MEDICAL EXAMINER
District Six

Pasco & Pinellas Counties

10900 Ulmerton Road
Largo, FL 33778
727-582-6800
(Fax 727-582-6820)
www.co.pinellas.fl.us/forensics

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REPORT OF AUTOPSY

Name: SCHIAVO, Theresa Case #5050439 Age: 41 Yrs. Race: White Sex: Female
Date of Death: March 31, 2005 Date of Autopsy: April 01, 2005 at: 0840 hrs

AUTOPSY FINDINGS

1. Anoxic-ischemic encephalopathy (see attached neuropathology report)
    a. Extremity muscle atrophy and contractures
    b. Bilateral bronchopneumonia
    c. Osteoporosis (with T11 endplate fracture)
    d. Urolithiasis
    e. Renal scar (right)
    f. Heterotopic ossification
    g. Degenerative joint changes
    h. Glossal, pharyngeal, and neck muscle atrophy
    i. Healing gastrostomy
    j. Implanted electrical stimulator
    k. Healed decubitus ulcer(s) and remote left fifth toe amputation
2. Dehydration
    a. Vitreous chemistry: sodium 207 mmol/L, chloride 184 mmol/L, urea nitrogen 133 mg/dL, creatinine 1.3 mg/dL, glucose 57 mg/L
    b. Dry skin and body cavities
    c. Renal tubular necrosis
3. Toxicology
    a. Heart blood: acetaminophen 8.8 mg/L
    b. Urine: acetaminophen detected
4. Status post cholecystectomy
5. Hyperostosis frontalis interna
6. Uterine leiomyoma
7. Cardiovascular (see attached cardiovascular pathology report)
    a. Heart weight 255 grams
    b. Focal pericardial adhesions
    c. Cardiac ion channel mutation screening: negative

Cause of Death: Complications of Anoxic Encephalopathy

Manner of Death: Undetermined

Jon R. Thogmartin, M.D.
Chief Medical Examiner

Date: 06/13/05

EXTERNAL EXAMINATION:
The body is that of a 62 inch, 112 pound white female who appears the recorded age of 41 years. The body is clad in a pink and white gown. Three pillows and a blanket are also received with the body. The scalp is covered in thick brown hair with flecks of gray. The irides are brown. There is bilateral tache noire. The eyelids are yellow and dry. The eyes have a sunken appearance. The ears and nose are normally developed. The mouth has partial natural dentition. The left upper first bicuspid through the molars are absent. The left lower bicuspid is absent. The right upper second molar is capped. The left lower first molar appears decayed. The teeth are otherwise in good repair. The lips and buccal mucosa have no trauma. The neck is unremarkable except for a 2.5 cm tracheostomy scar just above the suprasternal notch. The posterior portion of the neck is unremarkable and free of scars. An obliquely oriented 6 cm surgical scar is on the anterior left chest with an underlying, implanted, medical device. The breasts are pendulous and otherwise unremarkable. There is white powder underneath the breasts. A round, 8 mm scar is on the upper central abdomen. A horizontally oriented 2.5 cm linear scar is on the central upper abdomen. A faint, approximately 1 cm scar is on the right mid lateral abdomen. There are a few striae on the hips and lower abdomen. The external genitalia are normally developed and white powder covers the perineum. The labia are dry. The urethral meatus is visible and 3.5 mm in diameter. No objects or substances are in the vagina other than a slight amount of yellow-white discharge. The anus is patent and unremarkable. Faint, pink-white, flat, 1-2 cm scars are just above the superior portion of the gluteal cleft. A 2.5 cm, somewhat square shaped, brown macule is on the left buttock. There are no open and active decubitus ulcers. The upper extremities have flexion contractures with striae on the medial portions of the upper arms. The muscles of the extremities are atrophic. The lower extremities are partially shaved. The left fifth toe is absent. The skin on the back is intact. The spine has accentuated thoracic kyphosis and lumbar lordosis. The skin demonstrates tenting

RADIOGRAPHS:
Postmortem radiographs show radiopaque shadows extending from the periosteum of the femurs, left tibia, and right ischial tuberosity. Diffuse, severe osteoporosis is present. The 11th thoracic vertebral body has an endplate fracture. Degenerative joint changes are noted in the acromioclavicular joints, hips, right knee, left foot, and pelvis. The left fifth toe is amputated along with the distal portion of the left fifth metatarsal. Radiographs of anterior neck structures and iliac wings are not remarkable. Calculi are seen in the urinary tract. Staples are in the gallbladder bed.

INTERNAL EXAMINATION:
The muscles of the chest and abdominal wall are normally developed. The subcutaneous tissues are dry. The panniculus is 2.5-3 cm. In the left chest wall is an implanted medical device with a wire extending through subcutaneous tissues of the left neck and into the left scalp. A flat, four-prong electrical device is in the subgaleal area of the left scalp. A wire then further extends into the cranial cavity. The peritoneal cavity is unremarkable and dry. There are no intraperitoneal adhesions except for an adhesion of the anterior portion of the stomach to the anterior abdominal wall in the area of the previously described round abdominal scar. The organs are in the usual anatomic relations. The pleural cavities are dry. The lungs are well aerated. The pericardial sac is remarkable for a 1 cm focal area of anterior pericardial adhesion to the anterior portion of the right ventricle. There is some lateral adhesion of the right ventricle to the right lateral portion of the pericardial sac. No other adhesions are noted. The pericardial sac is dry. The diaphragm is intact. The sternum is unremarkable. The ribs have no trauma and are normally developed with somewhat prominent costochondral junctions.

CARDIOVASCULAR SYSTEM:
The pericardial sac is remarkable as previously described. The epicardial fat of the 255 gram heart is otherwise unremarkable. The root of the aorta has no atherosclerosis. The arch and descending aorta have minimal atherosclerosis (see attached CV pathology report).

RESPIRATORY SYSTEM:
The right and left lungs are 260 and 245 grams, respectively. The lungs have a normal number of lobes and have light pink-red outer surfaces. The bronchi are unobstructed. The well-aerated lung parenchyma is pink-red. There are no anthracosis, tumors, cysts, or infarcts. The upper lobe bronchi contain a scant amount pearlescent fluid. The proximal bronchi contain yellow pearlescent fluid. The lower lobe distal bronchi contain some scattered areas of yellow pearlescent fluid. The alveoli otherwise contain foamy, reddish-white fluid. The pulmonary arteries contain no emboli. The lower lobes have firm areas of partial consolidation with yellow-green pearlescent fluid. The firm area of the left lower lobe is ~4 x 4 x 3 cm. The right lung has scattered firm areas (<1cm).

HEMOLYMPHATIC SYSTEM:
The 215 gram spleen is covered in an intact, gray, somewhat wrinkled capsule. There are two hilar accessory spleens (1.4 and 1 cm in diameter). The splenic parenchyma is dark red-maroon and unremarkable. There is no interstitial fibrosis, tumors, cysts or infarcts. No enlarged lymph nodes are noted. The bone marrow of the lumbar vertebral bodies is red and soft.

GENITOURINARY SYSTEM:
The right and left kidneys are 100 and 130 grams, respectively. The right kidney has a central, 2-2.5 cm, obliquely oriented cleft/scar extending from the central renal pelvis to the upper lateral cortex. The brown-tan outer surfaces are otherwise slightly lobular and granular. The pelvis of the right kidney is mildly dilated. A 1 x 0.6 x 0.7 cm, green-brown stone is in the pelvis of the right kidney. The left renal pelvis has an approximately 0.5 x 0.6 x 1 cm, green-brown stone. The corticomedullary ratios are reduced. The pelvic fat is increased. The left ureter contains pearlescent fluid. The urinary bladder contains ~6 cc of brown-yellow fluid. A 3.8 x 1.2 x 1 cm, white-yellow, somewhat crescent shaped stone is within the lumen of the bladder. The uterus is present and has a normal shape. The cervix is normally developed. The cervical os is large (coned) and contains mucoid fluid. There are a few minute nabothian cysts (<2 mm). A 2 cm, spherical leiomyoma is in the posterior portion of the uterine corpus. The endometrial cavity contains 3 to 4 mm thick, tan endometrium. The ovaries are present, firm and otherwise grossly unremarkable. The fallopian tubes are unremarkable except for a few adhesions of the fimbriated ends.

GASTROINTESTINAL SYSTEM:
The stomach contains 60 cc of green-brown fluid without any solid food fragments. The gastric mucosa is flat, congested, and green-gray. The gastric mucosa is congested. No ulcerations are noted. There are a few congested vessels with minute petechiae around the previously healed ostomy site. An 8 mm blood clot is on the gastric mucosa near the healed/healing gastrostomy site. The wall of the stomach is thin (<3mm). No perforations are noted. The esophagus is not remarkable with gray/pink mucosa. The bowel contains progressively formed feces with the rectum containing hard stool. The appendix is present, but is atrophic/small. The bowel has no perforations. An abundant amount of greenish liquid is in the duodenum. No foreign objects are noted.

