JOHN R. CONTOS, ESQ. (Bar No. 56782)
CONTOS & BUNCH
A Professional Corporation
200 North Westlake Boulevard, Suite 204
Westlake Village, California 91362
Telephone: (818) 707-8887
Fax: (818) 707-8884

Attorneys for Plaintiffs ROBERT MANDLER, ANTHONY MANDLER, MARISA MANDLER and ELIZABETH MANDLER

SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

ROBERT MANDLER, ANTHONY MANDLER, MARISA MANDLER and ELIZABETH MANDLER, Plaintiffs, vs. SIMON LO, M.D., EDWARD FELDMAN, M.D., JEFFREY CONKLIN, M.D., ROBERT McKENNA, M.D., EDWARD PHILLIPS, M.D., ALLAN METZGER, M.D., CEDARS SINAI MEDICAL CENTER, and DOES 1 through 100, inclusive, Defendants.	CASE NO. BC373475 [Hon. William F. Fahey] **PLAINTIFFS' MEDIATION STATEMENT** DATE: November 24, 2008 TIME: 9:00 a.m. MEDIATOR: **Harrison Sommer, Esq.** Trial Date: 1/26/09

PARTIES	**COUNSEL**
Plaintiffs **ROBERT MANDLER, ANTHONY MANDLER, MARISA MANDLER and ELIZABETH MANDLER**	John R. Contos CONTOS & BUNCH A Professional Corporation
Defendant **ALLAN METZGER, M.D.**	Thomas M. O'Neil BONNE, BRIDGES, MUELLER, O'KEEFE & NICHOLS
Defendant, **EDWARD FELDMAN, M.D.**	Christopher P. Wend LA FOLLETTE, JOHNSON, DE HAAS

Defendant, **EDWARD PHILLIPS, M.D. and SIMON LO, M.D.**	Ken Pivo PIVO, HALBREICH, MARTIN, WILSON & AMO
Defendant, **BOSTON SCIENTIFIC**	Matthew L. Marshall MORRIS, POLICH & PURDY
Under Arbitration Agreement as a separate action: Defendant, **Robert McKenna**	Thomas F. McAndrews REBACK, MCANDREWS & KJAR

STATEMENT OF THE CASE:

This claim arises from the death of Nancy Sackett Mandler, a 57 year old mother of three and the wife of plaintiff, Robert Mandler, who was a successful television screenwriter and a professor at both USC School of Cinematics and the American Film Institute.

For several years prior to her death Mrs. Mandler suffered from a non-specific, auto-immune collagen-vascular disorder for which she was under medical care by defendants.

In **July, 2004,** she suffered iatrogenic perforation of the esophagus by defendant Dr. Robert McKenna, during a surgery to treat esophageal dysfunction. As a result, Dr. McKenna placed an Ultraflex stent, manufactured by defendant Boston Scientific, in Mrs. Mandler's esophagus. Boston Scientific has taken the position that Dr. McKenna placed the wrong type of stent, one *not* intended for removal and he should have known that when he attempted to remove the stent three months later, he would be unable to do so.

Over the next eighteen months defendants, Dr. Robert McKenna and Dr. Simon Lo, performed **seven unsuccessful surgical procedures** attempting to remove the Ultraflex stent.

As a result of the perforation and the succession of surgeries to remove tissue and the stent, Mrs. Mandler developed an **aortoesophageal fistula**, which is a direct communication between the esophagus and aorta, a well-recognized and life-threatening complication of esophogael stents. Symptoms include hematemesis, difficulty swallowing and chest pain. Despite these hallmark symptoms, defendants failed recognize and diagnose an aortic fistula.

When hospitalized for hematemesis on March 27,2006, for ***five* days**, under the care of defendants Feldman, Lo and Phillips, **no diagnostic studies were ordered or performed to determine the source of bleeding or the condition of the esophagus**. No ultrasound, upper GI, CT, MRI or angiography was performed, even though Mrs. Mandler required nineteen units of blood products in the first 4 days of admission and 24 units in her last four hours of life.

Plaintiffs also contend that the defendant physicians involved in Mrs. Mandler's care did not have the requisite expertise in esophageal motility disorders and surgery, resulting in an incorrect diagnosis, the incorrect treatment, iatrogenic esophageal perforation, use of the incorrect stent, incorrect positioning of the stent which could not be removed, followed by repeated and unsuccessful attempts to remove the stent, resulting in repetitive trauma and ultimately the development of a aortic fistula which defendants failed to recognize.

Defendants proceeded with an esophagectomy, unprepared for aortic repair, without placing Mrs. Mandler on cardiac bypass, without the equipment and devices necessary to effect

repair of the aortic fistula and without a cardiovascular surgeon present in the operating suite. The aorta ruptured, Mrs. Mandler exanguinated and died within fourteen minutes.

HISTORY OF ESOPHAGEAL DISEASE AND MEDICAL CARE:

For five years prior to her death, Mrs. Mandler was seen by physicians for lethargy and sporadic aching, swelling, inflammation in her rib cage, left eye, nose, finger, neck, shoulders and legs.

