CryoTransport Case Report:
Edward W. Kuhrt, Patient A-1110

by Linda Chamberlain, CryoTransport Manager
Alcor Life Extension Foundation

Author's Notes

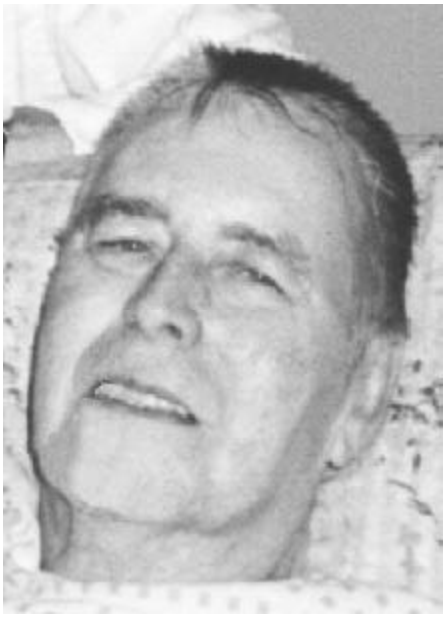
The format used in this report follows closely that used by Mike Darwin of BioPreservation, Inc. This, and the brevity used to describe events during the washout and cryoperfusion, was done in order to make it easier for those who will be using these technical reports in efforts to improve CryoTransport (both transport and preservation) protocols. This technical report was sent out for review and comment prior to publication. Special thanks is given to Hugh Hixon of Alcor and to Mike Darwin of BioPreservation, Inc. for comments and suggestions which improved both the form and substance of this report.

CryoTransport can be broken down into three major areas: (1) Remote rescue and transport to Alcor, which includes patient acquisition and stabilization, (2) Cryoprotective Perfusion, and (3) Cooldown and Long-Term Care. This report covers all areas except long-term care, which is just beginning.

Background History and Synopsis

Mr. Kuhrt was one of the earliest members of a cryonics organization. In a LifePact interview made in the hospital several weeks before his cardiopulmonary arrest, Mr. Kuhrt told me many interesting stories about his early involvement with cryonics. In the mid 1970's, newspapers ran an article about the Cryonics Society of New York and about how cryonics was being funded by life insurance. Insurance companies were concerned CSNY might be fraudulently selling insurance policies. As a private investigator, Mr. Kuhrt was retained to look into this. To their surprise, he was himself a member. He assured the insurance industry that cryonics was legitimate. Mr. Kuhrt and his wife became Alcor members in June, 1986.

In January of 1997, Mr. Kuhrt was diagnosed with an aggressive form of lung cancer that had already metastasized to the bones. Upon learning that his cancer was terminal, Mr. Kuhrt expressed a desire to move to Scottsdale, Arizona to be close to Alcor when he experienced cardiopulmonary arrest.



Edward W. Kuhrt
Date of Birth: December 28, 1931
Date of Biostasis: February 8, 1997

Due to insurance (HMO) rules, and the loss of strength resulting from his radiation treatments, Mr. Kuhrt was not able to relocate to Arizona. The relatively slow progress of his disease, however, allowed two members of Alcor's CryoTransport Team (Linda Chamberlain and Tanya Jones) to visit Long Island several weeks in advance of his cardiopulmonary arrest in order to make arrangements with his oncologist, the hospital, and a cooperating funeral home.

The oncologist and Mather Memorial Hospital in Port Jefferson, Long Island, were very supportive and gave Alcor unprecedented assistance. The positive, cooperative attitude displayed by the entire nursing staff comforted Mr. Kuhrt's family and proved invaluable to Alcor during its remote standby and transport.

At the time of arrest, a code team was called from the emergency room and cardiopulmonary resuscitation began, along with administration of heparin, sodium bicarbonate, streptokinase, and Maalox (through gastric tube). After the emergency room personnel finished this initial protocol, Alcor personnel continued cardiac compression, packed Mr. Kuhrt

in ice, and delivered additional medications to limit ischemic damage and stabilize cell membranes.

The patient was then transferred to the funeral home for whole-body washout before being shipped by air to Scottsdale for cryoperfusion and long-term storage. Mr. Kuhrt's long acquaintance with both cryonics and Alcor, as well as his aggressive involvement in his own care — particularly as it related to his impending cryonic suspension — established his informed consent. Both the washout and perfusion went well. Full details follow.

Medical History

(Although repeated attempts have been made to acquire full medical records, to date such records have not been received. The medical history below is, at the time of this publication, still limited and was primarily gained through personal conversations with family members.)

Mr. Kuhrt smoked two packs of cigarettes per day since 1945 (52 years). In 1987 he was diagnosed with colon cancer and received a colostomy. In 1995 he was diagnosed with Type II, adult onset diabetes. Mr. Kuhrt's diabetes was managed with 2000 mg. of Glucophage q.d., and 40 mg. Glucotrol q.d. The patient also took 20 mg. of Zestril q.d. for hypertension.

On January 6, 1997, at the age of 65, Mr. Kuhrt was admitted to Mather Memorial Hospital, Port Jefferson, NY, for right-hip pain that had been problematic for several months. Examination was performed with markers [*sic*] to include the right hip, the proximal shaft right femur and the right iliac bone, the ischial bone and the pubic bone. Mr. Kuhrt was diagnosed as having metastatic non-small cell lung cancer. Palliative radiation was prescribed to slow tumor growth and manage pain. Blood transfusions were also given.

