N=NPH 7/3=70/30 L=Lantus A=Apidra Use full name for other insulins.

Name

Docket #

Location

4E03
2465

For U.S. Residents Only

LANTUS
insulin glargine [rDNA origin] injection

Important Safety Information for Lantus®

Do not take Lantus® if you are allergic to insulin or any of the inactive ingredients in Lantus®.

You must test your blood sugar levels while using insulin, such as Lantus®. Do not make any changes to your dose or type of insulin without talking to your healthcare provider. Any change of insulin should be made cautiously and only under medical supervision.

Do NOT dilute or mix Lantus® with any other insulin or solution. It will not work as intended and you may lose blood sugar control, which could be serious. Lantus® must only be used if the solution is clear and colorless with no particles visible. **Do not share needles, insulin pens or syringes with others.**

The most common side effect of insulin, including Lantus®, is low blood sugar (hypoglycemia), which may be serious. Some people may experience symptoms such as shaking, sweating, fast heartbeat, and blurred vision. Severe hypoglycemia can be dangerous and can cause harm to your heart or brain. It may cause unconsciousness, seizures, or death. Other possible side effects may include injection site reactions, including changes in fat tissue at the injection site, and allergic reactions, including itching and rash. In rare cases, some allergic reactions may be life threatening.

Tell your doctor about other medicines and supplements you are taking because they can change the way insulin works. Before starting Lantus®, tell your doctor about all your medical conditions including if you have liver or kidney problems, are pregnant or planning to become pregnant, or are breast-feeding or planning to breast-feed.

Indications and Usage

Prescription Lantus® is a long-acting insulin used to treat adults with type 2 diabetes and adults and children (6 years and older) with type 1 diabetes for the control of high blood sugar. It should be taken once a day at the same time each day to lower blood glucose.

Do not use Lantus® to treat diabetic ketoacidosis.

Lantus® SoloSTAR® is a disposable prefilled insulin pen.

Click here for additional important information for Lantus®.

Click here for information on Sharps Medical Waste Disposal.

Click here for information on drug anti-counterfeiting.

The health information contained herein is provided for general educational purposes only. Your healthcare professional is the single best source of information regarding your health. Please consult your healthcare professional if you have any questions about your health or treatment.

#64

LANTUS®

Prescribing Information

(insulin glargine [rDNA origin] injection)

Rx only

HIGHLIGHTS OF PRESCRIBING INFORMATION

These highlights do not include all the information needed to use LANTUS safely and effectively. See full prescribing information for LANTUS.

LANTUS (insulin glargine [rDNA origin] injection) solution for subcutaneous injection
Initial U.S. Approval: 2000

INDICATIONS AND USAGE

LANTUS is a long- acting human insulin analog indicated to improve glycemic control in adults and children with type 1 diabetes mellitus and in adults with type 2 diabetes mellitus. (1)
Important Limitations of Use:

- Not recommended for treating diabetic ketoacidosis. Use intravenous, short-acting insulin instead.

DOSAGE AND ADMINISTRATION

- The starting dose should be individualized based on the type of diabetes and whether the patient is insulin-naïve (2.1, 2.2, 2.3)
- Administer subcutaneously once daily at any time of day, but at the same time every day. (2.1)
- Rotate injection sites within an injection area (abdomen, thigh, or deltoid) to reduce the risk of lipodystrophy. (2.1)
- Converting from other insulin therapies may require adjustment of timing and dose of LANTUS. Closely monitor glucoses especially upon converting to LANTUS and during the initial weeks thereafter. (2.3)

DOSAGE FORMS AND STRENGTHS

Solution for injection 100 units/mL (U-100) in

- 10 mL vials
- 3 mL cartridge system for use in OptiClik (Insulin Delivery Device)
- 3 mL SoloStar disposable insulin device (3)

CONTRAINDICATIONS

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*Sections or subsections omitted from the full prescribing information are not listed

BACK TO TOP

FULL PRESCRIBING INFORMATION

1. INDICATIONS AND USAGE

LANTUS is indicated to improve glycemic control in adults and children with type 1 diabetes mellitus and in adults with type 2 diabetes mellitus.

Important Limitations of Use:

- LANTUS is not recommended for the treatment of diabetic ketoacidosis. Intravenous short-acting insulin is the preferred treatment for this condition.

BACK TO TOP

2. DOSAGE AND ADMINISTRATION

2.1 Dosing

LANTUS is a recombinant human insulin analog for once daily subcutaneous administration with potency that is approximately the same as the potency of human insulin. LANTUS exhibits a relatively constant glucose-lowering profile over 24 hours that permits once-daily dosing.

LANTUS may be administered at any time during the day. LANTUS should be administered subcutaneously once a day at the same time every day. The dose of LANTUS must be individualized based on clinical response. Blood glucose monitoring is essential in all patients receiving insulin therapy.

Patients adjusting the amount or timing of dosing with LANTUS, should only do so under medical supervision with appropriate glucose monitoring *[see Warnings and Precautions (5.1).]*

In patients with type 1 diabetes, LANTUS must be used in regimens with short-acting insulin.

The intended duration of activity of LANTUS is dependent on injection into

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left posterior calf; skin irritation, erythema, and very early ulceration at the presacral, perianal, and posterior scrotal areas; postmortem pressure impressions at the mid right back and outer right ankle; and early decubitus ulceration at the medial right heel.

INJURIES

None significant detected or suspected externally. See above for medical and other minor "wounds."

ROUTINE INTERNAL EXAMINATION

Not performed—case investigation limited to circumstantial and medical history review with body external inspection and toxicology specimen collection. Ocular fluid is clear but present only in small volume. Large-bore needle-aspirations at the suprapubic area return no urine. Only a small volume of thick, dark, clotted blood is able to be collected from the subclavian and femoral areas. No antemortem blood or urine samples accompany the body.

TOXICOLOGY

1) Urine: None available for collection
2) Blood and Ocular Fluid: Collected, not tested

CIRCUMSTANCES of DEATH

Past medical history of insulin-dependent diabetes, hypertension, congestive heart failure, Crohn's disease, possible past myocardial infarction, substance abuse (Benzodiazepines), narcotic use (Lortab), and self-reported "peripheral neuropathy" and "nerves." Arrested and booked with Duval County Jail 6/11/09 for driving-related offenses. Admitted into Pre-Trial Detention Facility 6/11/09 with poor hygiene, urinary incontinence, demands for narcotics, and use of cane and wheelchair, with healing right flank herpes zoster lesions, blood pressure of 138/98, and fingerstick blood glucoses of 173-284 mg/dL. He had with him medications of Prednisone, Carvediol, Xanax, and Lantus insulin with insulin syringes.

On 6/15/09 "too sick to walk" and with nausea, vomiting, and hand tremors, and treated for "withdrawal" with Phenergan and Librium protocol, with Librium refused 6/15/09 but given variably (some doses refused, others not given) 6/16 through 6/20/09. Fingerstick glucose levels of 200's to low 300's until 6/15/09, then dropping from 90's to low to mid 100's through 6/17/09.

Complained of feeling cold all day, dizzy, and "really bad" on 6/18/09, with unstable blood glucose noted (fingerstick of 52 mg/dL), and treated with glucose tablets. Complained 6/20/09 of attack of Crohn's disease, and fingerstick glucose levels were consistently less than 80 from 6/23 through 6/24/09.

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#69

Taken to Duval County Courthouse 6/24/09 for arraignment, but was noted there to have altered mental status (mumbling, incoherent, moving feet back and forth in wheelchair, and then noted to be unresponsive, slumped over in wheelchair, and drooling). EMS called at 1231 hrs. and he was assessed

and found conscious but with respiratory distress, blood pressure 74/46, and blood sugar 92. Treated with fluids, oxygen, Narcan, and glucose, and transported at 1254 hrs. to local hospital E.R. for evaluation and treatment, arriving at 1300 hrs.

Noted in E.R. to be unresponsive with agonal respirations, atrial fibrillation, decerebrate posturing, body temperature of 94 degrees, blood glucose of 55 at 1320 hrs., cerebral atrophy with dilation of lateral ventricles on head CT scan, drugs of abuse urine screen with presence of Benzodiazepines (not confirmed, and no blood level performed), and no response to Narcan. Diagnoses given of acute respiratory failure, hypotension, hypothermia, and abnormal EKG.

Admitted and treated, but suffered complications of metabolic encephalopathy felt due to prolonged hypoglycemia and left lung collapse/dysfunction felt due to mucus plug, also anemia and dysphagia. Released from police custody ("On Own Recognizance") on 6/25/09 at 1625 hrs, with court date set of 7/6/09 at 0900 hrs. Variable blood glucose levels on 6/26/09 from less than 50 to 237. Made Do Not Intubate/Do Not Resuscitate by wife on 6/26/09, and a hospice admission was planned. Continued to do poorly in hospital and suffered blood oxygen desaturations, and comfort measures with Morphine IV drip were begun. He suffered cardiorespiratory arrest and was pronounced dead on 7/5/09 at 0135 hrs.

FINDINGS

1) No trauma
2) No evidence of Benzodiazepines overdose
3) Past medical history and current illness and treatment history as above, especially diabetes with complications

CONCLUSIONS

Cause of Death: Metabolic Encephalopathy, Pneumonia, and Respiratory Failure,
due to Prolonged Hypoglycemia,
due to Complications of Diabetes.

Contributory: Hypertensive Cardiovascular Disease.

Manner of Death: Natural.

COMMENT:

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#69

This case was not reported to the Medical Examiner's Office until 7/13/09, when a request for cremation authorization from the funeral home was received via a faxed completed death certificate. That certificate gave the cause of death as "Encephalopathy from prolonged hypoglycemia," "Pneumonia,"

and "Respiratory insufficiency" with a "Natural" manner of death, with "no autopsy" performed. The certificate was reviewed and questioned by the M.E. on duty, and some of the above information was learned. Additional information and clarification was needed, however, and requested by this M.E. on 7/13/09. Information was received on 7/16/09 and reviewed by two other M.E.'s and, later, by this M.E., as it appeared the case was probably falling to me per the other M.E.'s., if jurisdiction were to be taken.

The decedent had been released from police custody well before the death, and thus the case did not have M.E. jurisdiction or require an autopsy from an "in-custody" standpoint, but the admission urine drug screen and the decedent's wife's statements to doctors while he was still alive raised a concern of Benzodiazepines as the possible cause of his collapse at the courthouse and his complications and death. Jail records concerning his condition, course, and medications (especially) were requested immediately but not received in a timely-enough fashion to allow for review and a non-jurisdiction decision (if the information did not support an overdose) to keep the family and funeral home from waiting overly-long for the cremation authorization.

Thus, after further consultation with the other M.E.'s, it was decided to take jurisdiction on the case due to the possible overdose issue, but since significant time had passed since between the admission and the death and since no admission blood or urine samples were still available with which to verify and identify the Benzodiazepine(s) present and to obtain admission blood level(s) of the drugs, no autopsy was deemed necessary, and a body inspection with records review to attempt to answer the question was done on 7/17/09, and additional jail and other records were requested and subsequently received and reviewed by me, and a "Pending" amended death certificate and cremation authorization were done on 7/22/09. Finally, additional records and clarification of events were sought and received, and the above-listed findings and conclusions were reached.

10/22/09

Jesse C. Giles, MD
Florida M.E. Districts III and IV A.M.E.

#67
#68
#69

JCG

Patient Name: MERRIFIELD,WILLIAM M
Med Rec #: 13895767
Location: 7N 705-02
Physician: LAOS, LUIS F
Acct #: 0817500942

********** TOXICOLOGY AND TDM **********

DATE:	07/04/09	06/30/09	06/29/09	06/28/09	06/27/09	Ref Range	Units
TIME:	*0600	*0530	*0500	*0445	0200		
Amikacin Random	27.3	5.0	8.9	16.1	29.5		ug/mL
Vancomycin Random	19.5	17.3	21.9	29.6			ug/mL

---------- TOXICOLOGY AND TDM ----------

DATE:	06/25/09	06/24/09	Ref Range	Units
TIME:	0335	1320		
Amikacin Random	LOS			ug/mL
Vancomycin Random	Less than 1			ug/mL
Amphetamine		NEGAMP *	NEG	
Barbituate		DOA200 *	NEG	
Benzodiazepine		POSDOA *	NEG	
Cocaine		DOA300 *	NEG	
Opiates		DOA300 *	NEG	
Cannabinoids		DOA100 *	NEG	

---FOOTNOTES---
DOA100 Not detected. (Detection limit 100 ng/mL)
DOA200 Not detected. (Detection limit 200 ng/mL)
DOA300 Not detected. (Detection limit 300 ng/mL)
LOS Less than 0.5
NEGAMP Not detected. (Detection limit 1000 ng/mL)
POSDOA Presumptive Positive. Positive results are unconfirmed and should be used for medical evaluation only.

Shahla Masood, MD

INPATIENT MEDICAL RECORDS COPY
CONTINUED

MERRIFIELD,WILLIAM M
7N 705-02
M-10/30/1945
LAOS, LUIS F

INPATIENT MEDICAL RECORDS COPY

#68

OFFICE OF THE SHERIFF
CITY OF JACKSONVILLE
DEPARTMENT OF CORRECTIONS

RELEASE ON OWN RECOGNIZANCE CONDITIONS

MERRIFIELD, WILLIAM M DOCKET NUMBER: **2009024776**

YOU HAVE BEEN APPROVED FOR "RELEASE ON OWN RECOGNIZANCE" (ROR) ON THE CONDITION THAT YOU WILL RETURN TO COURT ON THE DATE AND TIME INDICATED BELOW

APPROVING JUDGE DRAKE

REPORT DATE: 7/6/2009 AT 09:00

REPORT TO: COURT ROOM, 23A
DUVAL COUNTY COURTHOUSE - CIRCUIT COURT
330 EAST BAY STREET, JACKSONVILLE, FL 32202

IF YOU FAIL TO REPORT AS INDICATED ABOVE,
A CAPIAS WILL BE ISSUED FOR YOUR ARREST

I HAVE READ, I UNDERSTAND, AND I WILL COMPLY WITH THE ABOVE

unable to sign / hospital / 7/4/09
DEFENDANT'S SIGNATURE

OFFICER'S NAME AND I.D. NUMBER: Off. J. Price 6686

DATE AND TIME: 6/25/09 1600

#69 1

MISDEMEANOR

First Appearance: 6-11	Continued: 6-24 2pm 23	Continued: 7-6 9/23	Continued:	Continued:
Continued:	Continued:	Continued:	Continued:	Continued:

Arrest And Booking Report
Jacksonville Sheriff's Office
Jacksonville Florida

ADULT

Yr: 2009 Inc # FHPG09020122 Amend #
Jail # 2009024776 6/11/2009 02:06 File Direct: YES
JSO ID # Court: County
SSN OBTS #
Day/Date/Time Arrested: Thursday 6/11/2009 00:12

Arresting Agency: FHP

PAULINE DRAKE

Name: MERRIFIELD, WILLIAM M
Aliases:
Nickname(s):
Sub-Sector: OJ
Arrestee's Home Address: 2306 SOUTHSIDE Apt./Lot #: 6F
City: BEAUFORT State: SOUTH CAROLINA Zip: 29902
OC deployed prior to/during Arrest: NO
Taz: Crossstreet:
Race: WHITE Sex: Male DOB: 10/30/1945 Age: 63 Eye Color: GRAY
Hair Color: GRAY/SALT & PEPPER Complexion: PALE Height: 5' 8" Weight: 180 Build: Thin
Miranda Rights Given? YES Suspect Invoke? NO Arrestee needs ADA Consideration? NO
Disability or Special Consideration:
Accommodations Requested:
Driver's License # 011355217 State: SOUTH CAROLINA Subject's Resident Type: OUT OF STATE
Arrest Made On: OV
Hm Phone # Bus. Phone # Phone Ext.
Subject's Residence Status: NON-RESIDENT Armed With:
Distinguishing Marks:
US Citizen: YES
Employer: UNKNOWN Place of Birth: UNKNOWN UNKNOWN UNKNOWN
School Last Attended: UNKNOWN
Domestic Violence Involved: NO Children under 18 Present: If No is it Domestic Related: NO
Day/Date/Time of Incident-From: Wednesday 6/10/2009 22:49 Day/Date/Time of Incident-To: Wednesday 6/10/2009 22:49
Sub-Sector:
Incident Address: I 95 EXPY S Apt./Lot #:
City: State: FLORIDA Zip:
Taz: Crossstreet:
Sub-Sector:
Offense Location Type: Interviewed by:
Where Arrested: I 95 EXPY S Apt./Lot #:
City: State: FLORIDA Zip:
Taz: Crossstreet:
Involved in Traffic Accident: YES Injuries from Accident:
Is Incident Gang Related: NO
Is Arrestee a Gang member? NO