HEPATOBILIARY SYSTEM:
The outer surface of the 965 gram liver is covered in a transparent intact capsule. There are very few inferior hepatic adhesions associated with an absent gallbladder. Surgical staples are imbedded in the area of the cystic duct. The hepatic parenchyma is brown-green with a slight pattern of congestion. The bile ducts and portal veins appear grossly unremarkable. No fibrosis, cysts or infarcts are noted. A yellow-white, round, 2 mm nodule is in the anterior portion of the right lobe of the liver.

ENDOCRINE SYSTEM:
The adrenals and pancreas are present and grossly unremarkable. The thyroid is mildly atrophic without nodules.

MUSCULOSKELETAL SYSTEM:
The upper and lower extremities are atrophic as previously described. The trunk musculature is atrophic. A 1 x 1.5 x ~1 cm area of induration/calcification extends from the anterior surface of the right femur. The anterior/lateral cortical surface of the distal right femur metaphysis is rough and irregular. The cortical bone of the lumbar and thoracic vertebral bodies is thin and soft. The iliac wings have no trauma or deformity.

NECK:
The strap muscles of the anterior neck have intact musculature with atrophy of the musculature on the right side. The right sternocleidomastoid is moderately atrophic. There are no hemorrhages. The larynx and piriform recesses contain yellow-tan, mucoid fluid. There is yellow-green, mucoid fluid on the base of the tongue and epiglottis. The larynx contains a scant amount of fluid. The thyroid and cricoid cartilages are intact. The hyoid bone is intact. The tongue is atrophic. There is a yellow-green dry crusted material on the surface of the tongue. The posterior pharyngeal musculature appears atrophic. There are no hemorrhages. A healed tracheostomy site is on the anterior trachea. The carotid arteries and jugular veins are not remarkable. The muscles and cervical vertebral bodies of the posterior neck are not remarkable. The spinal cord and column have no trauma. The posterior laminae are soft.

CENTRAL NERVOUS SYSTEM:
See attached neuropathology report.
JRT:sed

MICROSCOPIC EXAMINATION:
(Also see attached neuropathology and cardiovascular pathology reports)

LUNGS, LOWER LOBES:	WIDESPREAD BRONCHOPNEUMONIA INTRAALVEOLAR DEBRIS AND BACTERIA INTRAALVEOLAR FOAMY MACROPHAGES CONGESTION EDEMA
UTERUS:	LATE PROLIFERATIVE ENDOMETRIUM LEIOMYOMA
ACCESSORY SPLEEN:	NOT REMARKABLE
COLON:	AUTOLYSIS MELANOSIS
URINARY BLADDER:	CHRONIC INFLAMMATION
OVARIES:	CORPORA ALBICANTIA FOLLICULAR CYST
FALLOPIAN TUBES:	PARATUBAL CYST CONGESTION
VAGINA:	VASCULAR CONGESTION
ADRENAL GLANDS:	MILD CONGESTION
STOMACH, GASTROSTOMY:	CLOTTED BLOOD
LIVER:	FOCAL NODULAR HYPERPLASIA (SINGLE FOCUS) CENTRILOBULAR CONGESTION WITH STEATOSIS
KIDNEYS:	TUBULAR NECROSIS MILD VASCULAR CONGESTION
THYROID:	NOT REMARKABLE
PANCREAS:	EARLY AUTOLYSIS MILD INTERSTITIAL FIBROSIS
EPIGLOTTIS:	FOCAL ULCERATION WITH ACUTE INFLAMMATION
LARYNX, RIGHT ARYEPIGLOTTIC FOLD:	NOT REMARKABLE
TONGUE:	NO SIGNIFICANT HISTOLOGIC ABNORMALITIES

FEMUR, RIGHT:	HETEROTOPIC OSSIFICATION
FEMUR, THIGH, RIGHT:	HETEROTOPIC OSSIFICATION
RIBS, COSTOCHONDRAL JUNCTION:	NO SIGNIFICANT HISTOLOGIC ABNORMALITIES
BONE, THORACIC VERTEBRA:	SEVERE OSTEOPOROSIS UNREMARKABLE BONE MARROW
BREASTS:	NOT REMARKABLE

JR7

Pinellas County Forensic Laboratory
10900 Ulmerton Road · Largo Florida · 33778
(727) 582-6810 · Fax (727) 582-6822

Results of Laboratory Analysis
Toxicology Section

Laboratory Number	05-0002411
Submitting Agency	District Six Medical Examiner
Case Agency	District Six Medical Examiner
Agency Number	5050439

Date of Report April 11, 2005

Listed Subjects

Schiavo, Theresa

Description of Items Submitted

Item 1 A	Plastic collection tube (sodium fluoride preservative) containing approximately 10 ml of blood (heart).
Item 1 B	Large plastic tube (sodium fluoride preservative) containing approximately 22 ml of blood (heart).
Item 2 A	Small plastic collection tube (no additives) containing approximately 5 ml of blood (heart).
Item 2 B	Small plastic collection tube (no additives) containing approximately 4.5 ml of blood (heart).
Item 2 C	Small plastic collection tube (no additives) containing approximately >1 ml of serum (post mortem).
Item 3 A	Large plastic tube (sodium fluoride preservative) containing approximately 13 ml of blood (iliac).
Item 3 B	Large plastic tube (sodium fluoride preservative) containing approximately 8 ml of blood (iliac).
Item 3 C	Large plastic tube (sodium fluoride preservative) containing approximately 6 ml of blood (iliac).
Item 4	Large plastic tube (no additives) containing approximately 6 ml of urine.
Item 5	Small plastic collection tube (no additives) containing approximately 3 ml of vitreous.
Item 6 A	Large plastic tube (no additives) containing approximately 35 ml of gastric.
Item 6 B	Large plastic tube (no additives) containing approximately 33 ml of gastric.
Item 7	Large plastic tube (no additives) containing liver.
Item 8	Purple top collection tube (EDTA anticoagulant) containing approximately 2 ml of blood (heart).
Item 9	Large plastic tube (no additives) containing approximately 45 ml of cerebral spinal fluid.
Item 10	Small plastic collection tube (no additives) containing approximately <1 ml of vitreous.

Results of Analysis

Robin Neuenschwander
Chief Forensic Toxicologist

5/2/05

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(727) 582-6810 · Fax (727) 582-6822

Results of Laboratory Analysis
Toxicology Section

Laboratory Number	05-0002411	**Date of Report**	April 11, 2005
Submitting Agency	District Six Medical Examiner		
Case Agency	District Six Medical Examiner		
Agency Number	5050439		

Listed Subjects
Schiavo, Theresa

Continued. . . . .

Screens (Presumptive results only, see Identification and Quantification for confirmations.)

Item 1	Cocaine Metabolites	Negative
Item 1	Opiates	Negative
Item 1	Barbiturates	Negative
Item 1	Amphetamines/Methamphetamines	Negative
Item 1	Tricylic Antidepressants	Negative
Item 1	Cannabinoids	Negative
Item 1	Fentanyl	Negative
Item 1	Methadone	Negative
Item 1	Oxycodone	Negative
Item 1	Carisoprodol	Negative
Item 1	Acetaminophen	Positive/Confirmed
Item 1	Salicylates	Negative
Item 1	Benzodiazepines	Negative
Item 4	Cocaine Metabolites	Negative
Item 4	Opiates	Negative
Item 4	Basic Drugs	Positive/Confirmed
Item 4	Methadone	Negative
Item 4	Fentanyl	Negative
Item 4	Carisoprodol	Negative
Item 4	Amphetamines/Methamphetamines	Negative
Item 4	Cannabinoids	Negative
Item 4	Barbiturates	Negative

Robin Neuenschwander
Chief Forensic Toxicologist

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Results of Laboratory Analysis
Toxicology Section

Laboratory Number	05-0002411
Submitting Agency	District Six Medical Examiner
Case Agency	District Six Medical Examiner
Agency Number	5050439

Date of Report April 11, 2005

Listed Subjects
Schiavo, Theresa

Continued. . . . .

Item 4	Benzodiazepines	Negative
Item 4	Salicylates	Negative
Item 4	Tricylic Antidepressants	Negative
Item 4	Acetaminophen	Positive/Confirmed

Identification and Quantification

Item 1	Ethanol	Negative	Below Quant Limit: 0.005 g/dL
Item 1	Ethanol	Negative	Below Quant Limit: 0.005 g/dL
Item 1	Acetaminophen	Positive	8880 ng/ml^
Item 4	Acetaminophen	Positive	

Note(s):

^Immunochemical quantitation results are non-specific and should be intrepreted as relative values only.
Unless other arrangements are made, specimens will be discarded after a minimum two year retention.