During a logistics trip to Long Island (to make arrangements with a funeral director, the oncologist, and the hospital), we observed that Mr. Kuhrt had a normal level of consciousness, his spirits were high at being visited by Alcor members, and he was eager and happy to talk about his cryonics arrangements and his hopes for re-entry and rehabilitation. Nonetheless, he was obviously in pain and tired easily. The patient's circulation in both legs was badly compromised by tumors in his hips and buttocks. Premortem signs included almost total lack of color, lack of pedal pulse at either the dorsalis pedis or posterior tibial, and a greatly distended and rigid abdomen due to ascites (cause as yet not known). While we were there, Mr. Kuhrt's morphine level was doubled in an effort to maintain his comfort.

The day after returning from the reconnaissance trip (January 18, 1997) the author called the patient's wife, Anne Kuhrt. Mr. Kuhrt's level of consciousness (LOC) had declined remarkably; he remembered that we had been there, but did not appear to understand the purpose of our trip. Mr. Kuhrt did not even remember that his cancer was terminal. The increased morphine was making a marked difference in his LOC.

Cardiopulmonary Arrest

The oncologist had planned to implant an abdominal morphine pump on February 5, 1997, but before this could be done the patient experienced abdominal bleeding that brought on an emergency requiring intubation and artificial respiration. Mr. Kuhrt was no longer a candidate for the abdominal pump and had come very close to cardiopulmonary arrest.

Anne Kuhrt called Alcor with this information. After discussing the situation with the nurse on duty, it was decided that the transport team should be deployed as soon as possible the next day.

Date	Time	Pulse	Resp/Min	Temp (°F)	Skin condition	morphine drips/minute
2-5-97						10
2-6-97	100-110	12-14		97	cool, dry, grey	12
2-7-97	19:39	104	14	101.7	warm, dry	14
	21:12	BP = 98/58				16
	22:50	116	12	97.3	warm, dry	16
2-8-97	12:17					20
	03:57	weak, labored			cool, damp	26

Figure 1: Agonal Course Vital Signs

The transport team arrived on the evening of February 6, 1997. Mr. Kuhrt was experiencing uncontrollable pain and was not able to communicate well, but he did seem to recognize and respond positively to the two team members who had met previously (Linda Chamberlain and Tanya Jones). CryoTransport medications were drawn and put on ice, and washout equipment was set up at the mortuary. (For Vital Signs, see Figure 1.)

Time:	Medication Administered:
no record	60,000 IU heparin (for anticoagulation)
04:34	120,000 IU streptokinase (lysis of hemostatic fibrin)
no record	250 cc Maalox (to neutralize gastric hydrochloric acid)
04:37	700 mEq sodium bicarbonate (to combat acidosis)
04:40	patient's head packed with ice bags

Figure 2: Medications Administered by Hospital Personnel.

Mr. Kuhrt and his family (wife, son, and daughter, their respective spouses, and Anne's two sisters) requested that medical life support efforts be terminated. On doctor's orders, at 11:19 AM on February 7, 1997, the nursing staff discontinued the IV insulin drip. The patient was kept on oxygen, and his morphine was increased. The patient's urine output (to Foley catheter) was nearly nonexistent; extant urine was dark brown.

The nursing staff agreed to leave the patient's subclavian catheter and nasogastric tube in place for the administration of Alcor cryotransport medications. After saying farewell to Mr. Kuhrt and his family, the Alcor team retired to a nearby lounge at 10:00 PM. Thereafter, Linda Chamberlain checked the patient approximately once per hour. The patient's son and daughter-in-law remained at his bedside.

Remote Transport:
CPR, Medication, and Initial, External Cooling

Participants:
Steve Bridge, Logistics
Fred Chamberlain, Logistics
Linda Chamberlain, Transport team
Hugh Hixon, Transport team
Tanya Jones, Transport Manager

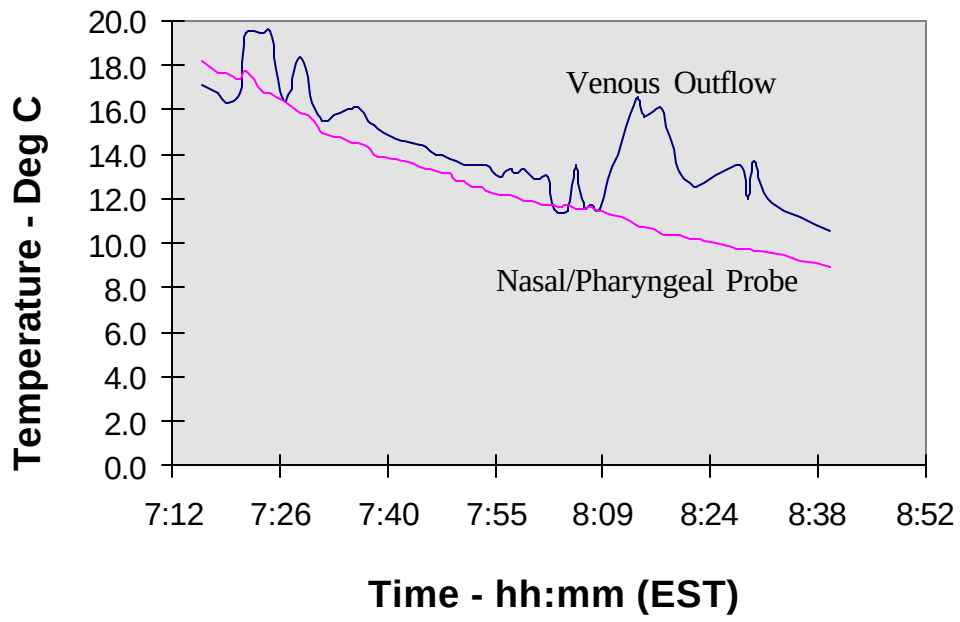
At approximately 4:15 AM on February 8, 1997, the patient was attended by his son, daughter, and daughter-in-law. As the patient's level of consciousness (LOC) had been declining over the early morning hours, they were watching him closely. When his rate of respirations dropped to less than 1 per 15 seconds, they summoned the Alcor team and the attending nurse. When the author arrived in the patient's room, the nurse was ascultating the patient's chest for lung sounds. The author stepped out into the hall to talk with other team members and heard the code called.