Statute or Ordinance Number(s): #1
Statute No: 322.34(2) Degree: CM UCR Code: 540A Attempt Code: Commit
LICENSE; KNOWINGLY OPER VEH W/ DL SUSP, CANCELLED, REVOKED
6/11/2009 - 02:02
Warrant Type: Not Applicable
Citation # SA#
Capias/Warrant # Case # 2009-CT- 13621
No. of Counts:
ROR Pretrial Bond Type: Bond
CT. Location/Div.:
JUDGE Drake
Judge
WAIVE CLEARANCE
YES ☑ NO ☐
OFFICER: K. Cater 5906
DATE: 6-25-09
ORIGINAL
Jurisdiction:
Bond Amount: $1503
Date of Issue:
Date of Return:
Disposition Date:
Disposition: ROR
Blanket Bond:
VOP/FTA ONLY

PER JUDGE DRAKE 06-25-09

Patient:MERRIFIELD,WILLIAM M
DOB: 10/30/45
Sex: MALE

Fin Class:CITY CONTRACT
Ins. Plan:
Guarantor:SELF

Address:2306 SOUTHSIDE BLVD
BEAUFORT
SC,29902
Phone #:(843)524-1714

Adm Date:06/24/09
Adm Time:18:00
Dis Date:07/05/09
Dis Time:02:58
LOS: 11

Adm Source:EMERGENCY ROOM
Adm Type: EMERGENCY
Trans From:
Service: INTERNAL MED C
Dis Status:X-EXPIRED

Acct. #: 0917500942
Unit. #: 13895767
Pt. Type:INPATIENT
Trans To:
Coder: JAA

Admitting DR:LAOS,LUIS F
Attending DR:ALEXANDRAKI,IRENE
ER Physician:

Referring DR:SELF,REFERRAL
Discharge DR:ALEXANDRAKI,IRENE
Primary DR: NO,PCP

DRG: 871 SEPTICEMIA OR SEVERE SEPSIS W/O MV 96+ HOURS W MCC

Admit Diagnosis: 518.81 ACUTE RESPIRATORY FAILURE POA: Y
Principal Diagnosis: 038.9 SEPTICEMIA NOS POA: Y

Secondary Diagnoses/POA:

518.81 ACUTE RESPIRATORY FAILURE/Y
410.71 SUBENDO INFARCT, INITIAL/N
584.9 ACUTE RENAL FAILURE NOS/Y
348.31 METABOLIC ENCEPHALOPATHY/Y
486 PNEUMONIA, ORGANISM NOS/Y
518.0 PULMONARY COLLAPSE/Y
578.1 MELENA/Y
555.9 REGIONAL ENTERITIS NOS/Y
276.0 HYPEROSMOLALITY/Y
276.3 ALKALOSIS/Y
263.9 PROTEIN-CAL MALNUTR NOS/Y
286.9 COAGULAT DEFECT NEC/NOS/Y
428.30 DIASTOLC HRT FAILURE NOS/Y
785.52 SEPTIC SHOCK/Y
934.8 FB TRACH/BRONCH/LUNG NE/Y
995.92 SEVERE SEPSIS/Y
250.80 DMII OTH NT ST UNCNTRLD/Y
790.5 ABN SERUM ENZY LEVEL NE/Y
275.42 HYPERCALCEMIA/Y
275.3 DIS PHOSPHORUS METABOL/Y
403.90 HY KID NOS W CR KID I-I/Y
585.9 CHRONIC KIDNEY DIS NOS/Y
285.9 ANEMIA NOS/Y
428.0 CHF NOS/Y
305.40 SED,HYP,ANXIOLYTC AB-NO/Y
305.90 DRUG ABUSE NEC-UNSPEC/Y
E915 FB ENTERING OTH ORIFICE/Y

Reason for Visit:

Principal Procedure:	Date:	Primary Surgeon:
96.71 CONT INV MEC VEN <96 HRS	06/24/09	GALLANTER,TISHA
Secondary Procedures:		
96.04 INSERT ENDOTRACHEAL TUBE	06/24/09	GALLANTER,TISHA
38.93 VENOUS CATH NEC	06/25/09	LAOS,LUIS F

HCPCS:	Modifiers:	Date:	Surgeon:	Pre APC:

Consultants:	Date:	Specialty:
HOPKINS,CHRISTOPHER Y	06/24/09	EMERGENCY MEDICINE
MICU,1		INTERNAL MEDICINE
CARD,CONSULT		CARDIOLOGY
SULLIVAN,ROBERT M	06/25/09	INTERNAL MEDICINE
MED,C		INTERNAL MEDICINE
RAYAPUDI,NAGARJUN		GENERAL SURGERY
SHOOT,CHRISTINA	07/02/09	# NSCG
LAMBERT,EDDIE L	07/03/09	GENERAL SURGERY

End of Report

Shands Jacksonville
ED Treatment Record-ICU
855 West 8th St
Jacksonville, FL 32209
904-244-2240

6/24/2009 13:18:20

MERRIFIELD, WILLIAM

T Swanson, MD

MERRIFIELD,WILLIAM M
Unit # 13895767 RR EDA1
ACCT # 0917500942 ADM:06/24/09
DOB:10/30/45 63Y M -

Critical Procedure Addendum

BP: / P: R: T: °F °C Weight: lbs kgs %02:

ENDOTRACHEAL INTUBATION:

INDICATION:
- ☑ Respiratory Failure / Arrest
- ☑ Airway Compromise / Obtundation
- ☐ Other: ETT tube placed by MD

PRE-OXYGENATION:
- ☑ BVM ☐ Nasal Canula
- ☐ NRB Mask

METHOD:
- ☑ Oral ☐ Nasal
- ☐ Cricothyotomy
- ☐ Other:

VISUALIZATION:
- ☐ Direct
- ☑ Laryngoscope
- ☐ Blind

RAP SEQ IND? ☐ N

Meds Used: Succinylcholine Etomidate Fentanyl Rocuronium

# of Attempts: 1 Tube Size: 8.0

PLACEMENT CONFIRMED BY:
- ☑ Equal BS
- ☑ + Capnometer Change
- ☑ + Stomach Auscultation
- ☐ CXR:

POST INTUBATION STATUS:
- ☐ Alert ☑ Sedated ☑ ↓ LOC
- ☑ On Respirator
- ☑ Tolerated Procedure Well

[signature] 1453 6/24/09 13:20
ED Clinician # Date Time

CENTRAL LINE PLACEMENT / CUTDOWN:

INDICATION:
- ☐ Venous Access
- ☐ Shock / Hypotension
- ☐ Other:

PREP / ANESTH:
- ☐ Betadyne
- ☐ Local: _____
- ☐ Other

DEVICE:
- ☐ Triple Lumen
- ☐ Catheter
- ☐ Intraosseous
- ☐ Other:

SITE: Catheter \ Needle Size: _____
- ☐ Subclavian (R L)
- ☐ Internal Jugular (R L)
- ☐ Upper Extremity (R L)
- ☐ Lower Extremity (R L)
- ☐ Femur \ Tibia (R L)
- ☐ Other:

PROCEDURE DESCRIPTION:

- ☐ CXR:
- ☐ Appropriate Blood Flow

_____ _____ / / :
ED Clinician # Date Time

CHEST TUBE / NEEDLE THORACOSTOMY:

INDICATION:
- ☐ Pneumothorax
- ☐ Hemothorax
- ☐ Other:

PREP / ANESTH:
- ☐ Betadyne
- ☐ Local: _____
- ☐ Other

DEVICE:
- ☐ Chest Tube (# Fr)
- ☐ Cook Catheter
- ☐ Needle (Size:)
- ☐ Other:

SITE:
- ☐ Anterior (R L) ☐ Posterior (R L) ☐ Lateral / Axillary (R L)

Intercostal Space: _____

PROCEDURE DESCRIPTION:

PLACEMENT CONFIRMED BY:
- ☐ CXR: ☐ Auscultation ☐ Appropriate Function

_____ _____ / / :
ED Clinician # Date Time

PERICARDIOCENTESIS:

INDICATION:
- ☐ Tamponade
- ☐ Effusion
- ☐ Other:

PREP / ANESTH:
- ☐ Betadyne
- ☐ Local: _____
- ☐ Other

DEVICE:
- ☐ Needle (Size:)
- ☐ Other:

GUIDED BY: ☐ Anatomical Landmarks ☐ EKG ☐ Other

PROCEDURE DESCRIPTION:

POST-PROCEDURE EVALUATON:
- ☐ CXR:
- ☐ Examination:

_____ _____ / / :
ED Clinician # Date Time

CARDIOVERSION:

INDICATION:
- ☐ Atrial Fibrillation
- ☐ Atrial Flutter
- ☐ Wide-Complex Tachycardia
- ☐ Ventricular Tachycardia
- ☐ Hemodynamic Instability
- ☐ Other:

CONTRAINDICATIONS/ CAUTIONS:
- ☐ No Sick Sinus Syndrome
- ☐ Not Digitalis Toxic

SEDATION:
- ☐ none
- ☐ Versed _____ mg
- ☐ Other: _____

NEW RHYTHM / EKG:

PROCEDURE:
- ☐ #1 Synchronized _____ Joules
- ☐ #2 Synchronized _____ Joules
- ☐ #3 Synchronized _____ Joules
- ☐

_____ _____ / / :
ED Clinician # Date Time

ADDITIONAL NOTES:

Time Out: ☐ Taken → to Verify
- ☐ Patient ☐ Procedure
- ☐ Site ☐ Position

Estimated Blood Loss (EBL) ☐ None ☐ _____ cc

- ☐ Patient Unable to Consent Due to Medical Condition
- ☐ Consent Given, After Risks (including death) and Benefits Discussed

F was present for entire procedure.

_____ _____ / / :
ED Clinician # Date Time

_____ / / : ☐ Key Portion Supervised by Attending [signature] 6/24/09 13:20
PA \ NP Signature Date Time Attending Signature Date Time

Shands Jacksonville
ED Treatment Record-ICU
655 West 8th St
Jacksonville, FL 32209
904-244-2240

6/24/2009 14:07:46

MERRIFIELD.

C Hopkins, MD

MERRIFIELD,WILLIAM M
Unit # 13895767 ER EDA1
ACCT # 0917500942 ADM:06/24/09
DOB:10/30/45 63Y M -

Critical Care Addendum

Critical Care Addendum © 1984 - 2008 RTQA

BP: / P: R: T: °F °C Weight: lbs kgs %O2:

CRITICAL CARE RECORD

Critical Care needed for: ☑ Treatment ☐ Prevention } of ☑ Clinically Significant: ☐ Life Threatening:

- ☐ Dysrhythmia
- ☐ Circulatory Failure
- ☑ Cardiovascular Instability
- ☐ Shock \ Hypotension
- ☐ Hemmorrhage
- ☐ Airway Dysfunction
- ☐ Angioedema
- ☐ Respiratory Insufficiency
- ☐ Respiratory Failure
- ☐ Other:
- ☑ CNS Failure
- ☐ CNS Catastrophe
- ☐ CNS Infection
- ☐ Toxic Exposure
- ☐ Hepatic Failure
- ☐ Metabolic Instability
- ☐ Renal Failure
- ☐ Anaphylaxis
- ☐ Precipitous Delivery
- ☐ Traumatic Injury
- ☐ CNS Injury
- ☐ Vascular Injury
- ☐ Chest Injury
- ☐ Abdominal Injury
- ☐ Surgical Emergency
- ☐ Psychiatric Instability
- ☐ Overwhelming Infection

Time CC Initiated: ☑ On Arrival ☐ Other: ____:____

Critical Diagnoses: Respiratory Failure
Hypothermia
Hypoxia

Physical Exam: ☑ See Primary Chart

Lab \ X-Ray Data: ☑ See Primary Chart

TIME:	REPEAT CRITICAL CARE ASSESSMENTS:	INTERVENTIONS \ PROCEDURES:	RESPONSE:
1310 - 1330	Initial Resuscitation & intubation (5 min) CLS min		
1446	Neuro consulted for EEG	Neuro exam WN	Pupils = A
	Pt 2 pinpoint pupils		Apnea A
	LP for Amis. Emergent		

DISPOSITION:

FINAL DIAGNOSES:
1. Hypothermia
2. Hypoxia
   acute respiratory failure
3.

ADMITTED TO:

BED:_____ AT: ____:____

PHYSICIAN:_____

STATUS AT DISPOSITON:
☐ Good ☐ Fair
☐ Guarded ☐ Poor
☐ Critical ☐ Deceased

CRITICAL CARE TIME:

Total CC Time (CC) _____ min of ☐ Periodic (Interrupted) Care ☐ Continuous (Uninterrupted) Care

Includes Time:
- ☐ at bedside
- ☐ reviewing test results
- ☐ discussing case with staff
- ☐ documenting medical record
- ☐ in cognitive consideration of case
- ☐ discussing decisions with family*
- ☐ Other:

Procedure Time (PT) 5 min

Final CC Time (CC – PT) _____ min ☐ < 30 min ☐ 30 – 74 min ☐ 75 – 104 min ☐ 105 – 134 min

☐ * patient unable \ incompetent (* family discussion necessary for immediate decision

Teaching Case? N Y
Teaching Attending Present?** na N Y
Active Participant?** na N Y
**For all CC times above:

_____ NP \ PA \ Resident Signature ____1 1____ Date _____:_____ Time

T. Gallanti MD Attending Signature 6/24/09 Date 14:15 Time

Name William Merrifield Age 63 Sex M Date of request 6/25/09 Time of Request 9:30
Rm. 7036 Consulted Service CARDS Urgent ___ Routine ___
Unit No ___ Date of Surgery ___
Preop. Surgery ___ Service
Requested by MICU (Attending/Resident) M.D. of the ___ CC / Reason for consult "ACS / Cardiogenic Shock"

IMPRESSION:
1. (+) Troponin in setting of Cr 2.2-2.5 + critical illness w/ a rise + fall
2. ~~Pt Non-diagnostic troponin elevation~~
3. ~~Pt is not in cardiogenic shock~~
4. LV dysfunction moderate
5. Renal Insufficiency (ARF vs acute on chronic, no baseline available)

RECOMMENDATIONS:
1. Medical therapy
2. ✓ lipid profile, liver profile + start statin
3. Start Coreg 3.125 mg po BID when BP tolerates
4. Contrast echo to better assess LV function
5.

Hx: (include hx elements, severity, quality, location, duration, context, timing, associated symptoms)

Hx obtained from chart

63 yo w/ h/o DM, HTN admitted 6/24 w/ respiratory distress / agonal breathing (brought in from jail). Pt w/ critically low blood sugar + Temp 93.4 upon arrival to ER. Pt w/ persistent hypoglycemia, requiring multiple rounds of D50. Pt has not been on any sedation. Pt is non-responsive to verbal stimuli (~~but tactile stimuli~~ + pain). Pt w/ melena this AM. Pt hypotensive ⇒ 5 L IVF since admission [illegible]

SOCIAL Hx: Unknown **FH:** Unknown **PMH:** IDDM, HTN **ALLERGIES:** Unknown

marital status / occupation / tobacco / alcohol / drugs
Mother / Father / Brother / Sister / Other

Surgical Hx: Unknown

MEDICATIONS: outpatient: Lantus.

ROS (describe or indicate negative (-)) Intubated
☐ All other systems negative

Constitution, Musculoskeletal, Hematologic, Neuro, Allergic/Immune, Eye, GI, Skin, Psychiatric, Respiratory, ENT, GU, Gyn, Endocrine, CV

MEDICATIONS: inpatient
Levophed 2mcg
Dobutamine 5mcg
Vancomycin
Amikacin
Zosyn
ASA 81
Nexium 40 BID

PHYSICAL EXAM: Vital Signs BP 86/52 HR 91 Resp Vent T 98 SAT 100% on 40% FIO2 Ht WT

General Appearance: Intubated, non-responsive to verbal stimuli + pain
Skin
Eyes: anicteric sclera
ENT/Neck: Ø JVD
Chest
Breasts
Lungs: coarse BS
CV: Ø JVD, S4 Ø, S3 Ø, Murmur Ø
Extremities: edema Ø, varicosities
GI: Soft, ND, BS
GU
GU penis/scrotum
Lymphatic
Musculoskeletal
Psychiatric
Neurologic: unresponsive to ~~pe tactile~~ verbal stimuli.
pulses: b/L LE w/ SCDs, warm to touch
vaginal
prostate

LABORATORY DATA: Ca: 6.4 Mag: 1.4
Lactic Acid: 2.7
Chem. 144 | 114 | 60 / 4.5 | 15 | 2.2
CBC 14.4 / 8.2K / 25 / 250
ECG LBBB (incomplete), Sinus Tachy, repeat: Sinus Tachy
CXR: no acute process (per pulm), pulm vasc congestion
OTHER: U/A (+) 120 WBC, (-) bacteria. Blood Cx: NGTD.
CT head (6/24): mild cerebral atrophy w/ disproportionate enlargement of lat ventricle, non-spec white matter dz.