In **September, 2002,** while under the care of defendant, Dr. Alan Metzger, Mrs. Mandler was referred to a rheumatologist, Dr. Louie, at UCLA. Dr. Louie felt that, based on her symptoms, elevated ESR and a positive rheumatoid factor, she had features of an autoimmune disease collagen-vascular disease, Relapsing Polychondritis, but could not confirm the diagnosis without biopsy. She was prescribed immunosuppressive therapy with Prednisone and Methotrexate.

In **March, 2003,** Mrs. Mandler developed severe respiratory symptoms of hoarseness and a severe cough, felt to be Methotrexate lung toxicity, after which the Methotrexate was discontinued and the Prednisone was tapered to prevent the deleterious effects of chronic steriod use.

Although her respiratory condition improved dramatically during the next twelve months, Mrs. Mandler developed gastric reflux and esophageal dysmotility for which Dr. Metzger referred her to defendant, Edward Feldman, M.D., a gastroenterologist.

Diagnosis of Esophagael Motility Disorders:

Standard diagnostic procedures are necessary to differentiate between symptoms and causes of various digestive diseases and to plan treatment. In Mrs. Mandler's case, the GI workup by defendants Feldman and McKenna, although incomplete, was completely negative for evidence of disease, reflux or Achalasia and the only finding was increased tone of the LES.

Standard Diagnostic Procedures	**GI workup of Nancy Mandler:**
1) an upper GI series, esophagogram and esophagoscopy with biopsy.	1) **July 26, 2004: Upper GI and Small Bowel Study**: showed a **normal** esophagus, without ulcerations or erosion, mesenteric thickening. Findings of Dr. Hamlin indicated that although a contracted LES suggested possible Acalasia, the motor activcity in the body of the esophagus mitigates against this diagnosis. He noted a small hiatal hernia, without reflux. **July 30, 2004: GI endoscopy and biopsy**: both gross and microscopic examination was negative for inflammation, atypia, dysplasia, atrophy, metaplasia, neoplasia, malignancy, bacterial or parasitic disease. There was no evidence of erosion, ulcerations or vascular abnormalities.
2) A Bernstein test (to determine if chest or back pain is coming from the esophagus or acid reflux)	2) Not Done
3) Breath Test-Breath-glucose and lactulose	3) Not done until 12-5-05; positive for bacteria.
4) 24 hour pH monitoring. Ambulatory 24-hour pH monitoring is the criterion standard in establishing a diagnosis of GERD - gastroesophagael reflux disease with 96% accuracy.	4) Not Done. The extent and severity of gastric reflux, as well as the effect of anti-reflux medications, cannot be determined in the absence of this test.
5) Esophageal manometry/Esophagael Function Test	5) **July 26, 2004: An Esophagael Function Test** indicated increased tone of the LES, but **normal** motility of the esophagus
6) 24-hour Esophageal Manometry	6) Not Done

7) Electrogastrography, used to diagnose and study stomach rhythm as a cause of nausea.	7) Not Done
8) Endoscopic ultrasound	8) **August 11, 2004: Endoscopy** Mature squamous cells with fragments of smooth muscle, negative for atypia
9) **Pharmacologic therapy** with anticholinergics, calcium channel blockers, vasodilators, anxiolytics, injections of botulinin toxin	9) Not Done

Achalasia

Presumed by defendants to be causing Mrs. Mandler's symptoms, Achalasia is a predominantly neuropathologic process causing loss of nerve cells from the esophagus at the lower esophageal sphincter (LES), resulting in failure of the LES to completely relax, with progressive inability to swallow solids and liquids.

In patients with true Achalasia, mucosal changes due to chronic irritation and food stagnation include erythema, friable mucosa, ulceration, and candidal infection. The LES is closed tightly and does not open with air insufflation, but the endoscope can pass into the stomach with gentle mechanical pressure.

Treatment Options of LES dysfunction include:

1) Drug therapy with Anticholinergics, calcium channel blockers, vasodilators, anxiolytics, injections of botulinin toxin;

2) pneumatic dilation of the esophagus at least twice and if repeated dilation is unsuccessful,

3) surgical intervention, known as

a) Heller Myotomy, a procedure which severs muscles at the gastroesophageal junction, allowing the valve between the esophagus and stomach to remain open. This procedure also causes unremitting gastric reflux as the esophagael sphincter is patulous and unable to effectively close. Therefore,

b) fundoplication (the gastric fundus, upper portion of the stomach is wrapped, or plicated, around the inferior part of the esophagus and stitched in place, reinforcing the closing function of the lower esophageal sphincter, and

c) pyloroplasty - usually unnecessary -(opening of the end of the pylorus in the lower portion of the stomach, so that stomach contents can empty into the duodenum (small intestine).

Defendants also failed to consider that esophageal motility disorders are common among patients with anxiety and depressive disorders. Contraction abnormalities reflect a functional motor impairment, which can be related to the effect of psychiatric medications. Upper endoscopy is usually normal in these patients, as was Nancy Mandler's. Defendants did not investigate the causal effect of psychiatric medications prior to proceeding with surgical intervention.