An Emergency Room code team responded, took an EKG, and pronounced the patient at 04:25 EST. From prior arrangement with Alcor, the code team then began manual cardiopulmonary resuscitation with 10 liters of oxygen per minute by bag valve mask from 04:25 until 04:49. Simultaneously, cryotransport medications (Figure 2) were administered by IV push.

Hospital regulations did not allow the presence of non-hospital personnel in the patient's room during initial resuscitation

Time:	Medication Administered: (Dosages determined for a 160 lb. patient.)
04:49	2.6 cc metubine iodide (to inhibit shivering)
04:51	37.5 cc potassium chloride (reduce cerebral metabolic demand)
04.54	15 cc epinephrine (to improve perfusion and blood pressure)
04:55	4 cc deferoxamine (to reduce free-radical damage)
04:55	2 cc gentamycin (to inhibit microbial overgrowth)
04:55	75 cc sodium citrate (to reduce cerebral reperfusion injury)
04:56	8 cc methylprednisolone (to stabilize cell membranes)
04:57	9 cc chlorpromazine (to stabilize cell membranes)
04:58	30,000 IU additional heparin (to inhibit clotting)
04:57	250,000 IU additional streptokinase (lysis of hemostatic fibrin)
05:05	manual cardiac compression discontinued

Figure 3: Medications Administered by Alcor Transport Team



Graph 1: Temperature Graph for Total Body Washout. Data log below (Figure 4).

Time AM	Temp °C Ven. Outflow	Temp °C Nasal	Note Number	Time AM	Temp °C Ven. Outflow	Temp °C Nasal	Note Number
			1				
7:16	17.1	18.2	2	7:56	13.0	12.2	
7:18	16.8	17.6		7:57	13.3	12.2	
7:19	16.3	17.6	3	7:58	13.1	12.1	
7:21	16.8	17.3		7:59	13.3	11.9	
7:22	19.4	17.7		8:00	13.0	11.9	
7:24	19.4	16.9	4	8:01	12.9	11.8	
7:25	19.5	16.8		8:02	13.0	11.7	7
7:27	16.4	16.4		8:03	11.5	11.7	
7:29	18.4	15.8		8:04	11.3	11.6	
7:31	16.1	15.5	5	8:05	11.4	11.7	
7:32	15.5	15.0		8:06	13.5	11.5	8
7:35	15.9	14.7		8:07	11.6	11.5	
7:36	16.0	14.5	6	8:08	11.7	11.6	
7:37	16.1	14.5		8:09	11.4	11.4	
7:38	15.5	14.3		8:10	12.5	11.3	9
7:39	15.3	14.0		8:14	16.5	10.9	
7:42	14.7	13.8		8:15	15.6	10.8	
7:43	14.6	13.7		8:17	16.1	10.5	
7:44	14.5	13.6		8:18	15.2	10.4	
7:45	14.4	13.4		8:19	14.2	10.4	
7:46	14.3	13.3		8:20	13.1	10.4	10
7:47	14.0	13.2		8:21	12.7	10.2	
7:48	14.0	13.1		8:22	12.5	10.2	
7:49	13.8	13.1		8:28	13.5	9.7	11
7:50	13.7	12.8		8:29	12.0	9.7	
7:51	13.5	12.8		8:30	13.7	9.6	
7:52	13.5	12.5		8:32	11.8	9.5	
7:53	13.5	12.5		8:40	10.6	9.0	12
7:54	13.5	12.4					

Figure 4: Temperature Log for Total Body Washout.

procedures. (We were not aware of this regulation until the moment hospital staff members asked us to leave the room.) This meant that the Alcor team was not able to direct the ER team in their efforts. As a result, no one documented the code team's protocol, and a signficant (though unrecorded) warm-ischemic period occurred before anyone placed ice on the patient's head.

Hugh Hixon took over manual compression at 4:49 when the ER team turned the patient over to Alcor. The Alcor team had anticipated using the hospital bag valve and oropharyngeal tube to continue giving oxygen to the patient; unfortunately, ER personnel departed with their code cart and other equipment. While continuing manual sternal compression, Linda Chamberlain and Tanya Jones administered further cryotransport medications (Figure 3).

The transport team had planned to place a thermocouple probe through the nasogastric tube. The team member assigned to get and place the probe was not able to find it and the temperature monitoring could not be done.