DOA: (+) trended CE:
1) 107/.18
2) 273/.187
3) 391/.19

proBNP: 635

RESIDENT/PHYSICIAN EXTENDER [signature] Provider # 636
Date/Time 6/25/09 — 10:05 AM

(Attending physician comment(s))
Seen + examined pt, seen + examined by me. Agree with Dr. Patel's assessment.
Rec — as above with re-check echo with contrast for function, LV c/o [illegible]

ATTENDING [signature] Provider # 751
Date/Time

MERRIFIELD, WILLIAM M
Unit # 13895767 IM MCU
Acct # 0917500942 ADM: 06/24/09
DOB: 10/30/45 63Y M

Consultation Request/Report

Shands Jacksonville

Form # 120017
Page 1 of 1
Approved: 01/08
Revised: 08/08

Distribution: White - Chart; Yellow - Physician; Pink - Physician

Name ______ Age ____ Sex ____ Date of request ___/___/___ Time of Request ______
Unit No ______ Rm ____ Consulted Service ______ Urgent ____ Routine ____
Preop. Surgery ______ Date of Surgery ___/___/___
Requested by ______ (Attending/Resident) M.D. of the ______ Service
CC / Reason for consult ______

IMPRESSION:
1. Altered mental status
2. Hypothermia / SIRS – shock (septic)
3. ARF
4.
5. R/O ACS
6.

RECOMMENDATIONS:
1. ✓ TSH, CAPol, EEG UA, UrCx, 2D ECHO, HTN
2. PRVC – TV 400 PEEP 5 – FiO2 40% ,1p NG
3.
4. Vanc, Zosyn, Amikacin ✓ VAz
5.
6. level [illegible] [illegible]

Hx: (include hx elements, severity, quality, location, duration, context, timing, associated symptoms)
Inmate brought in for resp. distress. Intubated in ER. Adm critically ill.
& lethargy since am.
⊕ Posturing decerebrate ⊕ gaze preference, agonal respirations

ER NS-4L Vanc Zosyn Amik Cefepime Acyclovir

SOCIAL HX: marital status, occupation, tobacco, alcohol, drugs — UTA
FH: Mother, Father, Brother, Sister, Other — UTA
PMH: UTA ? per ER Records IDDM, HTN, [illegible]
Surgical Hx: UTA
ALLERGIES: Unknown
MEDICATIONS: outpatient: ? ~~not known~~ Plendil (felodipine) Lotensin Coreg Lantus
MEDICATIONS: inpatient

ROS (describe or indicate negative ( )) UTA
Constitution, Musculoskeletal, Hematologic, Neuro, Allergic/Immune, Eye, GI, Skin, Psychiatric, Respiratory 18/5, ☐ All other systems negative, ENT, GU, Gyn, Endocrine, CV

PHYSICAL EXAM: Vital Signs BP 90/48 HR 110 Resp 34/20 T 95.2 SAT 98% Ht WT SIMV PEEP TV-450 PS FiO2 40%
General Appearance Intubated Vent
Skin
Eyes dry skin & m.m.
ENT/Neck
Chest Bilateral crackles
Breasts
Lungs
CV S1S2 JVD Ø S4 Ø S3 Ø Murmur Ø
Extremities Ø edema varicosities
GI
GU S/NT ND
GU penis/scrotum
Lymphatic
Musculoskeletal
Psychiatric off sedation Ø response moves all extremities
Neurologic
pulses 2+
vaginal
prostate

(Attending physician comments)

Admit to ICU

LABORATORY DATA:
Chem. 141 | 107 | 75 / 55 ; 4.8 | 18 | 2.5 — baseline not known
CBC (bands) 8 > 11 / 33.4 < 361
ECG
CXR
OTHER:
Ca 2.5
58
lactate 3.3
DOA – ⊕ benzo
CXR ? RML pna.
Bcx (P)
INR 1.2 PT 42
Troponin 0.186
ABG 100% Vent 7.33/30/532
Ct head – mild CAE ischemic
EKG – LBBB, TWIs
UA Ø

RESIDENT/ PHYSICIAN EXTENDER Kale Provider # 6651
Date/Time 6/24/09 6pm
ATTENDING [signature] Provider # [illegible]
Date/Time 6/25/09 6 pm

MERRIFIELD, WILLIAM M
Unit # 13855767 ER EDA1
Acct # 0917500942 ADM:06/24/09
DOB: 10/30/45 63Y M

Consultation Request/Report

Shands Jacksonville

Form # 120017
Page 1 of 1
Approved: 01/06
Revised: 06/08

Distribution: White - Chart; Yellow - Physician; Pink - Physician

Patient Name: MERRIFIELD,WILLIAM M
Med Rec #: 13895767
Location: 7N 705-02
Physician: LAOS, LUIS F
Acct #: 0917500942

HEMATOLOGY

DATE:	06/27/09	[----------06/26/09----------			06/25/09	Ref Range	Units
TIME:	0200	1445	0850	0313	1610		
RBC Morph	(cont)			(n)			
Platelet Morph	Adequate			Adequate			
Comment	(o)			(p)			
Diff Type	AUTO						

HEMATOLOGY

DATE:	[-------06/25/09-------]		[-------06/24/09-------]		Ref Range	Units
TIME:	1150	0335	2210	1320		
WBC	15.8 H	14.4 H	9.6	8.0	4.5-11.0	thou/mm3
RBC	2.58 L	2.78 L	3.03 L	3.76 L	4.50-6.30	mill/mm3
Hgb	7.5 L	8.2 L	8.9 L	11.0 L	14.0-18.0	g/dL
Hct	22.8 L	25.0 L	26.8 L	33.4 L	40.0-54.0	%
MCV	88.4	89.9	88.4	88.8	82.0-101.0	fL
MCH	29.1	29.5	29.4	29.3	27.0-34.0	pg
MCHC	32.9	32.8	33.2	32.9	31.0-36.0	g/dL
RDW	15.7 H	15.6 H	15.4 H	15.3 H	13.0-15.0	%
Platelets	178	250	216	361	140-440	thou/mm3
			CKD SMEAR			
MPV	11.5 H	11.9 H	11.7 H	11.2 H	7.4-10.4	fL
Granulocytes		64.0	60.0	80.2 H	34.0-73.0	%
Grans Abs		13.2	8.3	6.4		thou/mm3
Bands		28.0	27.0			%

---FOOTNOTES---
AUTO Automated Differential
(n) Slight Hypochromia
(o) Smear reviewed, manual differential not indicated.
(p) Manual Differential

Shahla Masood, MD

INPATIENT MEDICAL RECORDS COPY
CONTINUED

MERRIFIELD,WILLIAM M
7N 705-02
M-10/30/1945
LAOS, LUIS F

INPATIENT MEDICAL RECORDS COPY

Patient Name: MERRIFIELD,WILLIAM M
Med Rec #: 13895767
Location: 7N 705-02
Physician: LAOS, LUIS F
Acct #: 0917500942

HEMATOLOGY

DATE:	[-------06/25/09-------]		[-------06/24/09-------]		Ref Range	Units
TIME:	1150	0335	2210	1320		
Imm Gran				0.5	0-2	%
Imm Gran Abs			0.1	0.0		thou/mm3
Lymphs		6.0 L	6.0 L	12.2 L	25.0-45.0	%
Lymphs Abs		0.9	0.6	1.0		thou/mm3
Monos		2.0	6.0	4.1	2.0-6.0	%
Monos Abs		0.3	0.6	0.3		thou/mm3
Eos				2.4	1.0-4.0	%
Eos Abs				0.2		thou/mm3
Basos				0.6	0-1	%
Basos Abs				0.1		thou/mm3
Metas						%
WBC Morph			1			
RBC Morph		(q)	Few Vacuoles (r)			
		(s)	(s)			
Platelet Morph		Adequate	Adequate			
Comment			(t)			
Diff Type		(u)	(u)	AUTO		

---FOOTNOTES---
AUTO Automated Differential
(q) Slight Anisocytosis
(r) Few Microcytes
(s) Few Elliptocytes
(t) REVIEWED BY CYB
(u) Manual Differential

Shahla Masood, MD

INPATIENT MEDICAL RECORDS COPY
CONTINUED

MERRIFIELD,WILLIAM M
7N 705-02
M-10/30/1945
LAOS, LUIS F

INPATIENT MEDICAL RECORDS COPY

Patient Name: MERRIFIELD,WILLIAM M
Med Rec #: 13895767
Location: 7N 705-02
Physician: LAOS, LUIS F
Acct #: 0917500942

********** COAGULATION **********

DATE:	07/04/09	[----------07/03/09----------]			06/30/09	Ref Range	Units
TIME:	*1212	*2202	*2200	*0639	*0530		
PT	21.5 H			20.3 H	18.4 H	10.0-13.0	seconds
INR	1.8 H PTINR			1.7 H PTINR	1.5 H PTINR		
PTT	82 HP			70 HP		20-36	seconds
D-dimer			2.10 H DDCUT			0.0-0.6	ugFEU/ml
FibrinSplitProducts		Negative				NEG	

========== COAGULATION ==========

DATE:	06/24/09	Ref Range	Units
TIME:	1320		
PT	15.2 H	10.0-13.0	seconds
INR	1.2 H PTINR		
PTT	42 H	20-36	seconds

---FOOTNOTES---
DDCUT The FDA approved cut-off level recommended for the exclusion of pulmonary embolism (PE) or deep vein thrombosis (DVT) for the Biomerieux VIDAS D-Dimer Exclusion Assay is 0.50 ug FEU/ml.
PTINR The INR is only valid for the monitoring of patients receiving coumadin (Warfarin) therapy.

Shahla Masood, MD

INPATIENT MEDICAL RECORDS COPY
CONTINUED

MERRIFIELD,WILLIAM M
7N 705-02
M-10/30/1945
LAOS, LUIS F

INPATIENT MEDICAL RECORDS COPY

Patient Name: MERRIFIELD,WILLIAM M
Med Rec #: 13895767
Location: 7N 705-02
Physician: LAOS, LUIS F
Acct #: 0917500942

ELECTROLYTE AND METABOLIC VALUES

DATE:	06/25/09				Ref Range	Units
TIME:	1610	1150	0930	0335		
Alkaline Phosphatase		73			40-129	U/L
ALT/SGPT		21			10-42	U/L
AST/SGOT		37 H			14-33	U/L
Phosphorus	3.3		3.2	3.5	2.5-4.5	mg/dL
Magnesium	1.2 L		1.3 L	1.4 L	1.8-2.6	mg/dL
Lactic Acid	2.2 LACINF		2.2 LACINF	3.0 H LACINF	0.7-2.7	mmol/L
Lipase		5			0-60	U/L

ELECTROLYTE AND METABOLIC VALUES

DATE:	06/24/09			Ref Range	Units
TIME:	1320	1320	0205		
Sodium	141			135-141	mmol/L
Potassium	4.8 H			3.3-4.6	mmol/L
Chloride	107			101-110	mmol/L
CO2	18 L			21-29	mmol/L
Glucose	55 L			71-99	mg/dL
BUN	75 H			6-22	mg/dL
Creatinine	2.54 H			0.5-1.1	mg/dL
Calcium	8.8 CALCOM			8.6-10.0	mg/dL
Anion Gap	16			4-16	
BUN/Creat Ratio	29.5			7.5-34.0	
Calc. Osmolality	301 H			270-288	mOsm/kg
eGFR Non-Afr. Amer.	26 L			>60	mL/Min/1.73m2
eGFR for Afr. Amer.	31 L			>60	mL/Min/1.73m2

---FOOTNOTES---
LACINF Glycolate (a metabolite of ethylene glycol) may cause a false elevation in lactate.

Shahla Masood, MD

INPATIENT MEDICAL RECORDS COPY
CONTINUED

MERRIFIELD,WILLIAM M
7N 705-02
M-10/30/1945
LAOS, LUIS F

INPATIENT MEDICAL RECORDS COPY

DATE & TIME	DATE, TIME AND SIGN ALL ENTRIES
7/5/09 1354m	Death Summary I was called for this this pt - He was 63 yr M c DM. HTN. Crohn's disease who was admitted to Mercy a couple of weeks ago c AMS & encephalopathy. pt was made DNI/DNR & then transferred to medicine service. He was not doing well on floor so Hospice was consulted after confirming c family. When nurse called me this morning pt was desaturating. ~~Dr~~ pt was started on morphine drip for comfort measures at 1am. At 1354m nurse called me again and pt was breathless, pulseless. no pupillary reflex, Cog reflex, or corneal reflex. So I pronounced death @ 1354m. Family was informed & chaplain was been called D# 169158

6635

DATE & TIME	DATE, TIME AND SIGN ALL ENTRIES
7/5/09	NF addendum
1AM	I have called for this pt because
	pt was desatting. I went there. pt
	was saturating 70-80%. Rapid respond
	was called. pt already received breathing
	treatment - pt is DNI/DNR and
	HOSPICE have already been consulted
	earlier this week. I called the
	wife & son again. left a message
	for his son & talked to wife
	who said she wants comfort
	measures. So I started the pt
	on morphine drip. Discontinued the
	other meds & IVFs. Case was
	discussed c̄ Senior resident Dr Ahmed
	& agrees c̄ plan
	[signature] 6635

Physician's Progress Notes

Shands Jacksonville

Form # 120015
Page 2 of 2
Approved: 07/0C
Revised: 06/06

MERRIFIELD, WILLIAM M
Unit # 13895767 IMC 7N
Acct # 0917500942 ADM:06/24/09
DOB: 10/30/45 63Y M

Search

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J K L M N O P Q R
S T U V W X Y Z

In This Topic

Respiratory Failure
Causes
Symptoms
Diagnosis
Treatment

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Respiratory Failure

Respiratory failure (lung failure) is a condition in which the level of oxygen in the blood becomes dangerously low or the level of carbon dioxide becomes dangerously high.

- Conditions that block the airways, damage lung tissue, weaken the muscles that control breathing, or decrease the drive to breathe may cause lung failure.
- People may be very short of breath, have a bluish coloration to the skin, and be confused or sleepy.
- Doctors use blood tests to detect low levels of oxygen or high levels of carbon dioxide in the blood.
- Oxygen is given.
- Sometimes people need the help of a machine to breathe until the underlying problem can be treated.

Pronunciations

amyotrophic lateral sclerosis
apnea
arrhythmia
bronchi
bronchiectasis

Respiratory failure is a medical emergency that can result from long-standing, progressively worsening lung disease or from severe lung disease that develops suddenly, such as the acute respiratory distress syndrome (see Respiratory Failure and Acute Respiratory Distress Syndrome: Acute Respiratory Distress Syndrome (ARDS)), in otherwise healthy people.

Causes

Almost any condition that affects breathing or the lungs can lead to respiratory failure. Certain disorders, such as hypothyroidism or sleep apnea, can decrease the unconscious reflex that drives people to breathe. An overdose of opioids or alcohol also can decrease the drive to breathe by causing profound sedation. Obstruction of the airways, injury to the lung tissues, damage to the bones and tissues around the lungs, and weakness of the muscles that normally inflate the lungs are also common causes. Respiratory failure can occur if blood flow through the lungs becomes abnormal, as happens in pulmonary embolism (see Pulmonary Embolism (PE): Pulmonary Embolism). This disorder does not stop air from moving in and out of the lungs, but without blood flow to a portion of the lungs, oxygen is not properly extracted from the air.

Symptoms

Low oxygen levels in the blood can cause shortness of breath and result in a bluish coloration to the skin (cyanosis). Low oxygen levels, high carbon dioxide levels, and increasing acidity of the blood cause confusion and sleepiness. If the drive to breathe is normal, the body tries to rid itself of carbon dioxide by deep, rapid breathing. If the lungs cannot function normally, however,

Did You Know?

- Age-related reductions in lung function place older people at higher risk of severe symptoms after developing pneumonia.

this breathing pattern may not help. Eventually, the brain and heart malfunction, resulting in drowsiness (sometimes to the point of becoming unconscious) and abnormal heart rhythms (arrhythmias), both of which can lead to death.

Some symptoms of respiratory failure vary with the cause. A child with an obstructed airway due to the inhalation (aspiration) of a foreign object (such as a coin or a toy) may suddenly gasp and struggle for breath (see First Aid: Choking). People with acute respiratory distress syndrome may become severely short of breath over a period of hours. Someone who is intoxicated or weak may quietly slip into a coma.

Diagnosis

A doctor may suspect respiratory failure because of the symptoms and physical examination findings. A blood test done on a sample taken from an artery confirms the diagnosis when it shows a dangerously low level of oxygen or a dangerously high level of carbon dioxide. Chest x-rays and other tests are done to determine the cause of respiratory failure.

Treatment

People with respiratory failure are treated in an intensive care unit. Oxygen is given initially, usually in a greater amount than is needed, but the amount of oxygen can be adjusted at a later time. Occasionally, in people in whom carbon dioxide levels have remained high for some time, excess oxygen can result in slowing of the movement of air (ventilation) in and out of the lungs and a dangerous further increase in the carbon dioxide level. In such people, the dosage of oxygen needs to be more carefully regulated.

The underlying disorder causing the respiratory failure must also be treated. For example, antibiotics are used to fight bacterial infection, and bronchodilators are used in people with asthma to open the airways. Other drugs may be given, for example, to decrease inflammation or treat blood clots. Mechanical ventilation is necessary unless respiratory failure resolves rapidly.