LES tension was the only objective finding, based on diagnostic testing performed by defendants, the cause of which was probably pharmacologic. Even if psychiatric medications were not the cause of the LES dysfunction, proceeding with a Heller Myotomy was, at the very least, premature, and probably was not only unnecessary, but caused the cascade of medical complications that led to the death of Nancy Mandler.

It should be noted that her upper GI symptoms were not longstanding and there was no objective or destructive evidence of disease, such as erosions, atypical cells, inflammation or other pathology that justified the performance of a surgical procedure that had small, but significant inherent risks.

She was not described as malnourished, she had not sustained any significant weight loss and none of the conservative measures of treatment, such as medication, biofeedback, speech therapy for swallowing rehabilitation, diet changes or other non-invasive were ever attempted by defendants. It cannot be refuted that proceeding with surgical intervention, without first exhausting conservative treatment, in a patient with a paucity of objective evidence of disease was below the standard of care.

Notwithstanding these facts, in **August, 2004**, Mrs. Mandler was referred to defendant, Robert McKenna, M.D., for evaluation and treatment of presumed Achalasia. Dr. McKenna noted that Mrs. Mandler's symptoms were not typical Achalsia but felt she had "ill-defined collagen vascular disease." He recommended a Heller Myotomy and a biopsy for diagnosis of collagen vascular disease which was performed on **August 18, 2004.**

The esophagael wall biopsy was negative for atypia or inflammation, negative for epithelium and all immune reactants were negative in all locations, based on the pathology report, dated August 24, 2004.

Plaintiffs' experts will testify that defendants Feldman and McKenna were negligent in their diagnosis and care of

Nancy Mandler prior to and including the performance of a Heller Myotomy on August 18, 2004, that the diagnosis of Achalasia was incorrect, the performance of a Heller Myotomy was unnecessary and that the perforation which occurred as a result of that procedure was a significant factor leading to her death on March 31, 2006.

Perforation of the Esophagus

Two days after discharge Nancy Mandler developed fevers and severe left-sided chest pain. A barium swallow study showed a leak in the distal esophagus. A thoracotomy, performed by Dr. McKenna, found 800cc of murky fluid with barium. A 2mm opening in distal esophagus was found at myotomy site, which was sutured, but continued to leak. A CT showed a subphrenic abscess.

Dr. McKenna excised the perforation, creating an elliptical incision, sutured and placed an intercostal muscle over the area.

A repeat barium swallow showed recurrent leak. Although attempting a second repair and consulting an esophagael surgeon would have been a reasonable next step, Dr. McKenna elected to perform an esophagael stent placement, using a Boston Scientific Ultraflex stent, by laparotomy with a feeding jejunostomy and gastrostomy.

Defendant Boston Scientific is expected to present expert testimony that the Ultraflex stent, which was uncovered on its distal and proximal ends, was intended for palliative use in patients with terminal malignancies of the esophagus. This stent was not intended for temporary use in the treatment of benign

(non-malignant) conditions.

However, a review of the literature indicates that the Ultraflex stent has been used for treatment of esophagael perforations, strictures and other benign conditions. However, because the ends are uncovered, tissue ingrowth will occur if the stent is not removed shortly after placement. Therefore, monitoring of the patient's esophagus and the development of inflammation or tissue growth is critical if stent removal is planned.

Drs. McKenna and Feldman took no steps to monitor Mrs. Mandler's esophagus for ingrowth of the stent at any time after placement of the stent, until its attempted removal in December, 2004.

Post-esophagael perforation:

Following placement of the stent for perforation of the esophagus, Mrs. Mandler required a tracheostomy and bronchoscopy with lavage and she developed ARDS, Acute Respiratory Distress Syndrome, with respiratory failure, hypoxemia, with diffuse alveolar damage. She remained hospitalized through mid-November, 2004, and was discharged with a gastric tube in place.

December 8, 2004: Dr. McKenna attempted removal of the stent via flexible esophagoscopy. His findings indicate a noral appearing proximal esophagus. The stent was located in the distal 7cm, *it was not attached on the right side and the right lateral wall of esoph was densely adherent on the L side.* Multiple attempts were made to free the stent. The distal 3cm of stent was removed as it tore loose from main stent.

December 9, 2004: An esophagram was performed which showed normal

peristalsis in upper 2/3 of the esophagus, but on repeated swallow **a punctate spot of barium was seen outside esophagus, immediately lateral to the upper portion of the esophageal graft on the left, and pulsated with heartbeat, consistent with mediastinal location.**

On **February 8, 2005,** Mrs. Mandler was seen by Dr. Feldman. He noted she complained of mouth burning, substernal epigastric burning, that she could not lay flat and slept or rested on a wedge. Her appetite was decreased, she was able to swallow but she was very unhappy about the stent placement. Dr. Feldman noted most of her symptoms were related to the stent. He discussed her available options, because the stent could not be removed intact, perhaps they could try an esophagoscopy to pull the pieces apart or do esophagectomy. She was to consider these options.

April, 2005: When seen by Dr. Feldman, Mrs. Mandler experienced rare dysphagia (difficulty swallowing), had occasional left upper quadrant cramps and dark drainage from the G-tube site.

July 29, 2005: an upper GI showed prominent reflux throughout stent.