Remote Whole-Body Washout

The patient was transported to a local mortuary for whole-body washout. While the Alcor team finalized set-up of the roller pump and and elimination of air bubbles from the tubing, the mortician cannulated brachial vessels on the patient's medial right arm. This was a departure from the normal protocol, which was to cannulate femoral vessels for the washout.

The departure from normal protocol was necessitated by the patient's condition. Circulation in both legs was badly compromised by tumors and ascites. (Premortem signs: almost total lack of color, lack of pedal pulse at either the dorsalis pedis or posterior tibial, greatly distended and rigid abdomen.)

Cannulation was completed at 06:40 and the washout perfusion was begun. No clots were seen and the embalmer was vocally and visually impressed by the flow as well as by the amount of hemodilution achieved.

The perfusate used (20 liters, pH of 7.8 and 335 mOs) was a proprietary high potassium formulation developed by Alcor. An ice bath was used for heat exchange, rendering the perfusate at approximately 5°C at injection. (Graph 1 and Figure 4 show the temperature descent achieved during whole-body washout with external cooling and internal cooling.) Because of the small bore of venous cannula (20 Fr), venous return was less than one liter/min, resulting in a very slow systemic cooldown. In fact, most of the initial cerebral cooldown was accomplished by external ice packs; the procedure's principal benefit was the washout of blood.

Whole-Body Washout Notes:

(The following notes correspond to the data shown in Figure 4.)

1. 06:55 EST Cannulation of brachial vessels and start of washout within approximately 1 hour and 40 minutes of pronouncement (due to delay on part of funeral director in picking up patient). Immediate clearing of capillaries in the face was very noticeable. Line pressures taken from immediately above the arterial cannula ranged from 160 mmHg to 260 mmHg. Temperature probe placed in nasal gastric tube.

2. At 07:09 the venous effluent had cleared remarkably and the perfusion circuit was stopped to change to recirculation.

3. 07:19 Recirculation established with flow at about 1 liter/min. The hematocrit appeared to increase as the perfusate color darkened markedly. It was assumed that blood was leaking from the abdomen.

4. 07:24 Pump stopped to allow reservoir to refill. Upon palpation, the feet and femoral area felt very cool.

5. 07:31 Reservoir had refilled sufficiently and recirculation was reestablished with 0.6 liter/min flow and arterial pressure at 70 mmHg. We were not able to increase pressure due to the low venous return (which may have been due to the brachial cannulation).

6. 07:36 Temperature of the venous output rose due to the mortician massaging the patient's abdomen in an attempt to relieve distention.

7. 08:03 Flow rate rose to 0.8 liters/min.

8. 08:06 Mortician raised the patient's feet to increase the return of cold perfusate from the feet to the trunk.

9. 08:10 Pump stopped due to low venous return (to allow the reservoir to fill). Recirculation started again at 08:17.

10. 08:20 Line pressure was at 80 mmHg.

11. 08:28 Line pressure was at 100 mmHg.

12. 08:40 Perfusion was terminated, cannula removed, and vessels ligated. The nasal temperature was 9.0°C.

The patient was cleaned up on the mortuary preparation table and transferred to a heavy-duty (8 mil) vinyl body bag. A trocar was used to remove serous fluid from the peritoneal cavity. At this time it was noted that there was no rigor present.

The body bag containing the patient was then placed atop a bed of zip-lock bags containing crushed (water) ice, which had been laid down inside an insulated air transport box (Zeigler case). The patient was covered with additional bags of crushed ice, and the transport container was wrapped in R-20 insulation and closed for air transport to Scottsdale, Arizona. Air transport was uneventful.

Cryoprotective Perfusion at Alcor Life Extension Foundation

Participants:

Steve Bridge, Logistics
Fred Chamberlain, Logistics
Linda Chamberlain, Burr Hole
Tony Cerrulo, Funeral Director
Matt Day, OR Assistant
Keith Henson, Assistant Surgeon
Hugh Hixon, OR Assistant
Tanya Jones, Transport Manager
Judy Krantz, R.N., Surgical Nurse
Nancy McEachern, D.V.M., Surgeon
Judy Muhlestein, Blood Samples, Scribe
Mike Perry, Administrative
Derek Ryan, Blood Samples
Brian Shock, Refractometry
Mathew Sullivan, Cephalic Isolation, Scribe
Ralph Whelan, Perfusionist

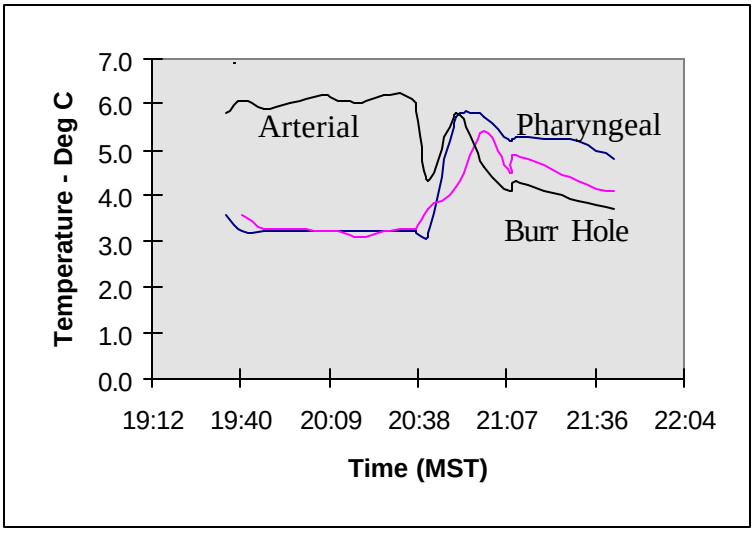
The patient was picked up by the Alcor ambulance at Phoenix Sky Harbor Airport on February 8, 1997 and transported to the Alcor facility in Scottsdale. Below are significant points in the cryopreservation of the patient.