What Causes Respiratory Failure?

Underlying Problem	Cause
Airway obstruction	Chronic obstructive pulmonary disease, asthma, bronchiectasis, cystic fibrosis, bronchiolitis, inhaled foreign bodies
Poor breathing (decrease in the drive to breathe)	Obesity, sleep apnea, hypothyroidism, drug or alcohol intoxication
Muscle weakness	Myasthenia gravis, muscular dystrophy, polio, Guillain-Barré syndrome, polymyositis, certain strokes, amyotrophic lateral sclerosis (ALS), spinal cord injury
Abnormality of lung tissue	Acute respiratory distress syndrome (ARDS), pneumonia, pulmonary edema (excess fluid in the lungs) from heart or kidney failure, drug reaction, pulmonary fibrosis, widespread tumors, radiation, sarcoidosis, burns
Abnormality of chest wall	Scoliosis, chest wound, extreme obesity, deformities resulting from chest surgery

Last full review/revision January 2008 by Brian K. Gehlbach, MD; Jesse B. Hall, MD

Back to Top

Previous: Mechanical Ventilation

Subendocardial or Non-Q-wave Infarct

Subendocardial infarct is an area of damaged and necrotic tissue on the innermost layer of the ventricle. Subendocardial infarct can result if subendocardial ischemia is allowed to persist without treatment. It can also be the result of a stenotic lesion in the associated coronary artery. Subendocardial infarct is not relieved with nitroglycerin, and the associated changes seen in the electrocardiogram do not revert to normal baseline.

The most notable ECG change is a persistent S-T segment depression. Subendocardial infarct affects ventricular repolarization, resulting in an S-T - T complex being seen without depolarization.

Return to Electrocardiography

Metabolic Encephalopathy

:: En Español (Spanish Version)

(Coma, Hepatic; Hepatic Coma; Hepatic Encephalopathy; Portal-Systemic Encephalopathy)

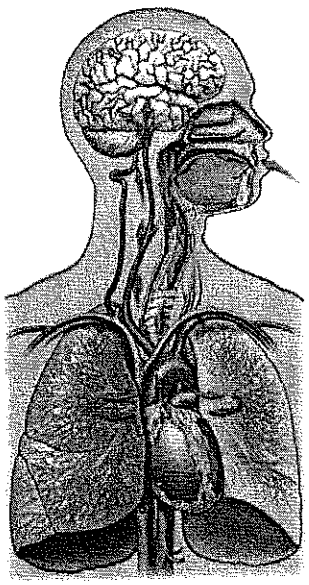
Pronounced: MET-a-bol-ik En-SEF-a-lo-PATH-ee

by :: Nathalie Smith, MSN, RN

Definition

This condition is temporary or permanent damage to the brain. The most common cause is an illness or condition that affects the liver. Toxins build up in the bloodstream because the liver is not working normally.

Normal Oxygen Flow to Brain



An interruption of this flow can lead to metabolic encephalopathy.

Causes

Metabolic encephalopathy is caused by:

- Poisoning
- Diseases (eg, :: cirrhosis , :: hepatitis , :: diabetes , :: heart disease , :: renal failure)
- Medical conditions that cause blood circulation to bypass the liver

Risk Factors

These factors increase your chance of developing metabolic encephalopathy:

- :: Acute renal failure (renal disease)
- :: Vitamin B1 deficiency
- :: Hypoglycemia
- :: Hyperosmolar hyperglycemic crisis
- Metabolic acidosis
- Hyponatremia
- Hypernatremia
- Hypercalcemia
- :: Sepsis
- :: Hashimoto's thyroiditis
- :: Acute adrenocortical insufficiency
- :: Meningitis
- :: Encephalitis
- :: Hypothermia
- Hyperthermia
- Previous history of brain injury (eg, :: tumor , :: stroke)

Tell your doctor if you have any of these risk factors.

If you have liver problems, the risk of metabolic encephalopathy is increased by:

- Low oxygen levels in the blood
- Infections
- Major surgery
- Any serious illness that causes changes in the body's chemical make-up or metabolism
- Use of certain medicines, such as sedatives and :: narcotics
- Bleeding within the intestines
- Persistent vomiting or :: diarrhea that lowers blood potassium levels

The following toxic overdoses can also increase your risk of the condition:

- :: Alcohol intoxication
- Sedative-hypnotic overdose
- :: Lead poisoning
- :: Carbon monoxide poisoning

Symptoms

Symptoms include:

- Confusion or agitation
- Changes in behavior and personality
- Forgetfulness
- Disorientation
- :: Insomnia
- Muscle stiffness or rigidity
- :: Tremor (particularly a flapping tremor of the hands)
- Difficulty speaking
- Asterixis (rapid momentary loss of tone in the muscles)
- Uncontrollable movements or :: seizures (rare)
- Stupor or :: coma

These problems can develop quickly. They may resolve when the condition is reversed. However, prompt treatment is needed before a coma occurs.

Diagnosis

This condition is very serious. It can quickly become an emergency. You will need to be hospitalized. Doctors will do an exam to assess your neurological condition.

The following tests may be done:

- Blood tests--usually show high blood ammonia levels and other abnormalities related to the failing liver
- :: Electroencephalogram (EEG)—may be used to determine how the brain has been affected
- Imaging studies of the brain (:: CT scan or :: MRI)—may be done to evaluate other causes

Treatment
Hospitalization and Emergency Care

In the hospital, the staff will treat the problems that caused the condition. They will try to remove or neutralize toxins that have built up in the bloodstream. The goal is to reverse the underlying condition. But, brain injury can still occur. In some cases, brain injury is permanent.

Medications

Medications may be used to:

- Neutralize toxins
- Treat the condition
- Reduce recurrence

Dietary Restrictions

You may need to eat a :: low-protein diet to help lower blood ammonia levels. (The body creates ammonia when it metabolizes and uses protein.) You may have other changes in your diet.

Tube feeding and life support may be needed, especially in the case of coma.

Transplantation

If this condition is due to organ failure, you may need a :: transplant .

Prevention

To help reduce your chance of getting this condition, take the following steps:

- Get early treatment for liver problems. If you have liver problems and any of the above symptoms, call your doctor right away.
- If you have a disease (such as cirrhosis), see your doctor regularly.
- Avoid overdosing. Avoid being exposed to poisons or toxins.

RESOURCES:

National Digestive Diseases Information Clearinghouse
http://digestive.niddk.nih.gov/index.htm/

National Institute of Neurological Disorders and Stroke
http://www.ninds.nih.gov/

CANADIAN RESOURCES:

Canadian Liver Foundation
http://www.liver.ca/Home.aspx/

Health Canada
http://www.hc-sc.gc.ca/index_e.html/

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4/2/2010 DynaMed's Systematic Literature Surveillance DynaMed's Systematic Literature Surveillance: FDA approves new use of Xifaxan for patients with liver disease. US Food and Drug Administration website. Available at:

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Pneumonia

URL of this page: http://www.nlm.nih.gov/medlineplus/ency/article/000145.htm

Pneumonia is a respiratory condition in which there is infection of the lung.

Community-acquired pneumonia refers to pneumonia in people who have not recently been in the hospital or another health care facility (nursing home, rehabilitation facility).

See also: Hospital-acquired pneumonia

Causes

Pneumonia is a common illness that affects millions of people each year in the United States. Germs called bacteria, viruses, and fungi may cause pneumonia.

Ways you can get pneumonia include:

- Bacteria and viruses living in your nose, sinuses, or mouth may spread to your lungs.
- You may breathe some of these germs directly into your lungs.
- You breathe in (inhale) food, liquids, vomit, or secretions from the mouth into your lungs (aspiration pneumonia)

Pneumonia caused by bacteria tends to be the most serious. In adults, bacteria are the most common cause of pneumonia.

- The most common pneumonia-causing germ in adults is *Streptococcus pneumoniae* (pneumococcus).
- Atypical pneumonia, often called walking pneumonia, is caused by bacteria such as *Legionella pneumophila*, *Mycoplasma pneumoniae*, and *Chlamydophila pneumoniae*.
- *Pneumocystis jiroveci* pneumonia is sometimes seen in people whose immune system is impaired (due to AIDS or certain medications that suppress the immune system).
- *Staphylococcus aureus*, *Moraxella catarrhalis*, *Streptococcus pyogenes*, *Neisseria meningitidis*, *Klebsiella pneumoniae*, or *Haemophilus influenzae* are other bacteria that can cause pneumonia.
- Tuberculosis can cause pneumonia in some people, especially those with a weak immune system.

Viruses are also a common cause of pneumonia, especially in infants and young children.

See also: Respiratory syncytial virus

Risk factors (conditions that increase your chances of getting pneumonia) include:

- Cerebral palsy
- Chronic lung disease (COPD, bronchiectasis, cystic fibrosis)
- Cigarette smoking
- Difficulty swallowing (due to stroke, dementia, Parkinson's disease, or other neurological conditions)
- Immune system problem (See also: Pneumonia in immunocompromised host)

- Impaired consciousness (loss of brain function due to dementia, stroke, or other neurologic conditions)
- Living in a nursing facility
- Other serious illnesses, such as heart disease, liver cirrhosis, or diabetes mellitus
- Recent surgery or trauma
- Recent viral respiratory infection (common cold, laryngitis, influenza)

Symptoms

The most common symptoms of pneumonia are:

- Cough (with some pneumonias you may cough up greenish or yellow mucus, or even bloody mucus)
- Fever, which may be mild or high
- Shaking chills
- Shortness of breath (may only occur when you climb stairs)

Additional symptoms include:

- Confusion, especially in older people
- Excessive sweating and clammy skin
- Headache
- Loss of appetite, low energy, and fatigue
- Sharp or stabbing chest pain that gets worse when you breathe deeply or cough

Exams and Tests

If you have pneumonia, you may be working hard to breathe, or breathing fast.

Crackles are heard when listening to your chest with a stethoscope. Other abnormal breathing sounds may also be heard through the stethoscope or via percussion (tapping on your chest wall).

The health care provider will likely order a chest x-ray if pneumonia is suspected.

Some patients may need other tests, including:

- Arterial blood gases to see if enough oxygen is getting into your blood from the lungs
- CBC to check white blood cell count
- CT scan of the chest
- Gram's stain and culture of your sputum to look for the organism causing your symptoms
- Pleural fluid culture if there is fluid in the space surrounding the lungs

Treatment

Your doctor must first decide whether you need to be in the hospital. If you are treated in the hospital, you will receive fluids and antibiotics in your veins, oxygen therapy, and possibly breathing treatments. It is very important that your antibiotics are started very soon after you are admitted.

You are more likely to be admitted to the hospital if you:

- Have another serious medical problem
- Have severe symptoms
- Are unable to care for yourself at home, or are unable to eat or drink
- Are older than 65 or a young child

- Have been taking antibiotics at home and are not getting better

However, many people can be treated at home. If bacteria are causing the pneumonia, the doctor will try to cure the infection with antibiotics. It may be hard for your health care provider to know whether you have a viral or bacterial pneumonia, so you may receive antibiotics.

Patients with mild pneumonia who are otherwise healthy are sometimes treated with oral macrolide antibiotics (azithromycin, clarithromycin, or erythromycin).

Patients with other serious illnesses, such as heart disease, chronic obstructive pulmonary disease, or emphysema, kidney disease, or diabetes are often given one of the following:

- Fluoroquinolone (levofloxacin [Levaquin], gemifloxacin [Factive], or moxifloxacin [Avelox])
- High-dose amoxicillin or amoxicillin-clavulanate, plus a macrolide antibiotic (azithromycin, clarithromycin, or erythromycin)
- Cephalosporin antibiotics (for example, cefuroxime or cefpodoxime) plus a macrolide (azithromycin, clarithromycin, or erythromycin)

If the cause is a virus, typical antibiotics will NOT be effective. Sometimes, however, your doctor may use antiviral medication.

You can take these steps at home:

- Control your fever with aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs, such as ibuprofen or naproxen), or acetaminophen. DO NOT give aspirin to children.
- Do not take cough medicines without first talking to your doctor. Cough medicines may make it harder for your body to cough up the extra sputum.
- Drink plenty of fluids to help loosen secretions and bring up phlegm.
- Get lots of rest. Have someone else do household chores.

Outlook (Prognosis)

With treatment, most patients will improve within 2 weeks. Elderly or debilitated patients may need longer treatment.

Those who may be more likely to have complicated pneumonia include:

- Older adults or very young children
- People whose immune system does not work well
- People with other, serious medical problems such as diabetes or cirrhosis of the liver

Your doctor may want to make sure your chest x-ray becomes normal again after you take a course of antibiotics. However, it may take many weeks for your x-ray to clear up.

Possible Complications

Possible complications include:

- Acute respiratory distress syndrome (ARDS), a severe form of respiratory failure
- Empyema or lung abscesses. These are infrequent, but serious, complications of pneumonia. They occur when pockets of pus form inside or around the lung. These may sometimes need to be drained with surgery.
- Respiratory failure, which requires a breathing machine or ventilator
- Sepsis, a condition in which there is uncontrolled swelling (inflammation) in the body, which may lead to organ failure

Pulmonary collapse

Introduction: Pulmonary collapse

Description of Pulmonary collapse

Pulmonary collapse: secondary **atelectasis** due to bronchial obstruction, pleural effusion or **pneumothorax** , cardiac **hypertrophy** , or enlargement of other structures adjacent to the lungs. Often used in the sens of massive collapse.

Source: **Stedman** **'s Medical Spellchecker** , © 2006 Lippincott Williams & **Wilkins** . All rights reserved.

Pulmonary collapse: collapse of all or part of a lung due to bronchial plugging or the **chest cavity** being opened to atomspheric pressure.

Source: Diseases Database

Pulmonary collapse: Related Topics

These medical condition or symptom topics may be relevant to medical information for Pulmonary collapse:

- Pulmonary
- Pulmonary symptoms (3280 causes)
- Collapse (105 causes)
- Atelectasis (22 causes)
- Bronchial
- Bronchial symptoms (273 causes)
- Bronchial disorder
- Obstruction
- Pleural
- Pleural diseases

Terms associated with Pulmonary collapse:

Terms Similar to Pulmonary collapse:

- Lung atelectasis

Source: Diseases Database

Hierarchical classifications of Pulmonary collapse

The following list attempts to classify Pulmonary collapse into categories where each line is subset of the next.

Disease Ontology - OBO (Open Biomedical Ontologies)

- Other diseases of lung
- OTHER DISEASES OF RESPIRATORY SYSTEM
- DISEASES OF THE RESPIRATORY SYSTEM
- DISEASES AND INJURIES
- Disease Ontology V2.1 2005

External links related to: Pulmonary collapse

- eMedicine - Atelectasis : Article by Sat Sharma, MD, FRCPC, FACP, FCCP, DABSM
- airless lung
- atelectasis
- lung collapse - General Practice Notebook
- eMedicine - Atelectasis, Pulmonary : Article by Michael R Bye, MD

Source: Diseases Database

Interesting Medical Articles:

- Symptoms of the Silent Killer Diseases
- Online Diagnosis
- Self Diagnosis Pitfalls

Melena

From Wikipedia, the free encyclopedia

In medicine, **melena** or **melaena** refers to the black, "tarry" feces that are associated with gastrointestinal hemorrhage.[1] The black color is caused by oxidation of the iron in hemoglobin during its passage through the ileum and colon

Melena	
Classification and external resources	
ICD-10	K92.1
ICD-9	578.1

Contents

Melena vs. hematochezia

Bleeding originating from the lower GI tract (such as the sigmoid colon and rectum) is generally associated with the passage of bright red blood, or hematochezia, particularly when brisk. Blood acts as a cathartic agent in the intestine, promoting its prompt passage. Only blood that originates from a high source (such as the small intestine), or bleeding from a lower source that occurs slowly enough to allow for enzymatic breakdown is associated with melena. For this reason, melena is often associated with blood in the stomach or duodenum (upper gastrointestinal tract), for example by a peptic ulcer. A rough estimate is that it takes about 14 hours for blood to be broken down within the intestinal lumen; therefore if transit time is less than 14 hours the patient will have hematochezia, and if greater than 14 hours the patient will exhibit melena. One often-stated rule of thumb is that melena only occurs if the source of bleeding is above the ligament of Treitz although, as noted below, exceptions occur with enough frequency to render it unreliable.

Diagnosis

Patients sometimes present with signs of anemia or those due to low blood pressure. Very often, however, aside from the melena itself, there are no other symptoms. The presence of blood must be confirmed with either a positive hemoccult slide on rectal exam, frank blood on the examining finger, or a positive stool guaiac from the lab. If a source in the upper GI tract is suspected, an upper endoscopy can be performed to diagnose the cause. Lower GI bleeding sources usually present with hematochezia or frank blood. A test with poor sensitivity/specificity that may detect the source of bleeding is the tagged red blood cell scan, whereas mesenteric angiogram is the gold standard.