October 20, 2005: Mrs. Mandler was seen by Jeffrey Conklin, M.D. an esophagael specialist at Cedars-Sinai. At the time of this exam, Mrs. Mandler had epigastric and left upper quadrant pain or tenderness that was constant, worse by wearing tight clothes and worse in certain positions. She also had a band-like pain from lower back to lower ribs anteriorly, worse with belching or hiccoughs. She complained of awakening with a burning tongue, had prominent symptoms of heartburn and acid reflux, but under

felt it was under control with pump inhibitors. She had a new complaint of regurgitation, usually after meals, and vomiting of brown mucous when she bent over. **Experts will testify this was likely evidence of bleeding into the stomach from the esophagus.**

On examination it tenderness and an area of hyperesthesia was noted over the lower thorax, reproducible with flinching to light touch. The greatest pain was a small area just below ribs over rectus muscle, with diffuse tenderness of abd wall.

A recent Upper GI showed narrowing of the stent and a CT of chest and abd revealed a stent with thickening of the wall of the esophagus. Dr. Conklin indicated Ms. Mandler's pain was neuropathic involving innervation of the diaphragm, likely producing hiccoughs. He noted the CT had not identified pathological process causing this, but symptoms were likely the result of the stent, making the entire lower esophageal sphincter entirely incompetent and that her problems with gastric emptying were due to a combination of achalasia and vagal nerve injury.

He recommended endoscopy to see if stent was widely patent and to look for signs of inflammatory injury.

November 3, 2005: Dr. McKenna performed removal of the tracheostomy and esophagoscopy and lavage to evaluate stent and clear the stent.

November 22, 2005: An upper endoscopy was performed by Dr. Lo and Dr. Conklin. The upper esophagus was normal and the Ultraflex stent was located in distal 6-8 cm of esophagus. The top expanded portion of the stent was fully embedded in esophagus. 1 cm below top edge, the mesh was exposed on one side and a *mass*

appeared to be mechanically altering and protruding through a part of the mesh. Ulcerations noted under that part of the stent. The Distal portion of stent had a luminal caliber of 12-13mm and the lower end of stent had flipped backward on itself into area of GE junction and gastric folds were noted to prolapse back into esophageal stent lumen.

December 6, 2005: Mrs. Mandler was seen by Dr. Conklin. He noted that recent endoscopy revealed the stent to be permeated and encased by esophagael tissue. The esophagael mucosa and wall were ulcerated over part of the stent indicating significant acid reflux. A recent gastric emptying study demonstrated delayed emptying and lactulose hydrogen breath test revealed small bowel bacterial overgrowth. He noted the significant trauma to the mediastinum had probably caused traumatic vagotomy and the symptoms of nausea and motility dysfunction were likely caused by damage to the vagal nerve.

January 10, 2006: Dr. Lo performed another upper endoscopy, attempting to remove the stent with Argon Plasma therapy (gas with electrocoagulation). The stent was treated with Argon Plasma quite extensively, but only partial destruction of the stent was made and the procedure terminated. He found a 1.2cm opening in the lower esophagus and ulceration, surrounded by "lumpy, bumpy tissue with a net-like appearance consistent with tissue growing through stent, this in spite of patient supposedly having a covered Ultraflex stent. Below the gastroesophagael junction the stent was folded up backwards and extended into stomach 2-3cm with the gastric wall touching the stent. The stent element was cauterized but only several wires were cut. He noted it was not

possible to cut all wire elements to allow endoscopic removal. Forceps were used to grab stent to move it, causing stent to flip back to natural position, but doubling the length of stent protruding into stomach. He was unable to trim the stent but tissue was cauterized in the middle of the stent.

January 20, 2006: Dr. Lo performed an esophagogastroduodenoscopy and again attempted removal of stent with argon plasma therapy. Half of the stent was found protruding into stomach. The stent was treated with argon plasma and olympus wire cutters were used to cut the stent circumferentially.

February 28, 2006: Mrs. Mandler was seen by Dr. Feldman who noted she suffered from spasms, dysphagia,she was barely able to tolerate any solids and her intake was mostly full liquids. She complained of increased emesis that was unpredictable and occasionally awakened regurgitating.

March 16, 2006: Mrs. Feldman was sent to the Emergency Room by Dr. Feldman for evaluation of hematemesis. She had been found to have moderately severe anemia with HGB of 7.4 (normal=11.6-15.4)indicating she had lost a significant volume of blood due to internal bleeding from the stent. She was lightheaded and dizzy, with melanotic stools. She was admitted and received transfusions.

March 17, 2006: An upper endoscopy was performed by Dr. Lo for stricture dilation, polypectomy, argon plasma coagulation therapy and **to identify the source of bleeding.** Nodular tissue impeding endoscope passage was removed and cauterized with Argon.

Again, the upper esophagus was normal but the lower lumen was narrow in a shelf-like fashion and despite balloon dilation at 12mm, the endoscope could not pass, although hemorrhagic tissue was seen beyond that point. A pediatric endoscope was passed through. No perforation was noted, nodular tissue was protruding from one side of the inlet and beyond 34cm the lumen was wider. It appeared that bleeding came from the top part of stricture site. The esophagus was dilated to 13.5, a snare was used to remove polypoid reactive tissue. Oozing was noted with filaments of the stent identified. The area was cauterized with argon and re-dilated to 15mm. The mucosal lining

was cauterized until hemostasis was achieved.