18:20 Patient moved into the Operating Room, laid on a bed of ice bags, re-packed with ice bags, and then prepared for a median sternotomy and cranial burr-hole by scrubbing with providone iodine solution and draping.

18:47 The first of two burr-holes begun.

18:53 Incision for the median sternotomy begun.

18:59 Sternum spread for access to great vessels of the heart.



Graph 2: Cryoperfusion Temperature Descent.
Series 1-- esophageal temperature.
Series 2--burr hole temperature.
Series 3--arterial temperature.

Cryoperfusion Data Collection Sheet

Time	Temp *C Esoph	Temp *C Burr	Temp *C Arterial	Pressure mmHg Arterial	Flow Rate L/min Arterial
19:36	3.6		5.8		
19:41	3.2	3.6	6.1		
19:48	3.2	3.3	5.9		
19:57	3.2	3.3	6.0		
20:07	3.2	3.2	6.2		
20:12	3.2	3.2	6.1		
20:20	3.2	3.1	6.0		
20:27	3.2	3.2	6.2		
20:37	3.2	3.3	6.0		
20:41	3.1	3.7	4.3		
20:51	5.7	4.2	5.8	110	
21:00	5.7	5.4	4.6	125	
21:08	5.2	4.5	4.1	122	1.60
21:10	5.3	4.9	4.3	121	
21:30	5.2	4.3	3.9	133	1.10
21:42	4.8	4.1	3.7	143	0.91

Figure 5: Cryoperfusion Temperature Descent, Pressure, and Flow Rates.

18:59 Second burr hole begun.

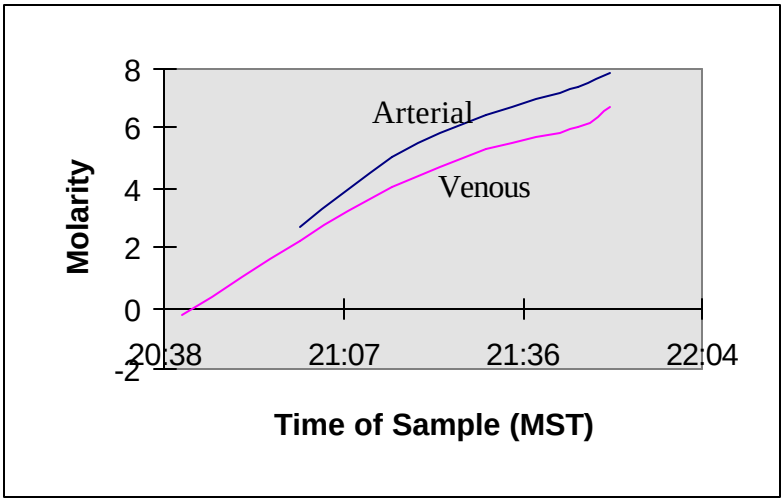
19:23 Thermocouple probes and crackphone probes inserted into left burr-hole.

19:28 Pressure monitor placed in the ascending aorta.

19:30 Brain observed to appear clear and translucent.

19:32 Pulmonary artery exposed.

19:34 Pharyngeal thermocouple probe identified as non-functional and replaced.
19:36 Second burr hole finished. Burr holes placed coronally, approximately two inches lateral of the center line.
19:36 Began nasal, esophageal, and burr-hole temperature monitoring (see Figures 5, Graph 2).
19:41 Placed pursestring in ascending aorta.
19:42 Ligated pulmonary artery and vein.
19:56 Ascending aorta cannulated.
20:16 Trouble encountered while attempting to clamp descending aorta.
20:18 Descending aorta ligated.
20:22 Placed pursestring in right atrium.
20:26 Right atrium cannulated.
20:32 Connection of arterial/venous loop to cannula.
20:36 Bypass flow started.
20:37 Pump started.
20:41 Venous sample #1 (see chemistries below). Samples taken every 15 minutes (Figure 6).
20:43 Glycerolization ramp started with 4% glycerol (Figure 7 and Graph 5).
20:54 Cerebral cortical volume rapidly decreased to 2-3 mm below the margin of the burr-hole.
20:54 Burr-hole sample #1. Samples taken every 10 minutes (Figure 8 and Graph 6).
21:00 Injection flow 1.6 liters/min, 125 mmHg (Figure 5).
21.55 Perfusion terminated. Glycerolization at 6.74 molar (Figure 7 and Graph 5).