****END OF REPORT****

Robin Neuenschwander
Chief Forensic Toxicologist

JRT / LDB

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Client: PINELLAS COUNTY MEDICAL EXAMINER
10900 ULMERTON ROAD
LARGO FL

Client #: 001094
Order Dr: THOGMARTIN, JON

Report copied to: JON THOGMARTIN

Name: **ME CASE, 5050439**
Acct#: 90000000306612 Age/Sex: 41 years / Unknown Accession #: N/A
Alt ID: Birthdate: 12/03/63 Requisition #: 10940000000128

URINALYSIS, BODY FLUID, FLOW CYTOMETRY

BODY FLUIDS

Collected Date/Time 03/31/05 12:50:00

Procedure	Results	Units	Reference Interval
BF Type	Other f		
Calcium	7.4	mg/dL	
BF Type	Other		
Chloride	184	mmol/L	
BF Type	Other		
Creatinine	1.3	mg/dL	
BF Type	Other		
Glucose	57	mg/dL	
Potassium	8.5	mmol/L	
Sodium	207	mmol/L	
BF Type	Other f		
Urea Nit	133	mg/dL	

03/31/05 12:50:00 Calcium:
"The analytical method used for this assay has not been approved by the manufacturer for this type of body fluid. No reference interval (normal range) has been established for this specimen type"

03/31/05 12:50:00 Sodium,BF Type,BF Type,BF Type,BF Type,BF Type:
Performed at Laboratory 5.
03/31/05 12:50:00 BF Type:
VITREOUS FLUID
03/31/05 12:50:00 Glucose,Urea Nit,Potassium,Calcium,Chloride,Creatinine:
Performed at Laboratory 5.
03/31/05 12:50:00 BF Type:
VITREOUS FLUID

Legend:
H=High L=Low *=Abnormal C=Critical ^=Corrected f=Footnote Started=Culture Setup ****** = Undefined range/Text response

STEPHEN J. NELSON, M.D.

A PROFESSIONAL ASSOCIATION

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ANATOMIC PATHOLOGY • FORENSIC PATHOLOGY • NEUROPATHOLOGY

D/B/A DISTRICT 10 MEDICAL EXAMINER
1021 JIM KEENE BOULEVARD
WINTER HAVEN, FLORIDA 33880-8010

(863) 298-4600
(863) 298-5264 TELECOPIER

June 8, 2005

Jon R. Thogmartin, M.D.
District 6 Medical Examiner
10900 Ulmerton Road
Largo, FL 33778-1633

In Re: Theresa Marie ("Terri") SCHIAVO, deceased
Your Medical Examiner Case No. 5050439

Dear Dr. Thogmartin:

On Friday, April 1, 2005, I, along with other forensic pathologists from your office, participated with you in the autopsy of the decedent. My role was as your designated consultant neuropathologist. As such, I personally removed the decedent's dura mater, brain, spinal cord, pituitary gland, a portion of the right gastrocnemius skeletal muscle, and examined them immediately after their removal at autopsy. The dura mater, brain and spinal cord were then fixed in a solution of 10% neutral buffered formalin for a period of eighteen (18) days with the formalin solution changed often during that time. They were again examined by me in your facility on Tuesday, April 19, 2005 and standard routine neurohistologic sections were obtained at that time.

Neuropathology Gross Description:

The head was explored via the standard intermastoid incision. The frontal bone, cut in the typical a horizontal fashion above the level of the zygomatic arches, demonstrated hyperostosis frontalis interna. There were coalescences of multiple granular bony excrescences, measuring between 1-2 centimeters each, overlying the frontal sinuses on the floor of the anterior cranial fossa. These excrescences were 4-5 millimeters thick anteriorly, and up to 1 cm thick posteriorly. There was no evidence of subgaleal or subscalpular blood, discoloration, or staining. The calvarial bones were intact throughout. There was no epidural or subdural blood, discoloration, or staining. The dura mater was intact and the venous sinuses were widely patent. There was no meningeal staining or discoloration.

A 9 centimeter long implanted neurological thalamic stimulator wire extended outward from the right parietal bone and it was surrounded by a 1 centimeter bony nodule on the inner table. This wire was traced and its tip terminated in the right thalamus.

The diaphragma sella was grossly concave and depressed. The pituitary gland was dissected free from the sella turcica using sharp dissection taking care to preserve a portion of the pituitary stalk. It appeared grossly unremarkable with appropriate demarcation of the anterior and posterior lobes. It measured 13 milimeters in transverse dimension, 6 milimeters in vertical dimension, and 9 milimeters in anterior-posterior dimension. There was no gross evidence of empty sella syndrome. The pituitary gland was then trisected in the anterior-posterior plane and submitted *in toto* in one cassette for subsequent histopathologic examination utilizing hematoxylin and eosin stain (H&E).

At autopsy, the brain weighed 615 grams. A total of 645 milliliters of cerebrospinal fluid (weighing 678 grams) were recovered upon opening the skull and exposing the brain. The brain was small, with widened sulci and narrow and thinned gyri. It was smaller in its vertical dimension as a result of the hydrocephalus *ex vacuo* tissue volume loss. The worst affected areas were the bilateral occipital lobes. The leptomeninges were opaque and golden-brown throughout. There were no leptomeningeal exudates. The cranial nerves are intact, though somewhat discolored golden-brown. The optic nerves were somewhat thinner than expected, but the remaining cranial nerves were otherwise within the expected limits of "normal." There was bilateral dilatation of the frontal-temporal-parietal opercula, covered by thickened leptomeninges.

The arterial circle of Willis at the base of the brain was that of a normal "adult" configuration. There was no significant gross evidence of atherosclerosis, vascular anomaly or aneurysmal dilatation.

The pineal gland was grossly remarkable only for the thickened surrounding leptomeninges.

The infratentorial structures were relatively unremarkable externally. There was no herniation. The leptomeninges were opaque, but were also less golden-brown than those covering the cerebrum.

Sequential horizontal sections of the brain (cerebrum, brain stem and cerebellum) were cut parallel to Reid's base line at 5 millimeter intervals. In all, nineteen (19) slices were made from the superior cerebral convexity, down to the level of the upper cervical spinal cord. There was prominent diffuse ventriculomegaly (hydrocephalus *ex vacuo*). There is no midline shift of any cerebral or cerebellar structures. The lateral ventricles were markedly dilated, but they were without displacement. The foramen of Monro and the aqueduct of Sylvius were also widely dilated. The choroid plexus and ependyma were intact and grossly unremarkable. The cortical gray ribbon, though intact throughout, was diffusely thinned and attenuated and for the most part, golden-brown. The subjacent cerebral white matter was soft, gelatinous and also diffusely discolored gray-tan-brown, including the bilaterally symmetric basal ganglia, thalami and hippocampal formations. The thalami

appear less affected than the basal ganglia. The mammillary bodies were shrunken and small and discolored golden-brown. The septum pellucidum was intact and thickened. There was no cavum at the level of the genu.

Sequential horizontal sections were cut through the midbrain, pons, cerebellum and medulla oblongata. The pigmentation of both the locus ceruleus and substantia nigra was appropriate for chronologic age. The cerebellum displayed no unusual gross developmental features. The deep midline nuclei of the cerebellum were symmetric and the folia demonstrated significant golden-brown discoloration and widespread atrophy. The medulla oblongata, including the pyramids, were smaller than expected, but bilaterally symmetric.

The entire length of the spinal cord was removed, including filum terminale and cauda equina. A few dorsal root ganglia were also included. The spinal cord was serially sectioned at 5 millimeter intervals, perpendicular to its long axis. There was a subjectively tan-gray discoloration in the lumbar region associated with some narrowing of the cord diameter. The leptomeninges were grossly unremarkable and free of exudates.

A portion of right gastrocnemius skeletal muscle, measuring 2.5 x 2.5 x 0.2 centimeters was excised in cross section to the long axis of muscle fibers and submitted *in toto* in one cassette for subsequent histopathologic examination utilizing hematoxylin and eosin stain (H&E), and Masson's trichrome stain.

Neuropathology Macroscopic Description:

A total of sixty-five (65) glass microscopic slides, from multiple representative neuroanatomic sections, were examined microscopically. All glass slides were stained with hematoxylin and eosin (H&E) and examined, and the paraffin blocks for all tissues (except the pituitary gland and gastrocnemius skeletal muscle) were then re-cut and additional glass slides were prepared utilizing the Luxol fast blue (LFB) staining protocol for myelin. The histologic sections included multiple sections of cerebral cortex and white matter, subcortical gray matter (basal ganglia and thalamus), hippocampal formations, midbrain, pons, cerebellum, medulla oblongata, spinal cord, pituitary gland, pineal gland, and skeletal muscle (right gastrocnemius muscle).