Experts will testify that the aggressive nature of this procedure, extensive cauterization with trauma from stent manipulation exacerbated the ulcerations and fistula formation. Despite the significant blood loss, no CT, MRI, angiography or ultrasound exam was performed to identify the source of bleeding, which could not be explained by oozing seen in the esophagus and to rule out fistula formation. The pain and hematemesis were hallmark symptoms of fistula formation that went unrecognized by defendants. Had the fistula been identified during this hospitalization, Mrs. Mandler could have undergone vascular surgery with grafting or a Blakemore tube to repair the defect and esophagectomy before her condition became unstable. Failure to recognize the symptoms of a fistula, given the history of perforation, abscess formation, stent placement and perform testing necessary to diagnose a fistula was below the standard of care and a significant factor leading to her death.

Despite the ominous symptoms of pain and hematemesis, Mrs. Mandler was discharged on March 21 without any further diagnostic procedures.

March 27, 2006: Mrs. Mandler was admitted to Cedars by ambulance for multiple episodes of gross hematemesis. She received transfusions and was admitted.

She was seen by Dr. Feldman on March 28, 2006, who noted she complained of "gnawing epigastric pain." His admission History and Physical Examination indicates her gastrointestinal

bleeding is "presumably from esophageal erosions." He consulted with defendant Dr. Simon Lo and a decision was made to perform another endoscopy to reexamine the site of the recent surgery.

There were no further episodes of hematemesis on March 27 and 28.

March 28, 2006: At 8:30 p.m. Dr. Lo performed another esophagael gastroduodenoscopy. His report indicates he found "lumpy-bumpy changes, bleeding in the general area. The distal 5mm was smooth, slightly narrow and there was tissue covering the straight part of the old Ultraflex stent." Dr. Lo removed additional hypertrophic tissue and noted an area of diffuse bleeding without any definite pinpoint area and again cauterized the tissue with argon plasma. He apparently consulted with Dr. Feldman and **a decision was made to place a second removable, totally covered stent to stop bleeding, provide tamponade and reduce acid washing of the area.**

No diagnostic studies were ordered.

The chart indicates Mrs. Mandler had **seven** episodes of hematemesis between Dr. Lo's procedure on the evening of March 28 and the morning of March 29, 2006.

Dr. Phillips

On **March 29, 2006,** defendant, Dr. Phillips was asked to evaluate Mrs. Mandler regarding a possible esophagectomy with colon interposition. A consult by Dr. Phillips following an examination of Mrs. Mandler notes she continued to bleed at a

low rate, **indicating he was unaware of the seven episodes of hematemesis in the previous ten hours.** Dr. Phillips recommended an esophagectomy with colon interposition, a procedure in which the esophagus is removed and replaced by a piece of colon. Preparation for this procedure would include a mesenteric angiogram, if not done recently, **also indicating he was unfamiliar with her history,** pulmonary function studies and a colonoscopy. He indicated this procedure could be done electively.

Although the dictated report bears the name of Dr. Phillips as its author, a chart entry, dated March 29, 2006, indicates it was Dr. Lyass who reviewed the chart, examined the patient and dictated the consult. Likewise, chart entries on March 30 and 31 indicate "Surgery for Phillips" signed by Dr. Lyass. In fact, Dr. Phillips did not sign a single order or progress note in Mrs. Mandler's chart.

In response to discovery requests, Dr. Phillips produced text messages which document communications he had regarding Mrs. Mandler. These messages document that he had delegated Mrs. Mandler's care to a surgical resident, Dr. Sergey Lyass on **March 29, 2006 at 12:16 p.m.**

"1.5 years ago wife of chin chin owner. Achalasia. **McKenna perforated.** Stented which helped. Continuous problem as stent is embedded. More problem with pain and swallowing. Fulgurated. Last night scope - cut lumen

through hypertrophied tissue. Put coated stent in. Still bleeding."

30 hours later:

March 30, 2006, 6:09 p.m. -, Dr. Phillips inquires via text message to Dr. Lyass:

"How's esoph bleeder?"

March 30, 2006, at 18:21:17 (6:21pm) Dr. Lyass responds:

"Just vomited blood. Getting blood I asked to call Lo

if he wants to do something. Any thoughts?"

March 30, 2006, 8:21 p.m. (Two hours later)Dr. Phillips responds:

"did you get my last email re her Hct and stability?

Are Surgery residents involved. Is she in ICU? Ed."

March 31, 2006 13:41:53 (1:41pm) **(16+ hours later)** Dr. Lyass' nurse Christina Kim sends a text to Phillips:

"Large bleed and patient almost code and unresponsive.

Please call x34356- Dr. Miasaki ."

Dr. Miyasaki is an intern.

March 31, 2006 2:29 pm, (another hour) Phillips to Dr. Clark Fuller and McKenna:

"She's in the 7th floor sap room 58. She's definitely

needing surgery and I'd **like** one of you to come by.