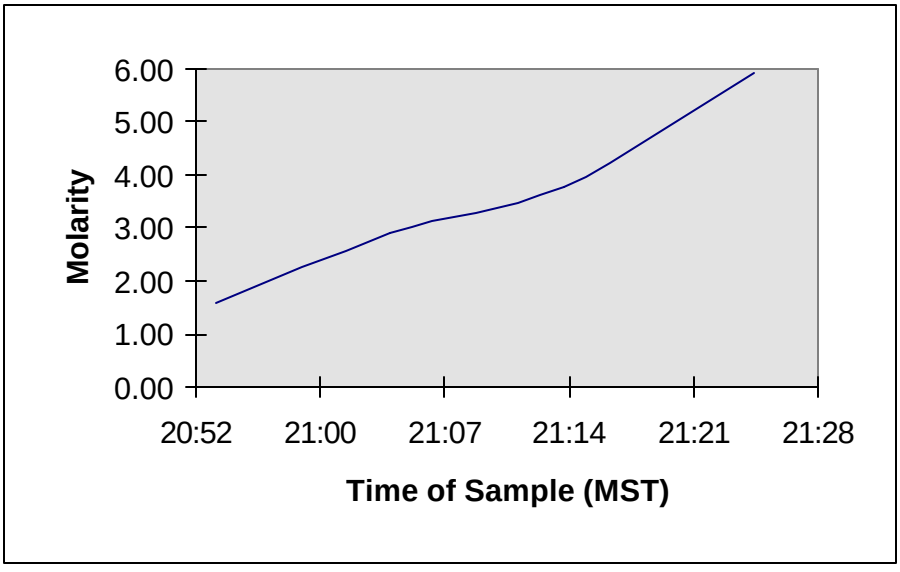
Graph 5: CPA Concentration Ramp
Series 1=Arterial, Series 2=Venous

CPA Concentration Log

Time	Sample	Arterial	Venous	Arterial	Venous	Arterial	Venous
19:10	4% Glycerol	12.00					
19:10	75% Glycerol	64.10					
20:41	Sample #1		8.00		-1.45		-0.20
21:00	Sample #2	23.60	20.80	19.81	15.99	2.71	2.19
21:15	Sample #3	36.40	30.90	37.25	29.76	5.10	4.07
21:30	Sample #4	43.60	37.30	47.06	38.48	6.44	5.27
21:45	Sample #5	48.60	41.20	53.97	43.87	7.39	6.01
21:50	Sample #6	51.00	45.20	57.15	49.24	7.83	6.74

Figure 7: CPA Concentration Ramp

Graph 6: Burr Hole Glycerol Concentration



Time	Sample	Refrac	V/V%	Molarity
19:10	4% Glycerol	12.00		
19:10	75% Glycerol	64.10		
20:54	Sample #1	17.50	11.50	1.57
21:04	Sample #2	24.50	21.03	2.88
21:14	Sample #3	29.20	27.44	3.76
21:25	Sample #4	40.80	43.25	5.92

Figure 8:
Burr Hole Glycerol Concentration Log

Perfusate Sample Data

Test	Normal Range	Units	Sample#1	Sample#2	Sample#3	Sample#4	Sample#5	Sample#6
Time of Sample		hours:minutes	20:41	21:00	21:15	21:30	21:45	21:55
GLUCOSE	65 to 115	MG/DL	68	74	76	81	86	93
BUN	5 to 25	MG/DL	10	11	13	12	12	10
CREAT	0.5 to 1.5	MG/DL	0.2	0.2	0.3	0.3	0.2	0.2
BUN/CRE	10.0 to 20.0	MG/DL	50	55	43.3	40	60	50
URIC ACID	2.2 to 8.0	MG/DL	0.8	0.9	1.3	1.1	1	0.8
SODIUM	133 to 145	MEQ/L	51	56	61	59	58	58
POTASSIUM	3.5 to 5.2	MEQ/L	24.2	27.6	29.9	30.5	31.7	32.7
CHLORIDE	95 to 112	MEQ/L	47	53	56	55	54	54
CO2	22 to 30	MEQ/L	11	11	12	13	12	11
GAP	4 to 18	MEQ/L	-7	-8	-7	-9	-8	-7
OSMO-CALC	275 to 295	MOSM/K	145	159	173	170	170	171
TPROT	5.9 to 8.4	G/DL	0.2	0.5	0.6	0.9	0.9	0.9
ALBUMIN	3.6 to 5.2	G/DL	0.1	0.2	0.2	0.2	0.2	0.1
GLOBULIN	1.9 to 3.4	G/DL	0.1	0.3	0.4	0.7	0.7	0.8
ALB/GLOB	1.1 to 2.2	MG/DL	1	0.7	0.5	0.3	0.3	0.1
CHOL	0 to 200	MG/DL	0	0	0	0	0	0
TRIG	30 to 175	MG/DL	452	114	35	247	175	227
CALCIUM	8.5 to 10.5	MG/DL	1.6	1.7	1.9	1.7	1.6	1.6
ION CA-CAL	3.5 to 5.2	MG/DL	1.5	1.5	1.7	1.4	1.3	1.3
PHOS	2.5 to 4.5	MG/DL	2.6	2.4	4	3.6	3.8	3.5
GGT	0 to 65	IU/L	0	0	1	0	0	0
ALK PHOS	30 to 130	IU/L	19	4	6	3	8	11
SGPT (ALT)	0 to 40	IU/L	53	45	117	102	118	58
SGOT (AST)	0 to 41	IU/L	204	188	471	415	477	239
LDH	95 to 250	IU/L	554	496	1158	1050	1222	726
CPK	25 to 225	IU/L	295	200	250	233	273	212
TBILI	0.2 to 1.2	MG/DL	0	0.1	0	0.1	0.1	0.1
DBILI	0.0 to 0.3	MG/DL	0	0	0	0	0	0
IBILI	0.0 to 1.2	MG/DL	0	0.1	0	0.1	0.1	0.1
IRON	40 to 150	MCG/DL	1	2	3	3	4	2
HIV 1 & HIV 2		NEG	NEG					
HEPATITIS A		NEG	NEG					
HEPATITIS B		NEG	NEG					
HEPATITIS C		NEG	NEG					