The changes seen were striking in their appearance, and global in their distribution. They predominately involved the border zone ("watershed") areas and were most severe in the occipital lobes, with relative preservation of the frontal and temporal lobes. There was a readily discernible and

noteworthy gradient loss when moving from the anterior to posterior regions. In the thalamus, the most medial portions were relatively preserved (from the frontal cortex). In the basal ganglia, the corpus striatum all but vanished, replaced by extensive astrocytosis (caudate nucleus greater than putamen). This volume loss was impressive and was all but completely represented by non-neoplastic astrocytes. An occasional rare neuron was located. The frontal and temporal poles and insular cortex demonstrated relative preservation. The granular neurons of the cerebral cortex were relatively preserved, while the larger pyramidal neurons were globally absent. There was laminar necrosis involving the middle cortical lamina, in most cortical sections examined microscopically, but this finding was patchy. Hyaline sclerosis of many of the smaller microscopic arterioles was noted and many arterioles also contained widely dilated perivascular spaces. There were calcific vasopathic changes and diffuse astrocytosis in the globus pallidus.

The pyramids demonstrated pyramidal tract Wallerian degeneration. Damage to the midbrain, including the red nuclei, appeared related to Wallerian degeneration from fibers passing through these neuroanatomic structures. The lateral geniculate nucleus (visual) demonstrated transneuronal degeneration with gliosis, while the medial geniculate nucleus (auditory) was relatively preserved. The hippocampal formations demonstrated diffuse neuronal loss (CA1 thru CA4, and endplate) associated with reactive astrocytosis. The fascia dentata was preserved.

Within the cerebellum there were no recognizable Purkinje cells found. The lost Purkinje neurons were replaced by reactive Bergmann astroglia. In the pons the descending fiber pathways were most affected. The inferior olivary nuclei had no remaining discernable neurons, suggesting this was a retrograde degeneration due to the marked cerebellar cortex damage. The deep midline cerebellar nuclei were relatively preserved, but with prominent astrocytosis. The dorsal motor nucleus was relatively preserved, as was the hypoglossal nucleus. The reticular activating system was also relatively preserved. The locus ceruleus and median raphe nucleus were relatively preserved. The cardiorespiratory centers in the medulla oblongata were relatively preserved.

Multiple representative levels of the spinal cord were examined microscopically. The crossed and uncrossed fibers of the corticospinal tract were abnormal. There was degeneration of the posterior columns. In the cervical-thoracic levels this involved only the fasciculus gracilis, leaving the fasciculus cuneatus relatively uninvolved. The lumbar levels contained a border zone ("watershed") infarct that was symmetrical, extending from central to peripheral. There were multiple foci of loss of the anterior horn cell neurons in the lumbar-sacral regions.

The right gastrocnemius skeletal muscle, stained with H&E and with Masson's trichrome stain

using colon tissue as a stain control, demonstrated a mixture of myopathic and neurogenic features. No inclusions were identified. No inflammatory cells were present.

The pituitary gland was histologically unremarkable. The pineal gland contained diffuse astrocytic gliosis.

Impression:

Hyperostosis frontalis interna, skull
Neurological stimulator, implanted, thalamus, right
Encephalopathy, anoxic-ischemic, multifocal/global,
laminar necrosis
border zone ("watershed") territories
transneuronal degeneration (lateral geniculate nucleus)
Wallerian degeneration, descending fiber pathways
Hyaline arteriolosclerosis, diffuse
Globus pallidus, with calcific vasopathy
Hydrocephalus, *ex vacuo*
Spinal cord, with
border zone infarct, lumbar
posterior column degeneration
anterior horn neuron loss, lumbar-sacral
Gastrocnemius skeletal muscle, right, with myopathic and neurogenic features
Pituitary gland, no specific neuropathologic diagnosis
Pineal gland, with diffuse astrocytic gliosis

Comment: Brain weight is an important index of its pathologic state. Brain weight is correlated with height, weight, age, and sex. The decedent's brain was grossly abnormal and weighed only 615 grams (1.35 lbs.). That weight is less than half of the expected tabular weight for a decedent of her adult age of 41 years 3 months 28 days. By way of comparison, the brain of Karen Ann Quinlan weighed 835 grams at the time of her death, after 10 years in a similar perisistent vegetative state[1].

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[1] Kinney, H.C., Korein, J., Panigrahy, A., et al.: Neuropathological findings in the brain of Karen Ann Quinlan – the role of the thalamus in the persistent vegetative state. ***The New England Journal of Medicine*** 1994; 330: 1469-1475.

There are numerous publications in both the neurologic and neuropathologic literature of the correlates that exist between the clinical examination and clinical diagnosis of a patient in persistent vegetative state and the subsequent neuropathologic findings at autopsy. The persistent vegetative state[2,3] and minimally conscious state[4], are clinical diagnoses, not pathologic ones. The neuropathologic findings of the persistent vegetative state[5,6,7] have been well described in the medical literature, including long survival after cardiac arrest[8], yet there are no similarly published neuropathologic descriptions specific to the minimally conscious state.

> The anatomical basis for a persistent vegetative state differs somewhat from case to case, for several reasons. The interval between brain injury and death affects the nature and severity of pathologic changes. Patients in a vegetative state who die early of medical complications are unlikely to undergo neuropathologic changes that would be sufficient to cause chronic unconsciousness in long-term survivors. Furthermore, in patients with chronic neurologic conditions, other complicating factors, such as severe atherosclerotic disease, may independently injure the brain. In such patients, it may be difficult to determine at autopsy exactly which neuropathologic changes accompanied

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[2] Multi-Society Task Force on PVS. Medical Aspects of the persistent vegetative state – first of two parts. ***The New England Journal of Medicine*** 1994; 330: 1499-1508.

[3] Multi-Society Task Force on PVS. Medical Aspects of the persistent vegetative state – second of two parts. ***The New England Journal of Medicine*** 1994; 330: 1572-1579.

[4] Giancino, J.T., Ashwal, S., Childs, N. et al.: The minimally conscious state. Definition and diagnostic criteria. ***Neurology*** 2002; 58: 349-353.

[5] Dougherty, J.H., Rawlinson, D.G., Levy, D.E. et al.: Hypoxic-ischemic brain injury and the vegetative state: Clinical and neuropathologic correlation. ***Neurology*** 1981; 31: 991-997.

[6] Adams, J.H., Graham, D.I., Jennett, B.: The neuropathology of the vegetative state after an acute brain insult. ***Brain*** 2000; 123: 1327-1338.

[7] Kinney, H.C., Samuels, M.A.: Neuropathology of the persistent vegetative state. A review. ***Journal of Neuropathology and Experimental Neurology*** 1994; 53: 548-558.

[8] Cole, G., Cowie, V.A.: Long survival after cardiac arrest: case report and neuropathological findings. ***Clinical Neuropathology*** 1987; 6: 104-109.

the initial failure to recover consciousness.[2]

Allowing for the above limitations, two major patterns have characterized most detailed reports on the neuropathology of a persistent vegetative state due to acute traumatic or non-traumatic brain injury: diffuse laminar cortical necrosis and diffuse axonal injury.[2]

In diffuse laminar cortical necrosis the pattern follows acute, global hypoxia and ischemia. The principal finding is extensive multifocal or diffuse laminar cortical necrosis with almost invariable involvement of the hippocampus. These abnormalities may be accompanied by scattered small areas of infarction or neuronal loss in the deep forebrain nuclei, hypothalamus, or brain stem. Relatively selective thalamic necrosis may also follow acute global ischemia, although the specific anatomical boundaries for this uncommon pattern have not been well described.[2]

In diffuse axonal injury this abnormality is usually due to a shearing injury after acute trauma. An extensive subcortical axonal injury virtually isolates the cortex from other parts of the brain. Sometimes a diffuse axonal injury is accompanied by small primary brain-stem injuries, as well as secondary damage to the brain stem that results from transtentorial herniation soon after the injury. In patients with an axonal injury complicated by acute circulatory or respiratory failure, diffuse laminar necrosis may also be present.[2]

Only a few pathological reports on the persistent vegetative state describe severe abnormalities of the brain stem. Those that do mainly concern patients in whom severe paramedian mesencephalic damage developed secondary to acute downward or upward transtentorial herniation during the early stage of illness. Lesions confined to the brain stem seldom, if ever, cause long-term unconsciousness, although there has been a report of four patients[9] with severe secondary brain-stem damage in whom coma persisted for as long as six weeks before death.[2]

The gross and microscopic neuropathologic findings here are very similar to the changes seen

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[9] McClellan, D.R., Adams, D.I., Graham, A.E. et al.: The structural basis of the vegetative state and prolonged coma after non-missile head injury. *In* Papo, I., Cohadon, F., Massarotti, M. eds. ***Le Coma Tramatique***. Padova, Italy: Liviana Editrice, 1986; 165-185.

in multicystic encephalopathy[10], an encephaloclastic defect originating in the third trimester of pregnancy in which the white matter and deeper parts of the cerebral cortex, as well as the basal ganglia, are transformed into an elaborate glial-vascular meshwork by bilaterally symmetric hemispheric cystic necrosis. There are no accompanying cortical malformations. Similar neuropathologic findings have been described in status marmoratus, a form of hypoxic-ischemic perinatal brain injury, involving the basal ganglia[11], like cerebral palsy. Smith and Rodeck[12], and Adams et al.[13], have both reported patients who also had lesions in both brain stem and spinal cord in patterns that were highly suggestive of cardiac arrest encephalopathy.