Maybe you guys can **start** and **I'll come in** as

I'm starting a case 7th floor room 1 now. Ed." (Emphasis added)

March 31, 2006 2:31 pm, Phillips to Dr. Simon Lo:

"I'm taking her to surgery today. Ed"

This statement is not exactly accurate as Dr. Phillips was intending to start surgery on another patient and had asked Drs. McKenna and Fuller to take the patient to surgery.

March 31, 2006 2:44pm, Dr. Lo to Phillips:

"thank you very much. She needs you.

I just worry that her stent is causing a great

deal of reactions in the mediastinum."

Apparently Dr. Lo still has not considered the possibility of a fistula.

March 31,2006 2:46pm, Phillips to Lo:

"Bad news"

The meaning of this last entry is unclear, but is followed by a note to Dr. Feldman, below, indicating only McKenna was going to handle the surgery.

March 31, 2006 2:48pm, Phillips to Feldman:

"I lined her up for surgery and asked McKenna

to see her. Ed"

Dr. Phillips' decision not to take part in Mrs. Mandler's surgery is confirmed by the chart note of his resident, Dr. Lyass, who

stated at 1:30 p.m.:

"Pt continue to bleed. Required more transfusion.

Ht ↓(down) to 25. Pt needs esophagogastrectomy. Colon interposition is the preferred method of reconstruction, but in the acute setting gastric pull-up will be the right and safe choice.

Await consult of Dr. McKenna and Dr. C. Fuller from thoracic service.

Lyass"

This note by Dr. Lyass makes it clear that Dr. Phillips was not going to be involved in the surgery and further, that an esophagael-aortic fistula as the source of bleeding had still not been considered.

It also appears, based on the chart entries of Dr. Lyass and Dr. Phillips' test messages, that, other than a dictated consult, Dr. Phillips had no contact with Mrs. Mandler and, indeed, ***Dr. Phillips never even examined her*** as the March 29, 2006, note indicates it was Dr. Lyass who performed the examination, chart review and consult.

The failure of Dr. Phillips to personally evaluate and follow Mrs. Mandler was not only a critical departure from the standard of care, but also misled the Mandler family as well as Mrs. Mandler's other physicians into believing that she had been evaluated and was being followed by an experienced surgeon who would be involved in her care.

Dr. Lyass attended the Second Moscow Pirogov State Medical Institute. He became licensed to practice medicine in California on August 14, 2002, and was a resident in surgery at Cedars-Sinai when he was caring for Mrs. Mandler. None of his chart notes, order, nor the consult he dictated for Dr. Phillips discuss the probable source of the bleeding or that an esophagael fistula had been considered. Although a CT angiography was mentioned, it was never ordered or performed, nor any other diagnostic studies, at any time during the five days Mrs. Mandler was a patient.

Dr. Phillips' apparent abandonment of Mrs. Mandler following his consult of March 29, 2006, his failure to accurately assess Mrs. Mandler's medical condition, lack of familiarity with her medical history and failure to consider and diagnose an esophageal-aortic fistula had been a mystery until a recent production of documents by counsel for Dr. McKenna. Chart notes not contained in records previously obtained from Cedars Sinai and text messages from counsel for Dr. Phillips clarified the unfortunate sequence of events.

Dr. McKenna:

The operative report, dictated five days ***after*** Mrs. Mandler's death, states that Drs. Phillips and Feldman "felt that a tiny ulceration in the esophagus was the etiology of the bleed, but upon arrival to the ICU, Dr. McKenna "felt this was an emergency and that she needed to be brought to the Operating Room immediately. This could potentially be a fistula from the

aorta." The sequence of notes indicates this was written after Mrs. Mandler was taken to the OR at 2:30 p.m.

However, if Dr. McKenna did believe Mrs. Mandler had an aortoesophagael fistula, he commenced surgery completely unprepared to effectuate aortic repair, without any cardiopulmonary bypass equipment available and without the requisite experience of a cardiovascular surgeon to handle this emergency.

The records do not support Dr. McKenna's claim that he suspected an aortoesophagael fistula prior to commencing surgery for the following reasons:

1)The Pre-Anesthesia assessment of Dr. Durra is the first mention of a possible esophagael-aortic fistula, that she is an anesthesia risk IV, very high risk.

2) The Operative record indicates the surgery lasted for more than an hour and a half, from 1:30 to 1610. If an aortic fistula was truly suspected, it defies immagination that Dr. McKenna would take the time to perform a flexible bronchoscopy, flexible esophagoscopy and perform an esophagectomy before looking for the aortic fistula.

3) Dr. McKenna's report, dictated April 5, 2006, states:

> "...there were extensive adhesions in the chest. These were bluntly taken down quickly in an effort to get behind the lung and put pressure on the aorta.... There

were **two brief episodes of cardiac arrest. With this resuscitation with CPR and with fluids and drugs the heart came back. Blood pressure was in the 80-90** range. The dissection and the operation continued... A Median sterotomy was performed. The pericardium was opened anteriorly and laterally. There was a 7cm length of the esophagus that was inflamed, densely adherent to the aorta. Once this was mobilized off the aorta...there was a 2cm transverse fistula in the aorta. This was sutured with 4-0 Prolene. Despite these efforts the patient **continued to have very unstable blood pressure. She arrested again** and at this point the heart muscle was flacid. Resuscitation was not successful. **The cardiac surgery service team was consulted. They felt this was not salvageable and resuscitation was discontinued.**

4) In fact, this recitation is not supported by the records which indicate that:

1430-1545 Mrs. Mandler's blood pressure remained a constant 100/45-50 from 1430 to 1545 with sinus tachycardia.