Causes

The most common cause of melena is peptic ulcer disease. Any other cause of bleeding from the upper gastro-intestinal tract, or even the ascending colon, can also cause melena. Melena may also be a sign of drug overdose if a patient is taking anti-coagulants, such as warfarin. It is also caused by tumours

especially malignant tumors affecting the esophagous, more commonly the stomach & less commonly the small intestine due to the bleeding surface of them. However,the most prominent and helpful sign in these cases of malignant tumours is haematemesis. It may also accompany hemorrhagic blood diseases (e.g. purpura & hemophilia). Other medical causes of melena include bleeding ulcers, gastritis, esophageal varices, and Mallory-Weiss syndrome.

Some causes of "false" melena include Iron supplements, Pepto-Bismol, Maalox, and Lead. Although blood swallowed as a result of a nose bleed (epistaxis) can lead to melena, it is obviously not due to bleeding in the gastro-intestinal tract.

Melena is often not a medical emergency because the bleeding is slow. Urgent care however is required to rule out serious causes and prevent potentially life-threatening emergencies.

A less serious, self-limiting case of melena can occur in newborns two to three days after delivery, due to swallowed maternal blood.

See also

- Blood in stool
- Hematemesis
- Dieulafoy's lesion

References

1. ^ MedlinePlus Encyclopedia *Bloody or tarry stools*, retrieved 19 July 2010.

Featured on MedicineNet
- Causes of Fatigue Slideshow
- Things to Do About Depression
- Fibromyalgia Friendly Exercises Slideshow
- Quiz: Are You Depressed?
- Depression Tips Slideshow

Definition of Regional enteritis Related Articles
- Abdominal Pain
- Crohn's Disease
- Diarrhea
- Teenagers

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Webster's New World Medical Dictionary
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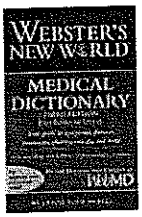

MedTerms Medical Word of the Day

Definition of Regional enteritis

Digestive Disease Myths Slideshow

Regional enteritis: Crohn's disease, a chronic inflammatory disease of the intestine primarily in the small and large intestines but which can occur anywhere in the digestive system between the mouth and the anus.

Named after Burrill Crohn who described the disease in 1932. The disease usually affects persons in their teens or early twenties. It tends to be chronic, recurrent with periods of remission and exacerbation. In the early stages, it causes small scattered shallow crater-like areas (erosions) called aphthous ulcers in the inner surface of the bowel. With time, deeper and larger ulcers develop, causing scarring and stiffness of the bowel and the bowel becomes increasingly narrowed, leading to obstruction. Deep ulcers can puncture holes in the bowel wall, leading to infection in the abdominal cavity (peritonitis) and in adjacent organs. When only the large intestine (colon) is involved, the condition is called Crohn's colitis. When only the small intestine is involved, the condition is called Crohn's enteritis. When only the end of the small intestine (the terminal ileum) is involved, it is termed terminal ileitis. When both the small intestine and the large intestine are involved, the condition is called Crohn's enterocolitis (or ileocolitis). Abdominal pain, diarrhea, vomiting, fever, and weight loss can be symptoms. Crohn's disease can be associated with reddish tender skin nodules, and inflammation of the joints, spine, eyes, and liver. Diagnosis is by barium enema, barium x-ray of the small bowel, and colonoscopy. Treatment includes medications for inflammation, immune suppression, antibiotics, or surgery. (The disease is also called granulomatous enteritis).

Last Editorial Review: 3/26/1998 2:28:00 PM

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Hyperosmolality

Introduction: Hyperosmolality

Description of Hyperosmolality

Hyperosmolality: Increased osmotic concentration of a solution expressed as osmoles of solute per kilogram of serum water.

Hyperosmolality: Related Topics

These medical condition or symptom topics may be relevant to medical information for Hyperosmolality:

- Osmotic
- Solution
- Solute
- Per
- Serum
- Serum disease
- Water

Terms associated with Hyperosmolality:

Terms Similar to Hyperosmolality:

- Hyperosmolarity
- Osmolarity raised (plasma)
- Osmolality raised (plasma)

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Alkalosis

URL of this page: http://www.nlm.nih.gov/medlineplus/ency/article/001183.htm

Alkalosis is a condition in which the body fluids have excess base (alkali). This is the opposite of excess acid (acidosis).

Causes

The kidneys and lungs maintain the proper balance of chemicals, called acids and bases, in the body. Decreased carbon dioxide (an acid) or increased bicarbonate (a base) levels make the body too alkaline, a condition called alkalosis.

Respiratory alkalosis is caused by low carbon dioxide levels in the blood. This can be due to:

- Fever
- Being at a high altitude
- Lack of oxygen
- Liver disease
- Lung disease, which causes you to breathe faster (hyperventilate)
- Salicylate poisoning

Metabolic alkalosis is caused by too much bicarbonate in the blood.

Hypochloremic alkalosis is caused by an extreme lack or loss of chloride, which can occur with prolonged vomiting.

Hypokalemic alkalosis is caused by the kidneys' response to an extreme lack or loss of potassium, which can occur when people take certain diuretic medications.

Compensated alkalosis occurs when the body returns the acid - base balance to normal in cases of alkalosis, but bicarbonate and carbon dioxide levels remain abnormal.

Symptoms

- Confusion (can progress to stupor or coma)
- Hand tremor
- Lightheadedness
- Muscle twitching
- Nausea, vomiting
- Numbness or tingling in the face or extremities
- Prolonged muscle spasms (tetany)

Exams and Tests

An arterial blood gas analysis or basic metabolic panel will confirm alkalosis and determine if it is a respiratory alkalosis or a metabolic alkalosis. Other tests may be needed to determine the cause of the alkalosis. These may include:

- Litmus paper (urine dipstick tests)
- Urinalysis
- Urine pH

Treatment

Treatment of alkalosis depends on finding the specific cause.

For alkalosis caused by hyperventilation, breathing into a paper bag causes you to retain more carbon dioxide and improves the alkalosis. If your oxygen level is low, you may receive oxygen to help the alkalosis.

Some people need medications to correct chemical loss (such as chloride and potassium). Your health care provider will monitor your vital signs (temperature, pulse, rate of breathing, blood pressure).

Outlook (Prognosis)

Most cases of alkalosis respond well to treatment.

Possible Complications

- Arrhythmias
- Coma
- Electrolyte imbalance (such as hypokalemia)

When to Contact a Medical Professional

Call your health care provider if you become confused, unable to concentrate, or unable to "catch your breath."

A visit to the emergency room or call to the local emergency number (such as 911) is warranted for:

- Loss of consciousness
- Rapidly worsening symptoms of alkalosis
- Seizures
- Severe breathing difficulties

Prevention

Prevention depends on the cause of the alkalosis. Normally, people with healthy kidneys and lungs do not have significant alkalosis.

References

Seifter JL. Acid-base disorders. In: Goldman L, Ausiello D, eds. *Cecil Medicine*. 23rd ed. Philadelphia, Pa: Saunders Elsevier; 2007:chap 119.

Update Date: 11/15/2009

Updated by: David C. Dugdale, III, MD, Professor of Medicine, Division of General Medicine, Department of Medicine, University of Washington School of Medicine. Also reviewed by David Zieve, MD, MHA, Medical Director, A.D.A.M., Inc.

Protein-calorie malnutrition

Protein-calorie malnutrition: Excerpt from Handbook of Diseases

One of the most prevalent and serious depletion disorders, **protein-calorie malnutrition** occurs as **marasmus** (protein-calorie deficiency), characterized by **growth failure** and wasting; and as **kwashiorkor** (**protein deficiency**), characterized by tissue edema and damage. Both forms vary from mild to severe and may be fatal, depending on accompanying stress (particularly **sepsis** or injury) and duration of deprivation. Protein-calorie malnutrition increases the risk of death from pneumonia, **chickenpox** , or measles.

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Causes

Both marasmus (nonedematous protein-calorie malnutrition) and kwashiorkor (**edematous** protein-calorie malnutrition) are common in underdeveloped countries and in areas where dietary amino acid content is insufficient to satisfy growth requirements. Kwashiorkor typically occurs at about age 1, after infants are weaned from breast milk to a protein-deficient diet of starchy gruels or sugar water, but it can develop at any time during the formative years. **Marasmus** affects infants ages 6 to 18 months as a result of breast-feeding failure or a debilitating condition such as chronic diarrhea.

In industrialized countries, **protein-calorie malnutrition** may occur secondary to chronic **metabolic disease** that decreases protein and calorie intake or absorption or trauma that increases protein and calorie requirements. In the United States, protein-calorie malnutrition is estimated to occur to some extent in 50% of surgical and 48% of medical patients. Those who aren't allowed anything by mouth for an extended period are at high risk for developing protein-calorie malnutrition. Conditions that increase protein-calorie requirements include severe burns and injuries, systemic infections, and cancer (accounts for the largest group of hospitalized patients with protein-calorie malnutrition.) Conditions that cause defective utilization of nutrients include **malabsorption syndrome** , **short-bowel syndrome** , and Crohn's disease.

Signs and symptoms

Children with chronic protein-calorie malnutritioin are small for their chronological age and tend to be physically inactive, mentally apathetic, and susceptible to frequent infections. Anorexia and diarrhea are common.

With acute protein-calorie malnutrition, children are small, gaunt, and emaciated, with no adipose tissue. Their skin is dry and "baggy," and their hair is sparse and dull brown or reddish yellow. Their temperatures are low; their pulse rates and respirations, slowed. Such children are weak, irritable, and usually hungry; however, they may have anorexia, with nausea and vomiting.

Unlike marasmus, chronic kwashi-orkor allows the patient to grow in height, but adipose tissue diminishes as fat metabolizes to meet energy demands. Edema commonly masks severe muscle wasting; dry, peeling skin and hepatomegaly are common. Patients with secondary protein-calorie malnutrition show signs similar to **marasmus** , primarily loss of adipose tissue and **lean body mass** , lethargy, and edema. Severe secondary protein-calorie malnutrition may cause loss of immunocompetence.

Diagnosis

Clinical features, dietary history, and anthropometry confirm **protein-calorie malnutrition** . If the patient doesn't suffer from fluid retention, weight change over time is the best index of nutritional status.

The following factors support the diagnosis:

- *height and weight* less than 80% of standard for the patient's age and sex, and below-normal arm circumference and triceps skinfold
- *serum albumin level* less than 2.8 g/dl (normal: 3.3 to 4.3 g/dl)
- *urinary creatinine (24-hour) level* is used to show lean body mass status by relating creatinine excretion to height and ideal body weight, to yield creatinine-height index

❑ *skin tests with standard antigens* to indicate degree of **immunocompromise** by determining reactivity expressed as a percentage of normal reaction

❑ *moderate anemia.*

Treatment

The aim of treatment is to provide sufficient proteins, calories, and other nutrients for nutritional rehabilitation and maintenance. When treating severe protein-calorie malnutrition, restoring fluid and electrolyte balance parenterally is the initial concern. A patient who shows normal absorption may receive enteral nutrition after anorexia has subsided. When possible, the preferred treatment is oral feeding of high-quality protein foods, especially milk, and protein-calorie supplements. A patient who's unwilling or unable to eat may require supplementary feedings through a nasogastric tube or **total parenteral nutrition** (TPN) through a central venous catheter. Accompanying infection must also be treated, preferably with antibiotics that don't inhibit protein synthesis. Cautious realimentation is essential to prevent complications from overloading the compromised metabolic system.

Special considerations

❑ Encourage the patient with **protein-calorie malnutrition** to consume as much nutritious food and fluid as possible (it may be helpful to "cheer him on" as he eats). Help the patient eat if necessary. Cooperate closely with the dietitian to monitor intake and provide acceptable meals and snacks.

❑ If TPN is necessary, observe strict aseptic technique when handling catheters, tubes, and solutions and during dressing changes.

❑ Watch for protein-calorie malnutrition in patients who have been hospitalized for a prolonged period, have had no oral intake for several days, or have cachectic disease.

❑ To help eradicate protein-calorie malnutrition in developing countries, encourage prolonged breast-feeding, educate mothers about their children's needs, and provide supplementary foods as needed.

CLINICAL TIP: *If the older patient is anorectic, consider asking family members and other visitors to bring in special foods from home that may improve the patient's appetite. In addition, encouraging the family to collaborate on feeding a dependent patient can help promote his recovery, enhance his feelings of well-being, and stimulate him to eat more.*

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Diastolic Heart Failure

The heart's pumping cycle has 2 phases:

Diastole
(pronounced : die ass tuh lee)
the heart muscle relaxes and fills with blood

Systole
(pronounced : sis tuh lee)
the heart muscle contracts (squeezes) and pumps blood out into the body

updated July 5, 2006 - People think of heart failure as a condition where the heart does not pump out enough blood. That is called systolic heart failure. However, many **CHFers** have a different kind of heart failure - caused when the heart does not fully relax, so it does not fill properly with blood. This is called diastolic heart failure. This page is about diastolic heart failure or DHF.

In mild DHF, **SOB** and fatigue usually only happen during stress or activity. More severe DHF causes many of the same symptoms that systolic heart failure or SHF, causes.

A person with DHF has high pressures in the arteries of their lungs - pulmonary pressure. Their heart's pumping chambers may not be enlarged and their **ejection fraction** may be normal, but they still have the same nasty symptoms as a person with SHF.

In people with diastolic heart failure, **Toprol-XL** may be a better beta-blocker choice than Coreg. Too-low blood pressure can be a real problem for DHF patients. Toprol-XL does not lower blood pressure as much as Coreg.

Causes of diastolic heart failure

- **Ischemia** - can be caused by **CAD** or by a chronic too-fast heart rate. Ischemia prevents the heart muscle from fully relaxing and increases heart stiffness. Chronic ischemia results in **remodeling** and DHF
- pressure overload caused by chronic high blood pressure or aortic valve problems
- infiltrative cardiomyopathies, also called restrictive cardiomyopathy
- pericarditis - inflammation of the sac around the outside of the heart
- normal aging can cause some DHF
- chemotherapy for diseases like cancer
- genetic causes

Diagnosing diastolic heart failure

How to always diagnose DHF accurately is uncertain but a correct diagnosis *is* important. Some guidelines propose that 3 requirements be met to make the call:

1. symptoms or signs of **CHF**
2. normal systolic function
3. abnormal diastolic function

There are some situations where symptoms alone may lead to a *mis*diagnosis. This is especially true in older patients who get short of breath mainly because they have very poor physical fitness. It can also happen with non-heart-related shortness of breath such as with lung disease. So diagnosing DHF requires some signs or symptoms of CHF like lung congestion, edema, raised jugular vein, etc. You do have to rule out problems like mitral valve disease and and lung disease.

DHF cannot usually be distinguished from **SHF** by patient history, physical exam, x-ray, and EKG alone. Diagnosis *requires* an estimate of LV size and **EF**. These measurements can be made using **echo, MUGA, or cath**. Really, DHF diagnosis is a matter of ruling out other possible causes in patients seeming to have heart failure but who have normal heart size and EF.

Since echo does have limits for spotting DHF and cath is invasive, one trial studied whether the BNP blood test could help spot diastolic heart failure. That study is shown on the **BNP Test page**. It turns out that a quick BNP test can help a doctor diagnose DHF.

Diagnosis: Systolic versus diastolic heart failure		
	Systolic Heart Failure - Reduced EF 30 patients	**Diastolic Heart Failure - Normal EF 20 patients**
Symptoms		
Shortness of breath on exertion	85%	96%
Waking at night short of breath	55%	50%
Difficulty breathing except upright	60%	73%
Physical Examination		
Jugular vein swollen	35%	46%
Sounds in lungs	72%	70%
Point of maximum impulse	50%	60%
S3	45%	65%
S4	45%	66%
Enlarged (congested) liver	15%	16%
Edema	30%	40%
Heart enlargement on chest x-ray	90%	96%
High pulmonary (lung) pressures	75%	80%

How common is DHF?

As many as 33% of patients with obvious heart failure and a normal EF may have DHF. Risk of DHF increases with age:

1. 15% in patients under 60 years
2. 35% in patients 60 to 70 years
3. 50% in patients over 70 years old

Mortality

Prognosis of patients with DHF is better than for SHF. Annual mortality for DHF is about 5 to 8%, while it is about 10 to 15% for SHF. In healthy people of the same age, mortality is about 1%. In DHF patients, prognosis is also affected by cause. When CAD is *not* the cause, annual mortality is much better, at about 2% to 3%. Age also affects risk of death. The 5-year mortality rate in DHF patients is:

1. 25% in patients under 60 years old
2. 35% in patients 60 to 70 years
3. 50% in patients over 70 years old

Complications

The complication rate is about the same as for SHF patients. DHF causes frequent outpatient visits and hospital admissions. The one year readmission rate is almost 50% in DHF patients.