In this case, the thalamus was less affected grossly than the basal ganglia, but microscopically they were similarly affected. The presence of Bergmann astrocytes in place of Purkinje neurons in the cerebellum is a typical neuropathologic finding associated with anoxic-ischemic encephalopathy. *Ex vacuo* hydrocephalus is merely the replacement of lost cerebral tissue with cerebrospinal fluid. Because no imbalance in fluid production and absorption exists, this technically is not hydrocephalus. The right gastrocnemius skeletal muscle demonstrated a mixture of myopathic and neurogenic features, consistent with the loss of anterior horn cell neurons in the lumbar and sacral levels of the spinal cord.

Hyperostosis frontalis interna, an overgrowth of bone at the inner table of the frontal bone, is usually bilateral and symmetrical and chiefly in females over 35 years of age. It has no known clinical significance and is of unknown etiology. It may be associated with irregular cortical thickening of the frontal area and it spares those areas occupied by superior sagittal sinus and venous channels. It may be 1 centimeter or thicker, and it occasionally extends to parietal bones and orbital roofs. It is distinguished by hyperostosis calvaria diffusa, a variant of hyperostosis frontalis interna, that shows more

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[10] Friede, R.L.: ***Developmental neuropathology***, 2nd ed. New York: Springer-Verlag, 1989.

[11] Kinney, H.C. and Armstrong, D.D.: Perinatal Neuropathology. *In* ***Greenfield's Neuropathology***, 7th ed. Edited by Graham, D.I. and Lantos, P.L. New York: Arnold Publishers/Oxford University Press, 2002.

[12] Smith, J.F., Rodeck, C.: Multiple cystic and focal encephalomalacia in infancy and childhood with brain stem damage. ***Journal of the Neurological Sciences*** 1975; 25: 377-388.

[13] Adams, R.D., Prod'hom, L.S., Rabinowicz, T.: Intrauterine brain death. Neuraxial reticular core necrosis. ***Acta Neuropathologica*** 1977; 40: 41-49.

diffuse thickening of vault (involving both inner and outer tables of bone). It is not clear that this disorder is actually rare. Some clinicians believe that it may be a common abnormality found in as many as 12 percent of the female population. The disorder may be found associated with a variety of conditions.

Much discussion took place in the media concerning why the decedent had not undergone an MRI scan of her brain, rather than only a brain CT scan while alive. Last month, the Director of the Center for Devices and Radiological Health at the U.S. Food and Drug Administration (FDA) issued an advisory to healthcare professionals that serious injury or death can occur when patients with implanted neurological stimulators – such as the decedent's implanted thalamic stimulator – undergo MRI (magnetic resonance imaging) procedures[14]. The FDA received several reports of serious injury, including coma and permanent neurological impairment, in patients with implanted neurological stimulators who underwent magnetic resonance imaging (MRI) procedures. The mechanism for these adverse events was likely to involve heating of the electrodes at the end of the lead wires, resulting in injury to the surrounding tissue. Although these reports involved deep brain stimulators and vagus nerve stimulators, it was believed that similar injuries could be caused by any type of implanted neurological stimulator, such as spinal cord stimulators, peripheral nerve stimulators, and neuromuscular stimulators.

Neuropathologic examination alone of the decedent's brain – or any brain, for that matter – cannot prove or disprove a diagnosis of persistent vegetative state or minimally conscious state.

Sincerely yours,

STEPHEN J. NELSON, M.D., P.A.

Stephen J. Nelson, M.A., M.D., F.C.A.P.
Chief Medical Examiner
10th Judicial Circuit of Florida

SJN:cld

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[14] Schultz, D.G.: ***FDA Public Health Notification***: MRI-caused injuries in patients with implanted neurological stimulators. May 10, 2005, Washington, D.C.

Spectrum Health

PATHOLOGY AND LABORATORY MEDICINE *Blodgett Campus*
1840 WEALTHY SE GRAND RAPIDS MI 49506-2968
616 774 7721 FAX 774 7749 *www.spectrum-health.org*

May 10, 2005

Jon Thogmartin, MD
District Six Medical Examiner
10900 Ulmerton Rd
Largo, FL 33778

Dear Dr. Thogmartin,

Enclosed please find the written report by Dr. Stephen Cohle on your case, ME#5050439. The heart tissue, tissue blocks and slides are being sent under separate cover.

Sincerely,

Susan Atwood
Forensic Administrative Assistant

Enclosure: report

Consult 935
Dr. Thogmartin
Florida Medical Examiner District Six
(Pinellas and Pasco Counties)
ME#5050439
1

Received is a previously dissected heart weighing 220 grams. The right and left ventricular outflow tracks have been opened but not the atria. The heart is moderately distorted postfixation. The apical two thirds of the heart have been breadloafed in the short axis plane. The anterior wall of the right ventricle at the base of the heart has a thin fibrinous area of old pericarditis measuring 3.5 cm x 3.5 cm. The posterior wall of the left ventricle at the base has a discrete area of fibrous adhesions between the visceral and parietal pericardium. This covers an area measuring 2.5 cm x 1.3 cm, in which a portion of the parietal pericardium is tightly adherent to the visceral pericardium. The coronary arteries are normally disposed, right predominant and are widely patent. They are dissected on the base of the heart and on the four breadloafed slices of myocardium. The myocardium is dark brown and is intact. There is distortion of the free wall as well as the base of the heart by fixation. The left ventricular free wall thickness is 8 mm, the interventricular septum measures 9 mm, the posterior wall of the right ventricle is 2 mm and the anterior wall ranges in thickness from 1-2 mm. There is biventricular dilatation. Because of distortion the exact diameter of the ventricular cavities cannot be measured. The endocardium is smooth and glistening. The valves are intact, without evidence of natural disease. The coronary ostia are normally situated and are widely patent. The ascending aorta has mild atherosclerotic plaques. The fossa ovalis is closed. Representative sections of the myocardium are taken for study as is the cardiac conducting tissue. Representative sections of the left ventricular and right ventricular free walls are submitted at two different levels.

SDC/sla
4/12/05

MICROSCOPIC

CARDIOVASCULAR SYSTEM: Slide A, from the superior vena cava/right atrial junction, has no nodal tissue, although the SA node artery is identified in each of the two pieces. The ganglia, nerves and supportive tissue around the artery are unremarkable. Slide B contains very early SA nodal tissue which is unremarkable. Slides C and D have unremarkable SA node. The supportive structures, mainly the artery, surrounding nerves, working myocardium and connective tissue are unremarkable. Slide E has residual nodal tissue in one of the two pieces. The supportive structures are unremarkable. Sections of the AV node, nodal artery, central fibrous body and the surrounding nerves and ganglia have no pathologic change. The working myocardium of the ventricular septum is unremarkable. The penetrating His bundle is unremarkable. The branching His bundle is left-sided. There is focal punctate calcification of the central fibrous body just above the branching His bundle. An intramyocardial arteriole in this slide (H) has mild fibromuscular hyperplasia. Slide I is from a piece of tissue that was twisted and is thus maloriented. The left side of the specimen is oriented such that the plane of sectioning is anterior-posterior rather than the intended left to right plane. This results in a course of the central fibrous body appearing to invaginate into the interior of the piece of tissue. There are no abnormalities of the branching His bundle. The right bundle branch, which is on slide K, is unremarkable. Sections of the anterior wall of the left ventricle are unremarkable. Sections of the lateral wall have mild subepicardial fatty infiltration. One section has mild perivascular fibrosis. Sections of the posterior wall have mild perivascular fibrosis. Sections of the interventricular septum are unremarkable. Sections of the anterior wall of the right ventricle have moderate fatty infiltration. No abnormalities are present. Sections of the posterior wall of the right ventricle have moderate fatty infiltration.