1530 Massive blood loss was noted.

1540 Cardio pulmonary bypass perfusion support was called for at more than **one hour** after the surgery started.

1545 Dr. Kass, cardiovascular surgeon was called

1550 Dr. Raissi, Director of the Thoracic Aortic Surgery Team,

arrived at 1550.

1556 (14 minutes prior to her death) Mrs. Mandler went into ventricular fibrillation, and was defibrillated.

1558 (twelve minutes prior to death) Ventricular fibrillation occurred again she was defibrillated.

1600 Cardiopulmonary bypass was started after the arrival of Drs. Kass and Raissi.

1600 Death was pronounced.

4) It is clear from the above sequence of events that Dr. McKenna did not anticipate an aortic fistula, the repair of which requires cardiopulmonary bypass, which wasn't even requested until one hour after the surgery began.

5) If Dr. McKenna was concerned about an aortic fistula, which is universally understood to be a catastrophic event, he would have been prepared to perform deep hypothermic circulatory arrest (DHCA), place *in situ* allograft for aortic replacement in association with subtotal esophagectomy, cervical esophagostomy, ligation of the abdominal esophagus, gastrostomy, and jejunostomy.

6) There is no evidence that Dr. McKenna or any of the physicians involved in Mrs. Mandler's care, recognized the characteristic Chiari's triad features of aortoesophageal syndrome, including chest pain and sentinel hematemesis of red blood followed at a variable interval of time by exsanguinating hematemesis.

7) An esophagael contrast study was never performed.

8) Dr. McKenna performed a midline sternotomy which requires cardiopulmonary bypass to allow aortic repair and to enable displacement of the heart for resection of the esophagus by the transpericardial route. The procedure of choice for aortic fistula repair is a left thoracotomy.

9) Neither Endovascular graft, allograft nor Blakemore tube were requested, noted in the records or on the billing, indicating repair of the aorta was not contemplated.

10) Finally, aortic repair is always done first, not after esophagoscopy, bronchoscopy and esophagectomy.

It is unfortunate that Dr. McKenna did not, in fact, consider the possibility of an aortoesophagael fistula prior to the surgery which began at 230 p.m. on March 31, 2006. Not a single chart notation of Dr. McKenna, nor any other physician involved with Mrs. Mandler's care prior to surgery, bears any mention of the possibility of an aortic fistula. The first time an aortic fistula is mentioned is at 2:30 p.m. in the Pre-Anesthesia Assessment by Dr. Durra.

Diagnosis of Esophagael-aortic fistula:

Cedars Sinai has a **Thoracic Aortic Surgery Program** that boasts "state-of-the-art medical and surgical approaches to aortic disease", including "state-of-the-art diagnostic testing required for accurate measurement and assessment" and that "even the most difficult aortic conditions in high-risk patients yield

to world-class innovation and the expertise in aortic surgery at Cedars-Sinai's Thoracic Aortic Surgery Program."

It is axiomatic that if Dr. McKenna thought Mrs. Mandler had an aortic fistula, he would have notified the Thoracic Aortic Surgery Department prior to commencing surgery.

Liability of Defendants:

Dr. Metzger:

1. Failure to undertake appropriate testing necessary to accurately diagnose decedent's esophagael motility disorder;

2. Failure to refer Mrs. Mandler to a physician who specialized in esophageal dysfunction and treatment of esophagael motility disorders. Although referral to such a specialist was considered it was never carried out;

3. Failure to follow Mrs. Mandler closely to ensure that the stent was immediately removed when no longer necessary, to avoid its becoming inbedded with tissue overgrowth;

4. Failure to have an understanding of achalasia, relapsing polychondritis and other related auto-immune disease affecting the esophagus sufficient to follow his patient.

5. Failure to consider psychiatric medications as the cause of the lower esophagael hypertension and adjust medications in an attempt to resolve the symptoms prior to consideration of surgial interventioin.

6. Failure to recognize symptoms of an aortoesophagael fistula;

8. Failure to order any testing necessary to determine

the source of gastric bleeding.

Dr. McKenna: Although Dr. McKenna has been severed from this action and his liability is being determined via arbitration, his involvement and share in liability relative to the other defendants follows:

1. Iatrogenic perforation of the esophagus in August, 2004, while performing a Heller Myotomy, which procedure was not indicated, as diagnostic testing was not sufficient to accurately diagnose Mrs. Mandler's condition;

2. Placement of a partially covered Ultraflex stent, which was contraindicated in patients with benign disease, because it cannot be removed;

3. Lack of knowledge regarding stents and risks inherent in their use; incorrect placement and positioning of the stent, obstructing the esophagael sphincter, which caused chronic severe reflux due to mechanical obstruction of the sphincter by the stent;

4. Performance of multiple surgical procedures, resulting in damage to decedent's esophagus and vagal nerve, exacerbating the effects of the esophagael motility disorder;

5. Damage of the stent covering during multiple attempts at removal, which resulted in gastric acid damage to both the stent and the esophagus;

6. Failure to recognize the symptoms of an aortoesophageal fistula;

7. Failure to order any testing necessary to determine the source of gastric bleeding;

8. Failure to refer Mrs. Mandler to a specialist in esophageal motility disorders prior to and after the surgical procedure performed in July, 2004.