Treatment

Treatment guidelines for systolic heart failure are based on large, properly done trials. Unfortunately, no such trials exist for DHF. So guidelines come from small trials, a doctor's experience, and his understanding of the disease. The *general* approach to treating DHF has 3 main steps:

1. Treatment should reduce symptoms, mainly by lowering **pulmonary** pressure. Ways to reduce pressure include reducing heart size, maintaining good pumping in the heart's upper chambers, and slowing the heart rate.
2. Treatment should target the underlying cause if possible. For example, high blood pressure should be controlled, **remodeling** should be reversed, the aortic valve replaced if necessary, and ischemia treated by increasing blood flow to the heart and reducing its need for oxygen
3. Treatment should target the bodily systems changed by the disease, mainly **neurohormonal** systems

Drug Therapy - General Principles

With some exceptions, many of the drugs used to treat systolic heart pressure are also used to treat diastolic heart failure. However, the reason they are used and the dose may be different for DHF.

For example, in DHF beta-blockers are used to make filling the heart with blood take longer, and to change the heart's response to exercise. In SHF, beta-blockers are used to increase pumping power and reverse heart remodeling. Diuretic dose for DHF is usually much smaller than for SHF. Calcium channel blockers have no place in SHF treatment but may help DHF.

The first step in treating DHF patients is to reduce lung congestion. You do that by lowering pulmonary (lung) pressure. This has 3 steps:

Reduce heart size

At first, heart size can be reduced by restricting fluid and sodium intake, by dialysis or filtering the blood, **plasmapheresis**, and diuretics. Relaxing (dilating) the blood vessels using nitro or morphine is effective but should be started at low doses to avoid low blood pressure. Low blood pressure can be a real problem in DHF patients. *Long-term* treatment should include small to moderate diuretic doses, mild doses of long-acting nitro, and **restricted sodium intake**. **Aldactone** (spironolactone) may be effective long-term because it suppresses the **RAS**. ACE inhibitors and **ARBs** reduce fluid retention and oxygen demand.

Sepsis and Septic Shock

Sepsis *is a serious bodywide response to bacteremia or another infection.* ***Septic shock*** *is life-threatening low blood pressure (shock) due to sepsis*

- Usually, sepsis results from certain bacterial infections, often acquired in a hospital.
- Having certain conditions, such as a weakened immune system, certain chronic disorders, an artificial joint or heart valve, and certain heart valve abnormalities, increases the risk.
- At first, people have a high (or sometimes low) body temperature, sometimes with shaking chills and weakness.
- As sepsis worsens, the heart beats rapidly, breathing becomes rapid, people become confused, and blood pressure drops.
- Doctors suspect the diagnosis based on symptoms and confirm it by detecting bacteria in a sample of blood, urine, or other tissue.
- Antibiotics are given immediately, and people with septic shock are given oxygen and fluids and sometimes drugs to increase blood pressure.

Pronunciations

bacteremia
cerebrospinal fluid
cirrhosis
corticosteroid
cytokines

Usually, the body's response to infection is limited to the specific area infected. But in sepsis, the response to infection occurs throughout the body—called a systemic response. This response includes an abnormally high temperature (fever) or low temperature (hypothermia) plus one or more of the following:

- Rapid heart rate
- Rapid breathing rate
- An abnormally high or low number of white blood cells

As sepsis worsens, organs begin to malfunction and blood pressure may decrease. Sepsis is considered severe if organs malfunction. Septic shock is diagnosed when blood pressure remains low despite intensive treatment. In the United States, about 90,000 people, usually those who are hospitalized, die of septic shock each year.

Sepsis occurs when toxins produced by the bacteria cause cells in the body to release substances that trigger inflammation (cytokines). Although cytokines help the immune system fight infection, they can have harmful effects:

- They can cause the blood vessels to widen (dilate), decreasing blood pressure.
- They can cause blood to clot in tiny blood vessels inside organs.

These effects lead to a series of harmful complications:

- Blood flow decreases to vital organs (such as the kidneys, heart, and brain).
- The heart attempts to compensate by working harder, increasing the heart rate and the amount of blood pumped. Eventually, the bacterial toxins and the increased work of pumping weaken the heart. As a result, the heart pumps less blood, and vital organs receive even less blood.
- When tissues do not receive enough blood, they release excess lactic acid (a waste product) into the bloodstream, making the blood more acidic.

All of these effects result in a vicious circle of worsening organ malfunction:

- The kidneys excrete little or no urine, and metabolic waste products (such as urea nitrogen) accumulate in the blood.
- The walls of blood vessels may leak, allowing fluid to escape from the bloodstream into tissues and cause swelling.
- Lung function worsens because leaking blood vessels in the lungs cause fluid to accumulate, making breathing difficult.

As blood clots continue to form, the proteins in blood that make up clots (clotting factors) are used up. Then, excessive bleeding may occur.

Causes

Most often, sepsis is caused by infection with certain kinds of bacteria, usually acquired in a hospital. Rarely, fungi, such as *Candida*, cause sepsis. Infections that can lead to sepsis begin most commonly in the lungs, abdomen, or urinary tract. If the initial infection involves a collection of pus (abscess), the risk of bacteremia and sepsis is increased. In most people, these infections do not lead to sepsis. However, sometimes bacteria spread into the bloodstream (a condition called bacteremia). Sepsis may then develop. Occasionally, sepsis is triggered by toxins released by bacteria, rather than from bacteria entering the bloodstream (bacteremia).

Risk Factors

The risk of sepsis is increased in people with conditions that reduce their ability to fight serious infections. These conditions include the following:

- Being a newborn
- Being over 35
- Being pregnant
- Having certain chronic disorders such as diabetes or cirrhosis
- Having a weakened immune system—due to use of drugs that suppress the immune system (immunosuppressants, such as chemotherapy drugs) or corticosteroids, or due to certain disorders (such as cancer, AIDS, and immune disorders)

The risk is also increased in people who are more likely to have bacteria enter their bloodstream. Such people include those who have a medical device inserted into the body (such as a catheter inserted into a vein or the urinary tract, drainage tubes, or breathing tubes). When medical devices are inserted, they can move bacteria into the body. Bacteria may also collect on the surface of such devices, making infection and sepsis more likely. The longer the device is left in place, the greater the risk.

Other conditions also increase the risk of sepsis:

- Injecting recreational drugs: The drugs and needles used are rarely sterile. Each injection may cause bacteremia to varying degrees. People who use these drugs are also at risk of disorders that can weaken the immune system (such as AIDS).
- Having an artificial (prosthetic) joint or heart valve or certain heart valve abnormalities: Bacteria tend to lodge and collect on these structures. The bacteria may continuously or periodically be released into the bloodstream.
- Being treated with antibiotics for other infections: Some bacteria that cause infections and sepsis are resistant to antibiotics. Antibiotics do not eradicate the resistant bacteria. Thus, if an infection persists in people who are taking antibiotics, it is more likely to be

caused bacteria that are resistant to antibiotics and that can cause sepsis.

Symptoms

Most people have a fever, but some have a low body temperature. People may have shaking chills and feel weak. Other symptoms may also be present depending on the type and location of the initial infection. Breathing, heart rate, or both may be rapid.

As sepsis worsens, people become confused and less alert. The skin becomes warm and flushed. The pulse is rapid and pounding, and people breathe rapidly. People urinate less often and in smaller amounts, and blood pressure decreases. Later, body temperature often falls below normal, and breathing becomes very difficult. The skin may become cool and mottled or blue because blood flow is reduced. Reduced blood flow may cause tissue, including tissue in vital organs (such as the intestine), to die, resulting in gangrene.

When septic shock develops, blood pressure is low, even despite treatment.

With treatment, the risk of death is about 15% for people with sepsis and 40% or more for people with septic shock.

Diagnosis

Doctors usually suspect sepsis when a person who has an infection suddenly develops a very high or low temperature, a rapid heart or breathing rate, or low blood pressure. To confirm the diagnosis, doctors look for bacteria in the bloodstream (bacteremia), evidence of another infection that could be causing sepsis, and an abnormal number of white blood cells in a blood sample.

Samples of blood are taken to try to grow the bacteria in the laboratory (blood culture)—a process that takes 1 to 3 days. However, if people have been taking antibiotics for their initial infection, bacteria may be present but not grow in the culture. Sometimes catheters are removed from the body, and the tips are cut off and sent for culture. Finding bacteria in a catheter that had contact with the blood indicates that bacteria are probably in the bloodstream.

To check for other infections that may cause sepsis, doctors take samples of fluids or tissue, such as urine, cerebrospinal fluid, tissue from wounds, or sputum coughed up from the lungs. These samples are cultured and checked for bacteria. Imaging tests may also be done.

Other tests are done to look for signs of organ malfunction and other complications of sepsis. They may include the following:

- Blood tests to measure levels of lactic acid, other metabolic waste products, and platelets (cells that help the blood clot). Levels of waste products may be high, and the level of platelets may be low.
- Blood tests or a sensor placed on a finger (pulse oximetry) to measure oxygen levels and thus evaluate how well the lungs and blood vessels are functioning
- Electrocardiography (ECG) to look for abnormalities in heart rhythm and thus determine whether the blood supply to the heart is adequate
- Other tests to determine whether shock results from sepsis or another problem

Treatment

Sepsis and septic shock must be treated immediately with antibiotics—even before test results confirm the diagnosis. A delay in antibiotic treatment greatly decreases the chances of survival. People with symptoms of septic shock are immediately admitted to an intensive care unit for treatment.

When choosing the initial antibiotics, doctors consider which bacteria are most likely to be present, which depends on where the infection started. Often, two or three antibiotics are given together to increase the chances of killing the bacteria, particularly when the source of

the bacteria is unknown. Later, when the test results are available, doctors can substitute the antibiotic that is most effective against the specific bacteria causing the infection.

If present, abscesses are drained, and catheters or other medical devices that may have started the infection are removed. Surgery may be done to remove dead tissue.

Severe sepsis or septic shock can be treated with drotrecogin alfa (activated protein C). This drug is an artificially produced human protein that prevents inflammation and blood clotting. It may reduce the risk of death due to severe sepsis or septic shock.

People with septic shock are also given large amounts of fluid intravenously to increase the amount of fluid in the bloodstream and thus increase blood pressure. Drugs, such as dopamine or norepinephrine (which cause blood vessels to narrow), may be needed to increase blood flow to the brain, heart, and other organs. Oxygen is given through a mask, through nasal prongs, or, if a breathing (endotracheal) tube has been inserted, through that tube. If needed, a mechanical ventilator is used to help with breathing.

Last full review/revision September 2008 by Lowell S. Young, MD

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Septic Shock

WHAT YOU SHOULD KNOW

Septic shock is a life-threatening reaction to a severe infection. During septic shock, the body tissues and organs do not get enough blood and oxygen.

Causes

The problem may start with a small infection that overwhelms the body's defenses and spreads. In some severe infections, the germs make harmful toxins that can cause fluid to leak from blood vessels out into the tissues. The toxins may also prevent the heart from beating strongly enough. Together, these reactions lower blood pressure. If blood pressure gets too low, the body and its organs become deprived of oxygen. The body tries to help itself, but without enough oxygen, it makes too much of certain waste products. These extra wastes can do additional harm.

Signs/Symptoms

If you develop a small infection in one part of the body, the symptoms may include redness, swelling, and tenderness. Signs that the infection has spread throughout the body are fever, fast breathing, dizziness, and fast heart rate.

Care

Septic shock is an emergency that requires treatment in the hospital. While there, you will get medicine to treat your infection, plus IV fluids, oxygen, and possibly medicine to raise the blood pressure.

Risks

Without treatment, septic shock is usually a killer. The sooner you receive treatment, the better your chances of recovery.

IF YOU'RE HEADING FOR THE HOSPITAL...

What to Expect While You're There

You may encounter the following procedures and equipment during your stay.

- **Taking Vital Signs:** These include your temperature, blood pressure, pulse (counting your heartbeats), and respirations (counting your breaths). A stethoscope is used to listen to your heart and lungs. Your blood pressure is taken by wrapping a cuff around your arm.
- **Oxygen:** Your body may need extra oxygen at this time. It is given either by a mask or nasal prongs. Tell the doctor if the oxygen is drying out your nose or if the nasal prongs are bothersome.
- **Pulse Oximeter:** While getting oxygen, you may be hooked up to a pulse oximeter (ox-IM-ih-ter). It is placed on an ear, finger, or toe and is connected to a machine that measures the oxygen in the blood.
- **IV:** A tube placed in the vein for giving

medicine or liquids. It will be capped or have tubing connected to it.

- **Blood:** Usually taken from a vein in a hand or from the bend in an elbow. Tests will be done on the blood.
- **Blood Gases:** For this test blood is taken from an artery in a wrist, elbow, or groin. It is tested for oxygen.
- **ET Tube:** This tube is passed through either the mouth or nose and down into the windpipe. It is often hooked up to a breathing machine. With the tube in place, you will not be able to talk.
- **Ventilator** (VENT-ih-lay-ter): A special machine used to help with breathing. This will help you save energy that your body needs to get better.
- **Chest X-ray:** This picture of the lungs and heart shows how they are handling the illness.
- **ECG:** Also called a heart monitor, an electrocardiograph (e-LEK-tro-CAR-dee-o-graf), or EKG. The patches on your chest are hooked up to a TV-type screen or a small portable box (telemetry unit). This screen shows a tracing of each heartbeat. The heart will be watched for signs of injury or damage that could be related to the illness.
- **Foley Catheter:** This tube drains urine from the bladder until you can urinate on your own.
- **Coughing and Deep Breathing:** It is important to do this often because it helps keep the lungs from getting infected.
  - To ease the pain during coughing and deep breathing, a 6 inch elastic bandage can be loosely wrapped around the rib cage.
  - Holding a pillow tightly against the chest when coughing can help ease any pain.
- **Medicines:**
  - **Antibiotics** will be used to fight the infection. They may be given by IV, in a shot, or by mouth.
  - **Blood pressure medicine** may be given through your IV in order to bring your blood pressure up to normal. Once the infection is under control and the body can take care of itself, this medicine can be stopped.
- **Heart Tubes/Wires:** You may be attached to many different tubes and wires. Some may enter your body under the collarbone or in the groin and be threaded into the heart. They are attached to monitors that measure the heart while it's working. These readings help the doctor guide your treatment.

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FACTS ABOUT SEPSIS AND SEVERE SEPSIS

DEFINITION

Sepsis is a syndrome characterized by an overwhelming systemic (whole body) response to infection, which can rapidly lead to loss of limbs, organ dysfunction, and ultimately death. The body's normal reaction to infection goes into overdrive, setting off a cascade of events that can lead to widespread inflammation and clotting. Severe sepsis is sepsis with associated acute organ dysfunction.

SYMPTOMS

Symptoms of sepsis include reduced mental alertness, confusion, shaking, chills, fever, nausea, vomiting, and diarrhea in the presence of infection. Patients with sepsis rapidly get worse.

PREVALENCE

Every year, severe sepsis strikes an estimated 750,000 people in the United States, 215,000 of whom die. The incidence of severe sepsis is expected to rise to 1 million by the end of the decade as the population ages. Sepsis is the leading cause of death in the non-coronary ICU. Severe sepsis takes more lives than breast, colon/rectal, pancreatic, and prostate cancer combined. In fact, a recent report from the CDC ranked septicemia, a form of sepsis, as the tenth leading killer in the United States. And more people die from sepsis each year than from septicemia.

CAUSES

Sepsis can be triggered by a bacterial, viral, parasitic, or fungal infection, often the result of events such as trauma, surgery, and burns, or illnesses such as cancer and pneumonia. Researchers are unsure why some patients progress to sepsis while others do not.

-more-

COMMON TERMS *Sepsis* -- characterized by a generalized inflammatory response, which can include abnormal clotting and bleeding, in the presence of infection.

Septicemia -- sepsis that begins with a blood-borne infection.

Severe sepsis -- sepsis with associated acute organ dysfunction.

Septic shock -- severe sepsis in which the cardiovascular system begins to fail, the blood pressure drops, and vital organs are deprived of adequate blood supply.

PEOPLE AT RISK Sepsis can strike anyone at any age, although the elderly, hospital patients, and people with pre-existing conditions (such as trauma, burns, surgery, or cancer) may be at greater risk.

COSTS The estimated costs associated with the treatment of patients with severe sepsis was approximately $17 billion annually in the U.S. in 1995.

TREATMENT Current treatment for patients with severe sepsis consists of supportive care, including antibiotics, mechanical ventilation, or kidney dialysis, but none of these measures specifically treats severe sepsis. Many companies have tried and failed to develop an effective treatment.

# # #

Hypercalcemia

Definition

By Mayo Clinic staff

Parathyroid glands

Hypercalcemia is a condition in which the calcium level in your blood is above normal. You need calcium for bone formation. It also plays an important role in contracting muscles, releasing hormones, and ensuring that your nerves and brain function properly. High calcium levels, however, can interfere with these processes.

The main cause of hypercalcemia is overactivity in one or more of your parathyroid glands, which regulate calcium. Post-menopausal women are most likely to develop hypercalcemia caused by overactive parathyroid glands. Other causes of hypercalcemia include cancer, certain other medical disorders, some medications, and excessive use of calcium and vitamin D supplements.