SDC/sla
4/18/05

Additional levels through the AV node have no pathologic change. Deeper sections through the His bundle and left bundle branch have no significant pathologic alterations. Deeper sections through the right bundle branch show no abnormalities.

SDC/sla
4/22/05

Additional sections through block H show a left sided His bundle. An intramyocardial arteriole has mild fibromuscular hyperplasia. This is deep in the septum. No other abnormalities are present.

Consult 935
Dr. Thogmartin
Florida Medical Examiner District Six
(Pinellas and Pasco Counties)
ME#5050439
3

DIAGNOSIS:
Adult heart with focal healed pericarditis and biventricular dilatation.
No significant abnormalities identified within the working myocardium or conducting tissue.

Stephen D Cohle 5-10-05

Stephen D. Cohle, MD
Forensic Pathologist

GENAISSANCE
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May 5, 2005

Noel Palma, MD
Florida Medical Examiners Office
District Six
10900 Ulmerton Road
Largo, FL 33778

Dear Dr. Palma,

Thank you for referring your patient for the *FAMILION*™ Test for cardiac ion channel mutations. Although the *FAMILION* Test did not identify any Class I or Class II Long QT Syndrome (LQTS) variants (see *FAMILION* Technical Information Sheet for explanations of Class I and Class II variants), this does not rule out a diagnosis of a cardiac channelopathy. In fact, approximately 25% of patients with a high index of suspicion for LQTS will have a negative genetic test.

If your index of suspicion for LQTS is high for this patient, mutational analysis for novel genetic mechanisms in LQTS is available as part of an IRB-approved research protocol through the LQTS/Sudden Death Genomics Laboratory at Mayo Clinic, Rochester, MN, under the direction of Michael J. Ackerman, M.D., Ph.D. To discuss enrolling this patient or for more information about Dr. Ackerman's LQTS research genetic testing, please contact Dr. Ackerman by telephone at (507-284-0101) or by email at ackerman.michael@mayo.edu.

Respectfully,

Carol R Reed, MD

Carol R. Reed, MD, FACP, FCCP
Vice President Medical Affairs
Genaissance Pharmaceuticals
cc: file

FAMILION™

A GENETIC TEST FOR CARDIAC ION CHANNEL MUTATIONS

FORM R1

Comprehensive Test Result Report to Physician (CONFIDENTIAL)

PHYSICIAN	SPECIMEN	PATIENT
Physician's Name: NOEL PALMA Hospital/Institution: FLORIDA MEDICAL EXAMINERS OFFICE DISTRICT SIX Mailing Address: 10900 ULMERTON ROAD, LARGO FL 33778 USA	Specimen Type: Blood Draw Date: 04-01-2005 Accession Date: 04-14-2005 Report Date: 05-03-2005	Patient's Name: THERESA SHIAVO Date of Birth: 12-03-1963 Patient ID: LQT9921343846 Gender: F Requisition #: HT0043995

TEST RESULTS

No Deleterious Mutation Detected

This individual is negative for a disease-associated mutation.

INTERPRETATION

No Class I or Class II disease-associated mutations were found. This result is inconclusive and DOES NOT EXCLUDE the presence of a heritable disorder predisposing to cardiac arrhythmias, as variants in genomic regions not included in this test may play a causative role in such disorders. Among patients with a high clinical index of suspicion of disease, up to 25% of patients with LQTS and 70% of patients with Brugada Syndrome have disease-associated variants that would not be identified with this test.

CLASS III Variant Found: This test result indicates identification of one or more genetic variants that have been identified previously in normal subjects or are considered common polymorphisms. Such variants are not likely arrhythmia syndrome-causing variants, but the actual significance of these variants has yet to be determined. Whether or not the presence of such variants confers additional risk during exposure to medications with QT prolonging potential is unknown at the present time. Family screening for the presence of such variants is, in general, NOT recommended but may be considered in families with clearly penetrant familial arrhythmia syndromes to determine co-segregation.

Result Summary:

Num	Gene	Region(G)	Nucl.Change	A.A.Change	Genotype	Region(P)	Region Type(P)	Class
1	KCNE1	exon 4	112 G>A	Gly 38 Ser	A/A	N-Terminal	N-Terminal	III
2	KCNH2	exon 11	2690 A>C	Lys 897 Thr	A/C	C-Terminal	C-Terminal	III

All regions of interest (100 %) were successfully sequenced in the *FAMILION*™ Comprehensive Test for the genes KCNQ1 (LQT1), KCNH2 (LQT2), SCN5A (LQT3), KCNE1 (LQT5) and KCNE2 (LQT6).

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Limitations: This assay will not detect large DNA rearrangements or deletions and will not detect all errors in RNA transcription or processing which are unrelated to coding sequence variants of DNA exons.

These test results should only be used in conjunction with the patient's clinical history and any previous analysis of appropriate family members. It is strongly recommended that these test results be communicated to the patient in a setting that includes appropriate counseling. The results of this test are not intended to be used as the sole means for patient diagnosis or patient management decisions. The performance characteristics of this test were validated by Genaissance and it is specific only for variants in genes that are associated with cardiac ion channel mutations. The U.S. Food and Drug Administration (FDA) has not approved this test; however, FDA approval is not currently required for clinical use of this test. This test meets the requirements for high complexity tests under the Clinical Laboratory Improvement Amendments Act and its implementing regulations. The test may use some reagents produced for research purposes only.

CT License Number: CL-0633 **CLIA Number: 07D0995237**

Authorized Signature:

Jeffrey M. Otto, Ph.D., Lab Director
Patricia D. Murphy, Ph.D. Associate Lab Director

X: ✓ Patricia D Murphy

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MEDICAL EXAMINER
District Six

Pasco & Pinellas Counties

10900 Ulmerton Road
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www.co.pinellas.fl.us/forensics

June 13, 2005

On March 31st 2005, the District Six Medical Examiner Office began the death investigation of Theresa Marie Schiavo (ME case # 5050439). The chain of events leading to this death investigation began 15 years ago in the early morning hours of February 25, 1990. The determination of her cause and manner of death would include an investigation of the events that occurred and review of available records produced subsequent to that date and time and, regarding some circumstances, extent to a review of events prior to that date.

On April 1st at 0840 hours, a complete autopsy was performed which included external and internal examinations including 72 external photographs and 116 internal photographs. A radiologic survey of her entire body was performed using a digital fluoroscopic C-arm (X-Ray) with 58 pre-autopsy images captured. During and after the autopsy a second radiologic examination was performed capturing an additional 28 images including multiple views of Mrs. Schiavo's anterior neck structures, torso and pelvis. Thus, a total of 274 images were recorded during this examination. Detailed dissections were performed on Mrs. Schiavo's anterior and posterior neck, spine, and brain. Multiple microscopic samples were taken and examined. A Board certified Neuropathologist examined her central nervous system, and a Board certified Anatomic/Clincal/Forensic Pathologist with special expertise in cardiovascular pathology examined cardiac tissue. Genetic testing for cardiac ion channel mutations was performed to detect markers for Long QT Syndrome. Complete toxicology testing was also performed. The vitreous humor chemistry was examined. Court, medical and other records, including public and confidential DCF and law enforcement documents, were reviewed as part of the death investigation.

1. What was the cause of Theresa Schiavo's collapse in 1990?
    a. Did she suffer from an eating disorder?

        Mrs. Schiavo was heavy as a teenager, but after high school she tried several standard diets and sought medical help. According to her parents, she was able to lose over 100 lbs after reaching a maximum weight of approximately 250 lbs. Reportedly, she desired to maintain the weight loss. According to those that knew Mrs. Schiavo, her eating and drinking habits included eating lots of salad, eating a large omelet on weekends and drinking large amounts of ice tea. No one observed Mrs. Schiavo taking diet pills, binging and purging or consuming laxatives, and she apparently never confessed to her family or friends about having an eating disorder. Recent interviews with family members, physicians, and coworkers revealed no additional information supporting the diagnosis of Bulimia Nervosa and, indeed, many other signs and symptoms of Bulimia Nervosa were not reported to be present.

After Mrs. Schiavo's resuscitation from her initial cardiac arrest, the first blood draw at Humana Hospital-Northside showed a low serum potassium level (2.0 mmol/L). Several other blood components were also at abnormal levels. Her low potassium level appears to be the main piece, if not the only piece, of evidence purported to show that she had an eating disorder.