Dr. Feldman: Dr. Feldman supervised Nancy's care after the stent was placed in January, 2005, through the time of her death. He ordered diagnostic testing, medication, saw her on a regular basis and was the attending physician during her hospitalizations. Dr. Feldman was responsible for:

1. Failure to refer Mrs. Mandler to specialists in esophageal motility disorders. Without adequate evaluation of her symptoms, an accurate diagnosis could not be made and treatment could not be planned.

2. Failure to order and perform adequate diagnostic testing prior to performing serial procedures on a patient with unknown collagen-vascular and neuropathic disease.

3. Failure to ensure adequate treatment of the esophageal perforation was carried out, including placement of the correct stent, ensuring correct placement and that the stent was removed as soon as possible to avoid its becoming inbedded with tissue overgrowth;

4. Failure to have an adequate knowledge base regarding stents and risks inherent in their use, likelihood of fistula as a complication of stent placement and failure to recognize the symptoms of an aortoesophagael fistula; failure to order any testing necessary to determine the source of gastric bleeding;

5. failure to diagnose an esophagael fistula.

Dr. Lo:

1. Failure to have adequate knowledge of stents and risks inherent in their use;

2. Repeated attempts to remove the stent, causing damage to decedent's esophagus and vagal nerve, exacerbating the effects of

the esophagael motility disorder;

3. Caused damage to the stent covering during attempted removal, permitting gastric acid to cause additional damage to both the stent and the esophagus;

4. Failure to recognize the symptoms of an aortoesophagael fistula;

5. Failure to order or recommend testing necessary to determine the source of gastric bleeding;

6. Failure to diagnose an esophagael fistula.

Boston Scientific:

1. Placement of partially covered stents on the market, without adequate education, instruction and warnings to physicians utilizing those stents in persons such as decedent, who had an esophagael motility disorder.

2. The defective lining of the Ultraflex stent allowed severe gastric reflux to damage the patient's esophagus and the stent, leading to ulcerations and, eventually, the development of an aortoesophagael fistula. The stent carried inadequate warnings regarding the inherent risks of stent placement in patients with benign disease.

Edward Philips, M.D.

1. Following evaluation of Mrs. Mandler for "elective" colon resection and esophagael reconstruction, defendant abandoned Mrs. Mandler and delegated her care to a surgical trainee who lacked the requisite experience and education to follow a patient such as Nancy Mandler.

2. Failure to consider and recognize the development of ulcerations and fistulas as a result of the stent placement.

3. Failure to consider the probability of an aortoesophagael fistula, given a history of multiple attempts to remove the stent, damage to the stent, tissue damage and trauma to Mrs. Mandler's esophagus on multiple occasions in the preceding two years, with recent symptoms of GI bleeding and bloody emesis increasing in frequency and amount, severe anemia and hypotension.

4. Failure to order diagnostic testing sufficient to diagnose the source of bleeding prior to commencing an esophagectomy procedure.

5. Failure to adequately evaluate Mrs. Mandler and failure to order diagnostic testing sufficient to diagnose the cause of her hemodynamic instability.

6. Failure to personally examine and evaluate a critically ill patient, delegating her care to an inexperienced surgery resident.

ECONOMIC LOSSES:

For three decades, Nancy Sackett Mandler was a television writer and producer of movies-of-the-week, dramatic specials, pilot and feature films for NBC, ABC, PBS, Disney, CBS, Warner Brothers and Lifetime Television. She was best known for *Skyward*, a Hallmark Special directed by Ron Howard, and *The Ring*, based on a Danielle Steele novel. She also wrote the ABC

mini-series, Charles and Diana, and Beverly Hills Madam which were two of broadcast networks' highest rated movies. My Darlin' Clementine, written for Showtime Theater, won the ACE Cable Award for outstanding writing. She wrote a novel, *That Day Again,* and three plays that were performed at the Odyssey Theater. She was a professor at both the USC School of Cinematic Arts and the American Film Institute. Following her death, the Nancy Sackett-Mandler Memorial Scholarship was established and was regarded was one of the highest honors that could be awarded by the American Film Institute. She established the Storytellers series for inner city children and was a member of several writers' associations.

Prior to the iatrogenic injury by Dr. McKenna, she was earning $45,000 per year and loss of her earnings is estimated at $450,000, plus cost of living and merit increases.

Dated: June 9, 2008

CONTOS & BUNCH
A Professional Corporation

By: _____
JOHN R. CONTOS
Attorneys for Plaintiffs
ROBERT MANDLER, ANTHONY MANDLER, MARISA MANDLER and ELIZABETH MANDLER