Figure 6: Perfusate Sample Data Log

Cryoprotective perfusion was started with 4% glycerol and ramped by mixing with 75% glycerol perfusate (see Figure 7). Cryoperfusion proceeded uneventfully. The cerebral cortical surface was repeatedly examined during cryoprotective perfusion. The brain was noted to be moderately dehydrated at the conclusion of cryoprotective perfusion with an estimated shrinkage of 2-3 mm from the surface of the bore hole. Terminal glycerol concentrations were 7.83 Molar arterial and 6.74 Molar venous at 21:55. Terminal burr hole glycerol concentration was 5.92 (see Figures 8 and Graph 6). Perfusion was discontinued at 21:55 MST.

Venous perfusate samples were drawn at 15 minute intervals during cryoprotective perfusion. Due to a lab error, CPK isoenzymes were not run. (For perfusate sample data, see Figure 6.)

Cephalic Isolation

Closure of burr holes was completed before cephalic isolation. Burr holes were filled with bone wax (with the thermocouple and crackphone probes in place) and the skin incisions over burr-holes were sutured. All probes were secured with surgical staples to the skin of the patient's head.

Surgery for cephalic isolation was begun immediately after closure of the burr holes. The skin,

Significant Blood Tests

Note: Levels will depend on specifics of washout and perfusion protocols, and of timing of samples.

TEST	SIGNIFICANCE	SIGNIFICANCE
BUN	NORMAL FUNCTION	CRYOPROTECTIVE PERFUSION
(Blood Urea Nitrogen)	Normally evaluates kidney function	Pre-mortem — may be elevated due to dehydration, common in terminal patients.
		Perfusion — extracted from tissues by perfusate
Triglycerides	Normally evaluates for heart disease	May be an indicator of cellular breakdown
SGPT/ALT — (alanine aminotransferase)	Normally evaluates for liver disease	Indicates damage to liver cells
SGOT/AST (aspartate aminotransferase)	Myocardial infarction or liver disease.	Indicates cell damage (many organs)
LDH (lactate dehydrogenase)	Normally used to indicate myocardial damage, but also indicates more general cellular damage.	Indicates generalized cellular damage
CPK (creatine phosphokinase)	Normally indicates cellular damage in skeletal muscle. Isozyme tests can localize to the specific organs.	Indicates damage in brain, heart, and brain, cardiac muscle, and skeletal muscle, specifically.
GGT (gamma-glutamyl transferase)	Normally used to indicate liver damage	Indicates damage to liver cells
Alkaline phosphatase	Used in identifying a wide range of diseases	Indicates generalized cellular damage

cervical musculature, and spinal cord all exhibited complete blood washout and typical signs of thorough, uniform glycerolization (dehydration, waxy texture, ambering of the skin and deepening of skeletal muscle color).

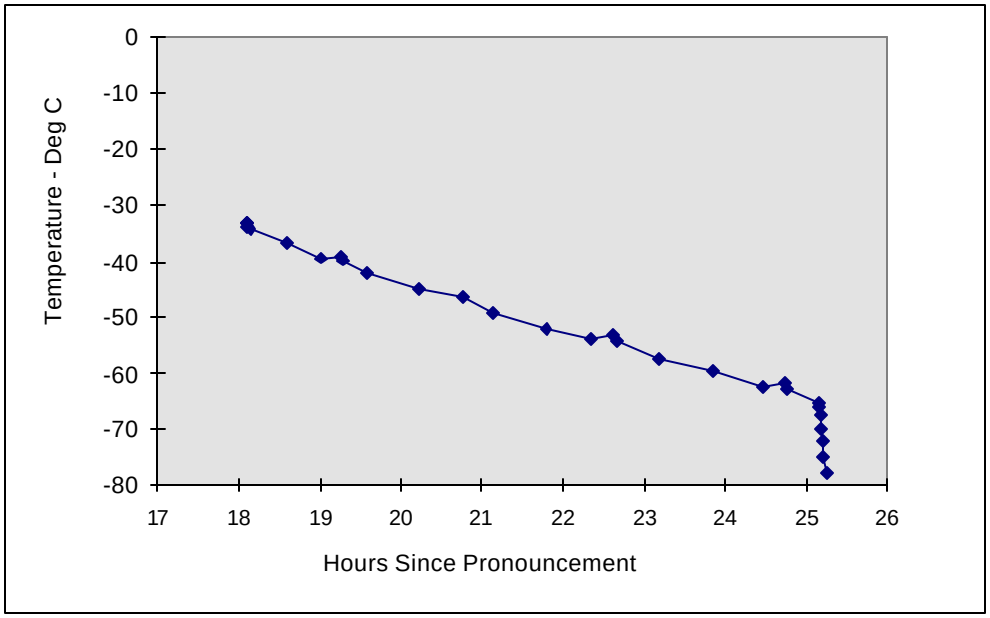
Cooldown

The patient (cephalon) was then placed in two 1 mil polyethylene bags with two thermocouple probes and two crackphone probes protruding from the bags' opening. At 20.08 hours (post-pronouncement) the patient was submerged in a 15 liter Silcool bath, which had been precooled to -31ºC. The first temperature readings after submersion in the Silcool were #1: pharyngeal, -22.1ºC (this probe was not securely placed and later failed to give accurate data, resulting in the data of Graph 3 being based on burr hole temperature); #2: Silcool bath, -30.9ºC; #3: Head surface, -38.9ºC; and #4: Burr hole, -33.2ºC.