Mr. Schiavo filed a malpractice suit against Mrs. Schiavo's primary care physician and gynecologist regarding the care she received prior to her collapse. Her primary care physician reached an out of court settlement with Mr. Schiavo. After a trial, a court entered a medical malpractice judgment against her gynecologist. Apparently, the opposing sides at trial generally accepted the theory that Mrs. Schiavo's low potassium level was the cause of her initial collapse and that Bulimia Nervosa or another eating disorder was the likely underlying cause of her problems. Witnesses presented at trial testified that Mrs. Schiavo did not confess to having an eating disorder nor was any testimony given of witnessed purging. Expert witnesses at the trial including outside experts in psychiatry and gynecology theorized that an eating disorder was the most likely cause of her low potassium level and, thus, the underlying cause of her heart problem and resulting brain damage. Several physicians at trial admitted to other explanations for her hypokalemia including artifact and polydipsia.

Her post-resuscitation potassium level and history of remote weight loss appear to be the only evidence that indicate that she may have had some type of eating disorder.

b. What caused her hypokalemia (low potassium)?

On February 25th 1990, according to available records, a 911 call was made at approximately 0540hrs. Both Mr. Schiavo and Bobby Schindler were present prior to arrival of emergency responders. They both describe her as lying prone and breathing or at least they describe her as "making gurgling noises". According to her medical records, paramedics began treating Mrs. Schiavo at 0552hrs. The Pinellas County EMS report records her as supine in the hallway with no respiration and her initial cardiac rhythm was ventricular fibrillation. She was intubated within the first 5 minutes. During her resuscitation, she received dextrose solution, five 1 mg doses of epinephrine (1 by ET tube, 4 IV), lidocaine, (bolus and drip), Narcan, dopamine and seven defibrillations. Although a pulse was documented at 0632hrs, a measurable systolic blood pressure was not recorded until 0646hrs almost one hour after resuscitation was begun. Her time of arrival at Humana Hospital-Northside was 0646hrs. At 0701hrs, her blood was drawn and that sample showed hypokalemia (2.0 mmol/L, normal 3.5-5.0) one hour after her initial collapse and after over 30 minutes of CPR. The results were available at 0725hrs and her doctors began potassium supplementation almost immediately resulting in a rapid rise of her potassium to 2.9 mmol/L by 0928hrs. Her potassium was measured at

2.6 mmol/L at 1212hrs, and, by 2145hrs, her potassium was within the normal range (3.6 mmol/L).

Mrs. Schiavo was in extremis for over 1 hour prior to her initial blood sampling. It appears that she received approximately 1250cc of fluid in the field (and/or at least by the time she was being worked up in the hospital). She had also received epinephrine, and had suffered a period of ventricular fibrillation all of which are known to cause factitious changes in blood electrolytes and other substances. Factitious potassium levels as low as 2.0 mmol/L have been reported and average decreases of 0.8 mmol/L have been documented experimentally. Potassium reductions of as much as 2 mmol/L have been reported. The dosage of epinephrine she received was sufficient to cause such factitious lowering of potassium. Even a cursory examination of cases from the District Six Medical Examiner Office revealed patients with measured antemortem, post resuscitation potassium levels as low as 2.6 mmol/L with various causes of death. Her parents report that on the evening prior to her collapse they all consumed a large meal, and the circumstances of the evening activities would have made it difficult for Mrs. Schiavo to covertly purge the meal. Thus, it is reasonable to conclude that Mrs. Schiavo's potassium level of 2.0 mmol/L measured after a period of ventricular fibrillation, epinephrine, and fluid administration was an unreliable measure of her pre-arrest potassium level. Thus, the main piece of evidence supporting a diagnosis of Bulimia Nervosa is suspect or, at least, can be explained by her clinical condition at the time of the blood draw.

In addition to the abnormal potassium, many of Mrs. Schiavo's initial laboratory values were also abnormal. As expected, some of her blood enzymes were elevated due to the period of cardiorespiratory arrest. Other blood values were also mild to markedly abnormal. The most significant were decreases in her hemoglobin, hematocrit, urea nitrogen, albumin, total protein, globulin, and calcium. Dilution of her blood proteins and electrolytes from excessive fluid consumption is possible, and many of her low blood values may reflect the large fluid bolus she received during resuscitation. Such anomalies have been described in patients in similar clinical settings; therefore, these values are unlikely to be clues to the etiology of her initial insult. Although in the malpractice proceedings the low protein values were suggested as indicators of malnutrition, this is unlikely and not generally characteristic of Bulimia Nervosa.

If her initial serum potassium is to be regarded as reliable, then multiple etiologies are possible given her nutritional history. Bulimia Nervosa involving binging and purging would be high on the list of differential diagnoses. In a young woman concerned with weight loss, use of diuretics, laxatives, or other potassium depleting substances are reasonable possibilities, but no evidence of their use exists. According to the original St. Petersburg Police Department report (90-024846) from

February 1990, "various" medications were noted at the scene, yet descriptions of the medications were not recorded. Two were prescribed to Mrs. Schiavo. The toxicology screen performed during her initial hospital admission was negative, but the screen would not detect such potassium depleting substances in that she was specifically screened for barbiturates, cocaine metabolites, opiates, benzodiazepines, amphetamines/methamphetamines, tricyclics, salicylate, acetaminophen and ethanol. The immunoassay technique used specifically tested for the drug classes listed above and no others, and the technique has limitations.

Her tea drinking habits may also have played a role. Reportedly, she was a habitual consumer of large amounts of tea and may have consumed as much as 1 gram of caffeine per day. Caffeine was not tested for in the hospital toxicology. Caffeine has been somewhat associated with cardiac arrhythmias and hypokalemia. However, considering her activities on the night prior to her collapse and the time of her collapse, caffeine toxicity is unlikely unless some sort of pill or supplement containing caffeine was consumed. No family member or friend reports use of any drugs.

c. Did she have a heart attack?

The common term "heart attack" is generally reserved to describe the medical condition of myocardial infarction. Mrs. Schiavo's heart was anatomically normal without any areas of recent or remote myocardial infarction.

Her heart (including the cardiac valves, conduction system and myocardium) was essentially unremarkable except for an apparent incidental finding of focal pericardial adhesions (see cardiovascular pathology report).

d. Was she strangled?

No trauma was noted on any of the numerous physical exams or radiographs performed on Mrs. Schiavo on the day of, in the days after, or in the months after her initial collapse. Indeed, within an hour of her initial hospital admission, radiographic examination of her cervical spine was negative. Specifically, external signs of strangulation including cutaneous or deep neck injury, facial/conjunctival petechiae, and other blunt trauma were not observed or recorded during her initial hospital admission.

Autopsy examination of her neck structures 15 years after her initial collapse did not detect any signs of remote trauma, but, with such a delay, the exam was unlikely to show any residual neck findings. Even bony anomalies would have likely resolved.

e. Did she collapse due to other trauma?

Mrs. Schiavo had no traumatic injuries observed or recorded by her initial treating physicians despite numerous physical exams and radiographs. Contusions, abrasions, recent fractures, and, particularly, healing fractures would have been visualized during her initial months of treatment (via her physical exams and multiple radiographs) and attempted rehabilitation.

f. What other etiologies are possible?

Subtle trauma related to commotio cordis or nontraumatic asphyxia is also possible, but no evidence of these exists. Drugs/toxins not typically detected by hospital toxicology testing are also possible. Also, she had received a fluid bolus prior to the toxicology screen and this has been shown to give false negative urine toxicology results. Other substances have also been shown to interfere with TDX urine toxicology screening. An underlying, undiagnosed cardiac anomaly is possible but diagnostics at that time along with postmortem examination of the heart were negative. Mutations associated with Long QT Syndrome were not detected.

2. Why was a bone scan performed in 1991 and what did the results indicate?

In early 1991, Mrs. Schiavo was undergoing intense rehabilitation at Mediplex in Bradenton. The medical records from that facility clearly indicate that in February 1991, she was experiencing redness and swelling in her knees. During her Mediplex admission (February 5, 1991), in response to this new knee swelling and redness, radiographs were taken that showed severe osteopenia and degenerative changes but no fractures. Her physicians ordered a bone scan to rule out heterotopic ossification (H.O.), infection, or trauma. (H.O. is abnormal growth of bone in extraskeletal soft tissues.) The bone scan was performed on March 5, 1991 at Manatee Memorial Hospital. The bone scan request form listed the history of Mrs. Schiavo as "closed head injury". This is clearly incorrect unless it was meant to imply that the hypoxia was the "injury". The phrase "the patient has a history of trauma" appears to have been derived solely from the erroneous history on the request form. The bone scan showed "focal abnormal areas" including "multiple bilateral ribs, the costovertebral aspects of several of the thoracic vertebral bodies, the L1 vertebral body, both sacroiliac joints, the distal right femoral diaphysis, both knees, and both ankles, right greater than left". Bone scans show the degree of metabolic activity in bone and correlative radiographs described in the bone scan report indicate only a likely compression fracture of the first lumbar vertebral body. No other fractures are noted. It appears that with little or no knowledge of the admitting diagnosis or clinical situation of Mrs. Schiavo, Manatee Memorial staff and radiologists completed the report. This assumption was confirmed after review of DCF report (FPSS#2003-091550) and a deposition taken of the radiologist of record on 11/21/2003. The interpretation made by her attending physicians at Mediplex was early H.O. (a very well known complication of paralysis), and Mrs. Schiavo was treated with Didronel (a common treatment for H.O.). Again, other concurrent radiographs showed no trauma.