Signs and symptoms of hypercalcemia may range from nonexistent to severe. Treatment depends on the underlying cause.

Symptoms

References

May 29, 2009 DS00976

Chronic kidney disease

U.S. National Library of Medicine
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Overview

Chronic kidney disease is the slow loss of kidney function over time. The main function of the kidneys is to remove wastes and excess water from the body.

Symptoms

The early symptoms of chronic kidney disease often occur with other illnesses, as well. These symptoms may be the only signs of kidney disease until the condition is more advanced.

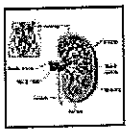
Symptoms may include:

- General ill feeling and fatigue
- Generalized itching (pruritus) and dry skin
- Headaches
- Weight loss without trying to lose weight
- Appetite loss
- Nausea

Other symptoms that may develop, especially when kidney function has worsened:

- Abnormally dark or light skin
- Bone pain
- Brain and nervous system symptoms
  - Drowsiness and confusion
  - Problems concentrating or thinking
  - Numbness in the hands, feet, or other areas
  - Muscle twitching or cramps
- Breath odor
- Easy bruising, bleeding, or blood in the stool
- Excessive thirst
- Frequent hiccups
- Low level of sexual interest and impotence
- Menstrual periods stop

Illustrations


Kidney anatomy


Kidney - blood and urine flow


White nail syndrome

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News From The American Journal Of Pathology
Medical News Today (press release) - 1 week ago
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Tips from the American Journal of Pathology
EurekAlert (press release) - 1 week ago
Chronic kidney disease and end stage renal disease affect more than 2 out of every 1000 people in the United States. Diabetes and high blood pressure are ...

Overview of chronic kidney failure
Helium - 1 week ago
For example, in **chronic renal failure** and acute renal failure there is retention in the blood of the compounds urea and creatinine. ...

Google Scholar More »

Clinical epidemiology of cardiovascular disease in Chronic Kidney Disease
RN Foley - Journal of Renal Care, 2010 - www3.interscience.wiley.com

Symptoms
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Complications

(amenorrhea)
- Sleep problems, such as insomnia, restless leg syndrome, and obstructive sleep apnea
- Swelling of the feet and hands (edema)
- Vomiting, typically in the morning

Treatment

Controlling blood pressure is the key to delaying further kidney damage.

- Angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs) are used most often.
- The goal is to keep blood pressure at or below 130/80 mmHg

Other tips for protecting the kdineys and preventing heart disease and stroke:

- Do not smoke.
- Eat meals that are low in fat and cholesterol
- Get regular exercise (talk to your doctor or nurse before starting).
- Take drugs to lower your cholesterol, if necessary.
- Keep your blood sugar under control.

Always talk to your kidney doctor before taking any over-the-counter medicine, vitamin, or herbal supplement. Make sure all of the doctors you visit know you have chronic kidney disease.

Other treatments may include:

- Special medicines called phosphate binders, to help prevent phosphorous levels from becoming too high
- Treatment for anemia, such as extra iron in the diet, iron pills, special shots of a medicine called erythropoietin, and blood transfusions
- Extra calcium and vitamin D (always talk to your doctor before taking)

You may need to make changes in your

Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization
AS Go,GM Chertow,D Fan,CE McCulloch,C Hsu - New England Journal of Medicine, 2004 - content.nejm.org

Accumulation of an endogenous inhibitor of nitric oxide synthesis in chronic renal failure
A Leone,S Moncada,P Vallance,A Calver,J Collier - The Lancet, 1992 - linkinghub.elsevier.com

Search Trends

People who searched for **Chronic kidney disease** also searched for:

Conditions

1. renal failure
2. kidney disease
3. diabetes mellitus
4. high blood pressure
5. acute kidney failure
6. anemia
7. chronic disease
8. end-stage renal disease

Symptoms

1. fever
2. cough
3. headache
4. weight loss
5. constipation
6. frequent urination
7. fatigue
8. inflammation

Drugs

1. regular insulin
2. furosemide
3. epoetin alfa
4. acetaminophen
5. urea
6. aspirin
7. heparin
8. thyroxine

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diet. See: Diet for chronic kidney disease for more details.

- You may need to limit fluids.
- Your health care provider may recommend a low-protein diet.
- You may have to restrict salt, potassium, phosphorous, and other electrolytes.
- It is important to get enough calories when you are losing weight.

Different treatments are available for problems with sleep or restless leg syndrome.

Everyone with chronic kidney disease should be up-to-date on important vaccinations, including:

- Pneumococcal polysaccharide vaccine (PPV)
- Influenza vaccine
- H1N1 (swine flu) vaccine
- Hepatitis B vaccine
- Hepatitis A vaccine

When loss of kidney function becomes more severe, you will need to prepare for dialysis or a kidney transplant.

- When you start dialysis depends on different factors, including your lab test results, severity of symptoms, and readiness.
- You should begin to prepare for dialysis before it is absolutely necessary. The preparation includes learning about dialysis and the types of dialysis therapies, and placement of a dialysis access.
- Even those who are candidates for a kidney transplant will need dialysis while waiting for a kidney to become available.

Causes

Chronic kidney disease (CKD) slowly gets worse over time. In the early stages, there may be no symptoms. The loss of function usually takes months or years to occur. It may be so slow that symptoms

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do not occur until kidney function is less than one-tenth of normal.

The final stage of chronic kidney disease is called end-stage renal disease (ESRD). The kidneys no longer function and the patient needs dialysis or a kidney transplant.

Chronic kidney disease and ESRD affect more than 2 out of every 1,000 people in the United States.

Diabetes and high blood pressure are the two most common causes and account for most cases.

Many other diseases and conditions can damage the kidneys, including:

- Problems with the arteries leading to or inside the kidneys
- Birth defects of the kidneys (such as polycystic kidney disease)
- Some pain medications and other drugs
- Certain toxic chemicals
- Autoimmune disorders (such as systemic lupus erythematosus and scleroderma)
- Injury or trauma
- Glomerulonephritis
- Kidney stones and infection
- Reflux nephropathy (in which the kidneys are damaged by the backward flow of urine into the kidneys)
- Other kidney diseases

Chronic kidney disease leads to a buildup of fluid and waste products in the body. This condition affects most body systems and functions, including red blood cell production, blood pressure control, and vitamin D and bone health.

Tests & diagnosis

High blood pressure is almost always present during all stages of chronic kidney disease. A neurologic examination may show signs of nerve damage. The health care provider may hear abnormal

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heart or lung sounds with a stethoscope.

A urinalysis may show protein or other changes. These changes may appear 6 months to 10 or more years before symptoms appear.

Tests that check how well the kidneys are working include:

- Creatinine levels
- BUN
- Creatinine clearance

Chronic kidney disease changes the results of several other tests. Every patient needs to have the following checked regularly, as often as every 2 - 3 months when kidney disease gets worse:

- Potassium
- Sodium
- Albumin
- Phosphorous
- Calcium
- Cholesterol
- Magnesium
- Complete blood count (CBC)
- Electrolytes

Causes of chronic kidney disease may be seen on:

- Abdominal CT scan
- Abdominal MRI
- Abdominal ultrasound
- Renal scan

This disease may also change the results of the following tests:

- Erythropoietin
- PTH
- Bone density test

Prognosis

Many people are not diagnosed with chronic kidney disease until they have lost much of their kidney function.

There is no cure for chronic kidney disease. Untreated, it usually progresses to end-stage renal disease. Lifelong treatment may control the symptoms of chronic kidney disease.

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Prevention

Treating the condition that is causing the problem may help prevent or delay chronic kidney disease. People who have diabetes should control their blood sugar and blood pressure levels and should not smoke.

Complications

- Anemia
- Bleeding from the stomach or intestines
- Bone, joint, and muscle pain
- Changes in blood sugar
- Damage to nerves of the legs and arms (peripheral neuropathy)
- Dementia
- Fluid buildup around the lungs (pleural effusion)
- Heart and blood vessel complications
  - Congestive heart failure
  - Coronary artery disease
  - High blood pressure
  - Pericarditis
  - Stroke
- High phosphorous levels
- High potassium levels
- Hyperparathyroidism
- Increased risk of infections
- Liver damage or failure
- Malnutrition
- Miscarriages and infertility
- Seizures
- Weakening of the bones and increased risk of fractures

Article Link: http://www.webmd.com/a-to-z-guides/understanding-anemia-basics

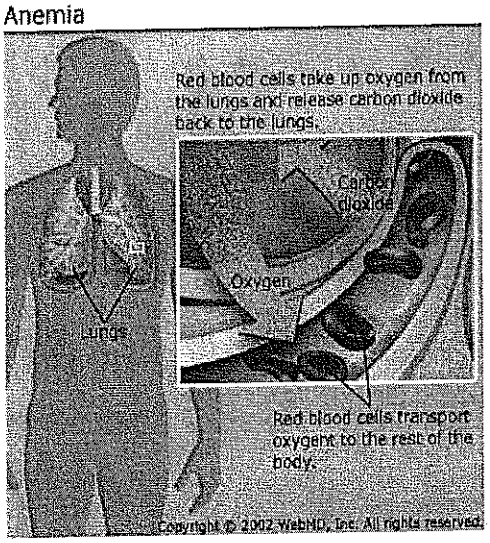
Information and Resources

Understanding Anemia - the Basics

What Is Anemia?

Anemia is a condition that develops when your blood lacks enough healthy red blood cells. These cells are the main transporters of oxygen to organs. If red blood cells are also deficient in hemoglobin, then your body isn't getting enough oxygen. Symptoms of anemia -- like fatigue -- occur because organs aren't getting enough oxygen.

Anemia



Anemia is the most common blood condition in the U.S. It affects about 3.5 million Americans. Women and people with chronic diseases are at increased risk of anemia. Important factors to remember are:

- Certain forms of anemia are hereditary and infants may be affected from the time of birth.
- Women in the childbearing years are particularly susceptible to a form of anemia called iron-deficiency anemia because of the blood loss from menstruation and the increased blood supply demands during pregnancy.
- Seniors also may have a greater risk of developing anemia because of poor diet and other medical conditions.

There are many types of anemia. All are very different in their causes and treatments. Iron-deficiency anemia, the most common type, is very treatable with diet changes and iron supplements. Some forms of anemia -- like the anemia that develops during pregnancy -- are even considered normal. However, some types of anemia may present lifelong health problems.

What Causes Anemia?

There are more than 400 types of anemia, which are divided into 3 groupings:

- Anemia caused by blood loss
- Anemia caused by decreased or faulty red blood cell production
- Anemia caused by destruction of red blood cells

See more about : Anemia Iron Deficiency Hemoglobin Red Blood Cells

Anemia Caused by Blood Loss

Red blood cells can be lost through bleeding, which can occur slowly over a long period of time, and can often go undetected. This kind of chronic bleeding commonly results from the following:

- Gastrointestinal conditions such as ulcers, hemorrhoids, gastritis (inflammation of the stomach) and cancer
- Use of nonsteroidal anti-inflammatory drugs (NSAIDS) such as aspirin or Motrin
- Menstruation and childbirth in women, especially if menstrual bleeding is excessive and if there are multiple pregnancies

Anemia Caused by Decreased or Faulty Red Blood Cell Production

The body may produce too few blood cells or the blood cells may not work properly. In either case, anemia can result. Red blood cells may be faulty or decreased due to abnormal red blood cells or the a lack of minerals and vitamins needed for red blood cells to work properly. Conditions associated with these causes of anemia include the following:

- Sickle cell anemia
- Iron deficiency anemia
- Vitamin deficiency
- Bone marrow and stem cell problems
- Other health conditions

Sickle cell anemia is an inherited disorder that affects African-Americans. Red blood cells become crescent-shaped because of a genetic defect. They break down rapidly, so oxygen does not get to the body's organs, causing anemia. The crescent-shaped red blood cells also get stuck in tiny blood vessels, causing pain.

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WebMD Medical Reference

Congestive Heart Failure

Heart Disease (Coronary Artery Disease) Slideshow

Lowering Cholesterol and Avoiding Heart Disease Slideshow

Heartburn Slideshow

Medical Author: Daniel Kulick, MD, FACC, FSCAI
Medical Editor: William C. Shiel, Jr., MD, FACP, FACR

What is congestive heart failure?

Congestive heart failure (CHF) is a condition in which the heart's function as a pump is inadequate to deliver oxygen rich blood to the body. Congestive heart failure can be caused by:

1. diseases that weaken the heart muscle,
2. diseases that cause stiffening of the heart muscles, or
3. diseases that increase oxygen demand by the body tissue beyond the capability of the heart to deliver adequate oxygen-rich blood.

The heart has two atria (right atrium and left atrium) that make up the upper chambers of the heart, and two ventricles (left ventricle and right ventricle) that make up the lower chambers of the heart. The ventricles are muscular chambers that pump blood when the muscles contract. The

Congestive Heart Failure

Congestive Heart Failure Symptoms and Signs

The symptoms of congestive heart failure vary among individuals according to the particular organ systems involved and depending on the degree to which the rest of the body has "compensated" for the heart muscle weakness.

The early symptoms are often shortness of breath, cough, or a feeling of not being able to get a deep breath.

In addition, the three major symptoms of congestive heart failure are:

1. exercise intolerance (a person may be unable to tolerate exercise or even mild physical exertion that he or she may have been able to do in the past);
2. shortness of breath (you may have difficulty breathing (dyspnea), especially when active, or even at rest); and
3. fluid retention and swelling (edema in the legs, feet, and ankles).

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Congestive Heart Failure

Electrolytes »

What are electrolytes?

Chemically, electrolytes are substances that become ions in solution and acquire the capacity to conduct electricity. Electrolytes are present in the human body, and the balance of the electrolytes in our bodies is essential for normal function of our cells and our organs.

Common electrolytes that are measured by doctors with blood testing include sodium, potassium, chloride, and

contraction of the ventricle muscles is called systole.

Read more about the signs and symptoms of congestive heart failure »

Many diseases can impair the pumping action of the ventricles. For example, the muscles of the ventricles can be weakened by heart attacks or infections (myocarditis). The diminished pumping ability of the ventricles due to muscle weakening is called systolic dysfunction. After each ventricular contraction (systole) the ventricle muscles need to relax to allow blood from the atria to fill the ventricles. This relaxation of the ventricles is called diastole.

Diseases such as hemochromatosis (iron overload) or amyloidosis can cause stiffening of the heart muscle and impair the ventricles' capacity to relax and fill; this is referred to as diastolic dysfunction. The most common cause of this is longstanding high blood pressure resulting in a thickened (hypertrophied) heart. Additionally, in some patients, although the pumping action and filling capacity of the heart may be normal, abnormally high oxygen demand by the body's tissues (for example, with hyperthyroidism or anemia) may make it difficult for the heart to supply an adequate blood flow (called high output heart failure).

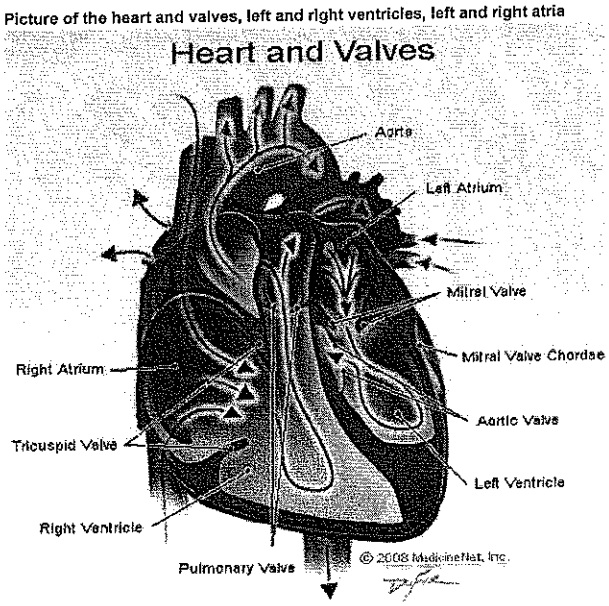
In some individuals one or more of these factors can be present to cause congestive heart failure. The remainder of this article will focus primarily on congestive heart failure that is due to heart muscle weakness, systolic dysfunction.

Congestive heart failure can affect many organs of the body. For example:

- The weakened heart muscles may not be able to supply enough blood to the kidneys, which then begin to lose their normal ability to excrete salt (sodium) and water. This diminished kidney function can cause the body to retain more fluid.
- The lungs may become congested with fluid (pulmonary edema) and the person's ability to exercise is decreased.
- Fluid may likewise accumulate in the liver, thereby impairing its ability to rid the body of toxins and produce essential proteins.
- The intestines may become less efficient in absorbing nutrients and medicines.
- Fluid also may accumulate in the extremities, resulting in edema (swelling) of the ankles and feet.

Eventually, untreated, worsening congestive heart failure will affect virtually every organ in the body.

Picture of the heart and valves, left and right ventricles, left and right atria



What causes congestive heart failure?