The patient's cooling curve to dry-ice temperature is shown in Graph 3. The computer-controlled temperature descent, proprietary to Alcor, was set for -4ºC/hour, to a temperature of -55ºC (6 hours). At -55ºC, controlled rate cooling was terminated and the bath filled with dry ice. The temperature descent to -79ºC took place over a period of approximately 7.5 hours. This data is based on the burr hole probe. Readings on the pharyngeal probe were erratic. Surgical staples used to secure the pharyngeal probe had not been placed tightly, resulting in temperature readings which were not a reliable indication of the cranial interior.

The patient's cooling curve to liquid nitrogen is shown in Graph 4. The bath probe was calibrated at liquid nitrogen temperature, and the other probes were set to it while at dry ice temperature. On February 10, 1997, computer-controlled temperature descent was set for -1ºC /

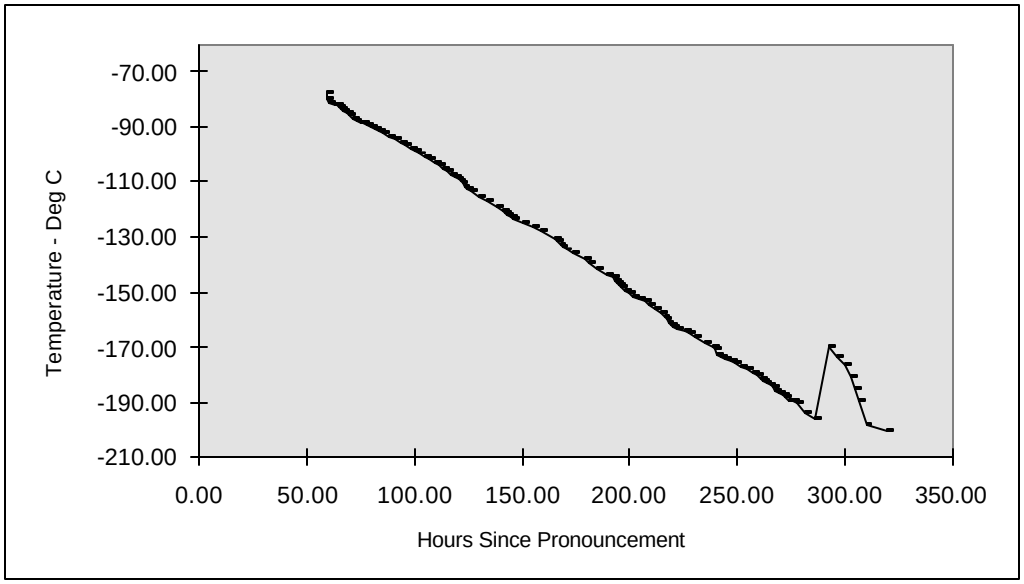
hour to a temperature of -90ºC. On February 11, 1997, computer-controlled temperature descent was set for -1/2ºC/hour to a temperature of -190ºC. Temperature descent to -196ºC took place over a period of approximately 300 hours (12.52 days). At 287.8 hours post pronouncement (-196.1ºC) a computer crash was experienced. At 291.4 hours post pronouncement (172.7ºC) the cool down was resumed.



Graph 3: Temperature Descent to -79ºC (burr hole temperature).

Crackphone Analysis

The final venous glycerol concentration was 6.74 Molar. The response (amplitude) of the Channel 1 crackphone was consistently about one-half the response of the Channel 2 crackphone. 22 of the events recorded by the crackphone have been identified as cracks. The amplitude of crack signature ranges from approximately 0.05 volt to 2.4 volts (Channel 2 amplitude).



Graph 4: Temperature Descent to -196ºC (burr hole temperature).

The largest amplitude crack was the first one recorded. A second, smaller crack occurred within one second after this. The amplitude of the second, smaller crack was about 1/4 that of the first crack. The temperature was about -107°C. One other double crack occurred, on LN_2 fill at liquid nitrogen temperature. Again, the amplitude of the second, smaller crack was about 1/4 that of the first crack and occurred within a second. (The limit of resolution of the event clock is one second. The record length for each event is four milliseconds.)

The events are dispersed fairly evenly along the time-temperature ramp. However, a plot of amplitudes vs. temperatures appears to show a trailing-off of events that might indicate a relationship between cracks; that is, cracking events do not occur at random, but have a propagating structure, even though long periods of time (up to 32 hours observed in this case) may elapse between recorded events.

Discussion

During the medial sternotomy, the descending aorta was surgically ligated. Post perfusion examination revealed (an expected) transition from perfused to un-perfused tissue which was strikingly sharp. We believe that both observations and the data show that the patient received good total-body washout and cryoperfusion of the upper body and head which resulted in an excellent degree of glycerolization and cryoprotection.