Postmortem findings including radiographs and histology support the diagnosis of heterotopic ossification. Postmortem radiographic evidence of H.O. is also seen on the proximal left tibia, left femur, and right ischial tuberosity (adductor magnus area). Degenerative changes and/or fusion were seen in her acromioclavicular joints, hips, right knee, left foot, and pelvis. She also had abnormal bone growth (hyperostosis frontalis interna) on the inner surface of her skull.

This 1991 Mediplex incident was not the first time Mrs. Schiavo had joint problems. She had suffered from swelling of her right knee during her initial hospitalization (early May 1990) and this was treated with "medication and local care". Just like the swelling at Mediplex, this episode of knee swelling occurred during an initial period of physical therapy. Radiography of the knee showed no fracture. The knee was rested and shortly thereafter she was discharged from Humana Hospital-Northside.

As far back as 1991, Mrs. Schiavo was noted to have osteoporosis (a common complication in a patient with immobility and /or paralysis). Compression fractures of the spine and other fractures are common and often incidental complications of this condition, and the compression fracture of the spine was the only diagnosed fracture on concurrent radiographs taken to correlate with the bone scan. A compression fracture of the spine is much more typical of osteoporosis and, possibly, routine handling of the patient than of physical abuse. Multiple radiographs taken during her February-May 1990 Humana Hospital-Northside admission reported no fractures of the spine, and this is the most significant evidence that the L1 fracture of her spine diagnosed in the 1991 bone scan was a later complication of osteoporosis and not a complication of traumatic injury related to her initial insult.

Her postmortem radiographs and autopsy findings confirm the diagnosis of severe osteoporosis. Indeed, the cortical bone of the vertebral bodies was palpably soft. Her 11th thoracic vertebral body was noted to have an endplate fracture in postmortem radiographs. Her first lumbar vertebral body (previously described as fractured in 1991) was noted to have severe osteoporosis.

The other 1991 bone scan findings may also reflect the aftermath of remote intense CPR, infection, bone turnover, artifact or intense physical therapy that was occurring during this period. Indeed, differential diagnoses were offered in the original bone scan report, in the previously described deposition, and in the DCF interview of the involved radiologist.

In summary, any rib fractures, leg fractures, skull fractures or spine fractures that occurred concurrent with Mrs. Schiavo's original collapse would almost certainly have been diagnosed in February 1990 especially with the number of physical exams, radiographs, and other evaluations she received in the early evolution of her care at Humana Hospital-Northside. During her initial hospitalization, she received twenty-three chest radiographs, three brain CT scans, two abdominal radiographs, two echocardiograms, one abdominal ultrasound, one cervical spine radiograph, and one radiograph of her right knee. No fractures or trauma were

reported or recorded. Although in the acute phase, rib fractures may be difficult to visualize, any initial rib fractures would have been going through the healing process during the months of hospitalization, and, with the serial nature of the chest radiographs, callus formation from any healing fractures would likely have been visible. Moreover, hot spots on bone scans of the ribs do not always represent fractures. By far, the most likely explanation for the bone scan findings in Mrs. Schiavo are prolonged immobility induced osteoporosis and complicating H.O. in an environment of intense physical therapy. Without the original bone scan and radiographs from that period, no other conclusions can reasonably be made.

3. Could Mrs. Schiavo eat by mouth?

   The neuropathologic findings, oropharyngeal anatomic findings, and medical records clearly indicate that Mrs. Schiavo would not have been able to consume sustenance safely and/or in sufficient quantity by mouth. In fact, the records and findings are such that oral feedings in quantities sufficient to sustain life would have certainly resulted in aspiration. Swallowing evaluations and speech pathology evaluations repeatedly record that Mrs. Schiavo was a high risk for aspiration and not a candidate for oral nutrition/hydration. Although in her early rehabilitation, she received speech pathology services, she was later repeatedly evaluated and determined not to be a candidate for speech/dysphagia therapy. According to medical records, she had been treated in the past for aspiration pneumonia. Thus, Mrs. Schiavo was dependent on nutrition and hydration via her feeding tube. Claims from caregivers of past oral feedings are remarkable, and, based on the autopsy findings and medical records, these feedings were potentially harmful or, at least, extremely dangerous to Mrs. Schiavo's health and welfare.

   Mrs. Schiavo's postmortem lung examination had findings that could be considered consistent with aspiration of secretions; however, her decline and dehydration over almost 2 weeks could also have played a role in these findings.

4. After her initial collapse, was Mrs. Shiavo given substances to speed her demise or otherwise alter her medical condition?

   In 2003, a former employee of Palm Garden of Largo filed an affidavit with the Court regarding a 1996 incident(s) at that facility. The affidavit detailed incidents the employee reportedly witnessed regarding the care of Mrs. Schiavo. Reports from the complainant regarding incidents at Palm Garden were taken by the Pinellas County Sheriff's Office in 1996 and 2003 (report #96-164479, 03-1183/1, 03-1183/2). The Court and the Department of Children and Families investigated these claims in 2003 (FPSS#2003-091550). Review of the Palm Garden records and the timeline of the allegations do not support the claims made in 2003. Comparison of the 2003 affidavit with the 2005 press interviews and review of the above listed reports is essential in evaluating these claims.

   On March 29th 2004, a Hospice nurse noted apparent injection sites on Mrs. Schiavo's arms, and what appeared to be a plastic needle cap was also found in her room. Reportedly, these were discovered by Hospice shortly after a visit by her parents. In response to this, Michael

Schiavo had her examined at a hospital. Law enforcement investigated and found rational, innocent explanations for the findings. A drug screen was negative and the remaining factors of the case are described in a press release from the Clearwater Police Department (05/14/04) and in a DCF report (FPSS#2004-008306).

There is no evidence to support or the evidence does not support that Mrs. Schiavo was given harmful substances related to these incidents.

In late March 2005, it was alleged in the press that Mrs. Schiavo was being given a morphine drip or otherwise being drugged with morphine to expedite or otherwise ease the dying process. Orders were written and Mrs. Schiavo received morphine sulfate (5mg) suppositories during her final days at Hospice. Her medical records indicate that she received a dose on 3/19/05 and a second dose on 3/26/05. No other morphine treatments were recorded. Her postmortem toxicology showed no trace of morphine in her body. Acetaminophen was detected in her postmortem blood samples at what would be considered therapeutic levels. The finding of acetaminophen is consistent with acetaminophen suppositories ordered by her treating physician, and this drug had no role in her demise. No other drugs were recorded in the medical records nor were any other drugs detected in postmortem toxicology testing. Specifically, according to the Hospice records, no intravenous or intramuscular injections of morphine or any other opiate were ordered or infused.

5. Was Mrs. Schiavo in a persistent vegetative state (PVS)? (See attached neuropathology report)
   PVS is a clinical diagnosis arrived at through physical examination of living patients. Postmortem correlations to PVS with reported pathologic findings have been reported in the literature, but the findings vary with the etiology of the adverse neurological event.

6. What diagnoses can be made in regards to the brain of Mrs. Schiavo? (See attached neuropathology report)
   Mrs. Schiavo's brain showed marked global anoxic-ischemic encephalopathy resulting in massive cerebral atrophy. Her brain weight was approximately half of the expected weight. Of particular importance was the hypoxic damage and neuronal loss in her occipital lobes, which indicates cortical blindness. Her remaining brain regions also show severe hypoxic injury and neuronal atrophy/loss. No areas of recent or remote traumatic injury were found.

7. By what mechanism did Theresa Schiavo die?
   Postmortem findings, including the state of the body and laboratory testing, show that she died of marked dehydration (a direct complication of the electrolyte disturbances brought about by the lack of hydration). The state of her fatty tissue and laboratory findings indicate that she did not starve to death.

8. What was the cause and manner of death?
   Mrs. Schiavo suffered a severe anoxic brain injury. The cause of which cannot be determined with reasonable medical certainty. The manner of death will therefore be certified as undetermined.

It is the policy of this office that no case is ever closed and that all determinations are to be reconsidered upon receipt of credible, new information. In addition to fading memories, the 15-year survival of Mrs. Schiavo after her collapse resulted in the creation of a voluminous number of documents many of which were lost or discarded over the years. Receipt of additional information that clarifies outstanding issues may or shall cause an amendment of her cause and manner of death.

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Chief Medical Examiner

Date: 06/13/05

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