Many disease processes can impair the pumping efficiency of the heart to cause congestive heart failure. In the United States, the most common causes of congestive heart failure are:

bicarbonate. The functions and normal range values for these electrolytes are described below.

Sodium

Sodium is the major positive ion (cation) in fluid outside of cells. The chemical notation for sodium is Na+. When combined with chloride, the resulting substance is table salt. Excess sodium (such as that obtained from dietary sources) is excreted in the urine. Sodium regulates the total amount of water in the body and the transmission of sodium into and out of individual cells also plays a role in critical body funct...

Read the Electrolytes article »

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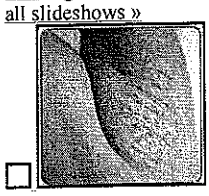
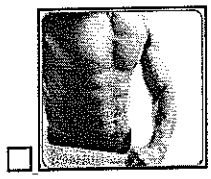


Scales, Plaques & Eruptions



- coronary artery disease,
- high blood pressure (hypertension),
- longstanding alcohol abuse, and
- disorders of the heart valves.

Less common causes include viral infections of the stiffening of the heart muscle, thyroid disorders, disorders of the heart rhythm, and many others.

It should also be noted that in patients with underlying heart disease, taking certain medications can lead to the development or worsening of congestive heart failure. This is especially true for those drugs that can cause sodium retention or affect the power of the heart muscle. Examples of such medications are the commonly used nonsteroidal antiinflammatory drugs (NSAIDs), which include ibuprofen (Motrin and others) and naproxen (Aleve and others) as well as certain steroids, some medication for diabetes (such as rosiglitazone [Avandia] or pioglitazone [Actos]), and some calcium channel blockers.

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Congestive Heart Failure Index | Glossary | Find a Local Doctor

Next: What are the symptoms of congestive heart failure (CHF)? »

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Mailed + Emailed
10/22/10

JACKSONVILLE SHERIFF'S OFFICE
501 E. BAY STREET
JACKSONVILLE, FLORIDA 32202-2975

EMPLOYEE COMPLAINT FORM

Nature of Incident/Event	I.A. # (*Administrative Use Only*)
Falsifying Police Reports/Medical Records	

SHERIFF'S OFFICE EMPLOYEE INFORMATION

Employee's Name and ID# (if known): Mary Leach, Patricia M. Woodbary, Yvonne V. Walker, Jhasmine P. Perez, Lung, Freddie Sanders #63431, K.E. Norton #7421, Jessie C. Giles, MD

Race	Sex	Height	Weight	Approximate Age
VAR		10	U S	

Other Identifying Characteristics: N/A

Vehicle or Tag #	Vehicle Model	In Uniform
—	N/A	—

Employee Vehicle Description (Marked/Unmarked, Color): N/A

Location of Incident/Event: various

Day and Date of Incident/Event	Time
various	various

Today's Date and Time: October 22, 2010

CITIZEN INFORMATION

Citizen's Name (First MI Last): Kerrie Merrifield on behalf of William Merrifield (deceased)

Home Address	Apt#
31452 Paseo De La Plaza	

City	State	Zip Code
Laguna Niguel	CA	92677

Race	Sex	D.O.B.
White	Female	May 9, 1968

Home #	Business #	Cell #
N/A	N/A	949-280-9306

Name of Witness (First MI Last): same as above

Witness Address	City, State	Zip Code

Witness Phone Number(s):

EXPLANATION OF EVENT: (Also list on the back any additional information, employees, witnesses, etc)

Please see attached.

F.S.S. 837.06: Whoever knowingly makes a false statement in writing with the intent to mislead a public servant in the performance of his or her official duty shall be guilty of a misdemeanor of the second degree.

Signature of Citizen: Kerrie Merrifield PR for William Merrifield
All information is true and correct to the best of my knowledge.

Employee Accepting Form	Assignment	Date and Time

Internal Affairs Complaint

Inbox x

Smith, Lolita M. Kerrie Merrifield, I have attached a copy of our agenc Oct 19 (6 days ago)

Smith, Lolita M. to me show details 8:51 AM (40 minutes ago) Reply

Kerrie Merrifield,

This email serves as my acknowledgement that I have received your complaint. After reviewing the details of your complaint, Lt. Eason will send you a written response via the mail. The letter will contain information regarding who has been assigned to investigate your complaint. I am so sorry that you had a negative experience with a member of JSO. If you have any additional information or concerns, please feel free to contact me directly via email or at 904 630-2207.

Have a wonderful day,

Sgt. Smith

From: Kerrie Merrifield [mailto:kerriemerrifield@Gmail.com]
Sent: Friday, October 22, 2010 9:34 PM
To: Smith, Lolita M.
Subject: Re: Internal Affairs Complaint

Dear Sergeant Smith,

I am attaching a copy of the completed form along with the back up information. please note that page 27 should be placed after page 7. I have also mailed you a hard copy today as well. Please do not hesitate to contact me with any questions. I look forward to hearing from you soon.

Kind regards,

Kerrie Merrifield

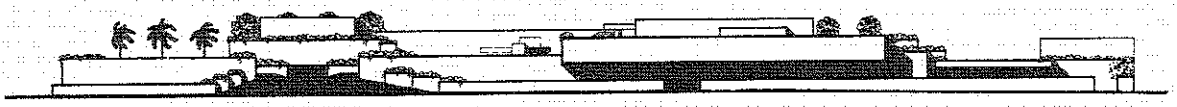
Reply Forward

Map this
501 East Bay Street
Jacksonville, Florida 32202

#72

OFFICE OF THE SHERIFF

CONSOLIDATED CITY OF JACKSONVILLE



501 EAST BAY STREET • JACKSONVILLE, FLORIDA 32202-2975

John H. Rutherford
Sheriff

RE: Internal Affairs Case No: 10-00683

Kerrie Merrifield
31452 Pasco De La Playa
Laguna Niguel, CA 92677

Dear Ms. Merrifield

As Medical Director of the Division of Health Services for the Department of Corrections at the Jacksonville Sheriff's Office I appreciate bringing your concerns to our attention. Your observations are of great importance to us and as part of our Continuous Quality Improvement process we welcome and encourage the community to communicate with us to ensure that services delivered to our patients are commensurate with community standards, national guidelines and accreditation requirements.

I have reviewed in detail your concerns and I clearly understand the perception that you have about the care of your father. I will address your concerns one by one as you have listed them. I hope that at the end of this review you will find my response satisfactory with the assurance that your father had access to adequate medical care and that the corresponding documentation is honest and thorough. Nevertheless we have taken some actions in our continuous education for our health care workers making emphasis in proper encounter documentation and, most importantly, the consistency of such documentation.

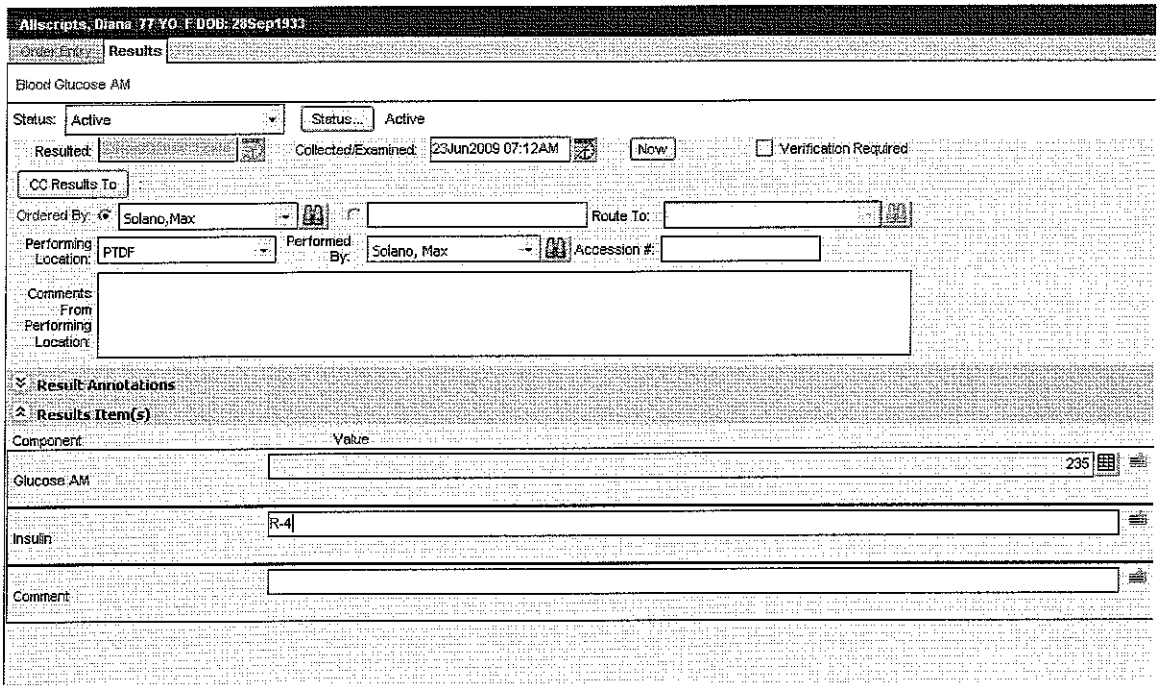
Before responding to your observations, please allow me to give you an overview of our internal processes that I am sure will clarify some of your concerns.

The Diabetes Mellitus Flow Sheet is the official Medication Administration Record (MAR). Orders and clinical decisions are made based on this information. In other words, when the patient is called to the clinic for his blood sugar testing, the results are recorded on the MAR as well as the instructions for Insulin. These are followed by the health care personnel performing the test. Any changes on insulin requirements or frequency of blood sugar testing are done after review of this MAR.

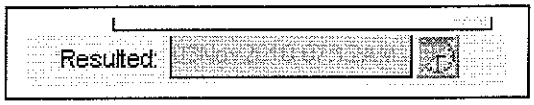
BB (Before Breakfast) readings are scheduled every day around 4:00 A.M.; BL (Before Lunch) readings are scheduled every day between 10:00 AM and 11:00 AM coinciding with lunch time; BD (Before Dinner) readings are scheduled every day around 4:00 PM. Other readings are scheduled as needed.

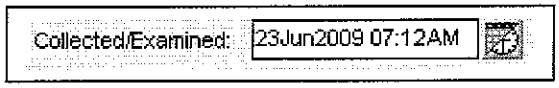
The purpose of documenting the results of the blood sugar testing in the E.H.R. is to have a general overview at the chronic care visits of the patient's evolution. Every patient with a chronic condition such as diabetes is automatically scheduled for periodic review of his/her condition. Thanks to the documentation of the blood sugar tests the clinician has a general idea of blood sugar variations and is able to educate the patient on Diabetes care. However, any medical decision on medication change is based on variations of the Hemoglobin A1c and the results documented in the MAR.

The health care personnel made every effort to document the results of the blood sugar tests and the corresponding action taken (i.e. how much insulin was given) in the E.H.R. (Electronic Health Record – Allscripts Enterprise V11). However, this goal is not accomplished one hundred percent of the times and unfortunately our blood glucose testing devices are not connected to the E.H.R, so the recording process still is manual and therefore operator dependent. Please notice two things in the following image:



In the upper left corner you see two fields: Resulted and Collected/Examined.

The first one  (the Resulted field) is grayed out, and not modifiable by the user. The E.H.R. automatically records the date that the Result Entry window is opened. In this example the actual date when I opened this window was November 3rd.

The second one open to the operator  (Collected/Examined) is the field that is to manually enter the date and time that the actual specimen was collected. In this example I was able to change the date back to 2009.

Another thing that I want to bring to your attention is the button "Now" next to the Collected/Examined field. If the test is being resulted at the time of collection this button makes the documentation process faster by recording the current time upon clicking on it. However, the resulting in the E.H.R. usually happens after the actual time of collection and the operator is supposed to change the date/time to reflect the actual collection time.

The document referred in your letter as "Diagnostics Details Sheet" is a generic document used by the E.H.R. to print results from different activities including vital signs and laboratory tests. There is no expectation of agreement in date and time since the activities described are independent and not intended as back up as you mentioned.

Now my response the items that you listed:

1. The blood sugar testing results were documented in the official MAR as you stated. However, the health care workers did not enter the results in the E.H.R. As stated before this direction is not followed a hundred percent of the times. The back up documents you referred to do not correspond to the blood sugar testing activities but they represent documentation that vital signs were taken at different times by different staff members. Please see **Procedure Reinforcement Education** number one (PRE 1) below.

2. The MAR is the official record for the blood glucose testing results and the action taken were based on the MAR records. Clearly the health care worker entry in the E.H.R. is inaccurate. Please see PRE 2 below.

3. The Resulted field was 8:46 P.M. This is the time that the E.H.R. resulting window opened as explained above. This field is non-modifiable and is the electronic stamp of the resulting activity. In the Collected field was 8:47 P.M. The operator clicked the NOW button recording the current time instead of entering the actual time that the blood sugar test was performed. Please see PRE – 3 below.

4. Your observation here is similar to Item 1. The MAR evidences that the actual test took place.

5. Your observation here is similar to Item 3. The Result window was opened at 11:10 P.M. meaning the E.H.R. is stamping the activity at this time and it's not modifiable by the user. In the Collected field the operator clicked the NOW button recording any time after the resulting window was opened. The operator should have changed the collected time to the actual collection time. Please see PRE – 3 below.

6. The MAR shows 3 readings, the first is 60 mg/dL L-20 given, patient called back at 10:30 BS 163 (This reading was not recorded in the E.H.R.). The BD was performed around 4:30 PM (usual time for BD as stated before). The operator entered the results in the E.H.R. at 9:46 PM, however, selected AM instead of PM in the collected field. See PRE - 2 below.

7. The MAR shows 4 readings. 80 mg/dL BB, 56 mg/dL mid morning, 62 mg/dL at 13:10, repeat reading at 13:15 of 75 mg/dL and 176 mg/dL BD. The repeated reading at 13:15 was not documented in the MAR but it was postdated in the E.H.R. The resulted time 6/26/09 reflects again the time that the resulting window was opened. The collected time is the time of the actual testing. See PRE - 4 and 5 below.

8. See item 7 above. The blood sugar of 75 mg/dl was not documented in the MAR and the difference between the resulted time and collected time as explained above.

9. The image encounters not necessarily correlate when the patient is seen. The encounter is stamped automatically by the computer at the time that the operator opens the E.H.R. to document any action taken on the patient. The documentation certainly can be entered at a later time.

10. What you refer as back up detail page is not a correct interpretation of the way the blood sugars are recorded. The MAR is the official document upon clinical decisions are made. The documentation in the E.H.R. is operator depending and not necessarily all of the blood sugar readings are documented. The documentation in the E.H.R. as explained above may be entered at a later time and the system registers the time of entry in the E.H.R. as resulted and the operator change or accepts the default in the collected field. The lack of signature in one of the entries is definitively a nursing error that is being actively addressed. These kinds of errors are unacceptable. Please see PRE - 5

11. The entry in the E.H.R. is post-dated; therefore the resulted time is 6/26/09. However, the blood sugar test was performed on 6/24/09, though it was not recorded in the MAR.

Procedure Reinforcement Education (PRE)

PRE 1 – Documentation of Blood Sugar in the E.H.R. All personnel have been reminded that every blood sugar test documented in the Diabetic MAR must be entered in the E.H.R. without exception. The goal is 100% and a monthly audit will be performed as a quality indicator.

PRE 2 – Accuracy transmission of data from MAR to E.H.R. All personnel have been reminded of the need to accurately enter the blood glucose test in the E.H.R. The goal is 100% accuracy and a quality indicator/audit tool has been implemented to review accuracy at least twice a month.

PRE 3 – Use exact time of collection instead of the NOW button. All personnel have been reminded of the need to accurately enter the time and date of the collected/examined blood glucose levels. The goal is 100% and it will be incorporated as a quality indicator along with the previous item.

PRE 4 – All BS must be documented in the MAR. All personnel have been reminded that the MAR is the official document to document every blood glucose test done even if not scheduled.

PRE 5 – Postdated data. All personnel have been reminded on the proper procedure to document postdated data in the E.H.R.

PRE 5 – MAR signatures. All personnel have been reminded on the mandatory nursing regulations regarding the signature/initials on every activity done in the MAR. Written reprimand is considered in the failure to sign/initial any of these activities.

Summary
Your observations have been thorough studied and I have found inconsistencies that are being constantly addressed with our personnel. However, there is no evidence of malicious intent to falsify documentation. The majority of the discrepancies noted by your observations were based on the assumption that the resulted time in the E.H.R. was the time that the blood sugar test was performed. As you realize now, the resulted time is the time that the E.H.R. documents the activity of entering the results. The actual time of the test is documented by the operator in the collected time. The two major issues from the quality improvement point of view is to better document post-dated entries and the absence in one entry of the signature/initials of the operator performing the blood sugar levels. I am determining who that worker is and it will be reprimanded.

We take your observations seriously and I hope that my review and recommendations answer to your questions. You are welcome to visit us and see our operation. You will find dedicated health care personnel, genuinely compassionate and caring for every patient in our facility.

Sincerely,

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