Cover:

"Atomscript: An exercise in atomic manipulation. Scanning Tunneling Microscope picture of xenon atoms on nickel substrate at 4øK superimposed on drawing of common cellular components at the same scale. Accompanying article on page 7."

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EDITORIAL MATTERS
by Steve Bridge

Editorially, there is good news and there is bad news. The good news is that we are catching up on the monthly issues. We have cleared out a lot of backlogged material and have actually put out three issues in six weeks, PLUS completely reediting the Suspension Transport Manual, preparing for several training classes, sending out thousands of promotional mailings, and occasionally sleeping. With the Reanimation Conference, two Transport Team training sessions, and the Space Development Conference scheduled for May, it may be difficult to keep this schedule up, but we'll try. At least we'll have plenty to write about!

We are actively seeking some new material, though. We can't write everything. We get bored, and you get bored seeing a limited number of writers every month. We've added Russell Whitaker as a contributor in this issue. We know that YOU have ideas, too. Let's see them.

The bad news is that we still make mistakes. We made errors on two (count 'em, two!) Michael Perry pieces. In Dr. Perry's article, "Freezing Before Death" in the March, 1990 issue of Cryonics, we got lost in the footnotes. On the bottom of page 37, the last three references were numbered [4], [3], and [3]. They should have been numbered [5], [4], and [3]. Each one of you will immediately correct your copy. No slackers, now!

In the April, 1990 issue we treated Dr. Perry even worse -- we failed to acknowledge his existence. We left his name off of his letter to the editor concerning capital punishment (on page 21.) Fortunately for us, he...
is against capital punishment, so we only had to supply this correction.

We also left the listing for the Asilomar Conference on August 24-26 out of the Events Calendar. ("Hugh did it." "No, Mike did it!") It should be there this month. We apologize to the Chamberlains for the goof. (If you all attend, you'll make us feel a lot better about this.)

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ONE TO GET READY

by Mike Darwin and Steve Bridge

On the weekend of March 17th and 18th we (Steve Bridge and Mike Darwin) journeyed to San Jose to carry out a training session for the Alcor Northern California (ANC) rescue and transport team. This session was more than an academic exercise; a member in Northern California is terminally ill and the purpose of the training session was to polish skills and to prepare the local group to deal with the upcoming suspension.

The session had another purpose as well; to train us to use a lot of the new equipment and procedures developed by Alcor Southern California (ASC) over the past year or so. The Portable Ice Bath has finally metamorphosed into a stable form; we have a new Michigan Instruments high impulse CPR (HI-CPR) Heart Lung Resuscitator (HLR), as well as new simultaneous compression-ventilation CPR (SCV-CPR) units; and a variety of new ischemia "blocking" medications, some of which require special handling. One of us (Mike Darwin) had extensively re-written the training manual and now it was time for the other of us (Steve Bridge) to see how well the "theoretical" translated to the practical.

Attending the session were ANC stalwarts Fred and Linda Chamberlain, Arel Lucas, Keith Henson, Naomi Reynolds, Thomas Donaldson, and Leonard Zubkoff. Saturday's session was not as productive technically as we had wished, since so much time was spent talking over changes in the training manual and answering the dozens of questions people had. Everyone (including us) seemed more focused when we returned to the training on Sunday. That session was especially hard work, since it included five hours of uninterrupted practice on the new HLR machines. Talk about sore backs and aching knees!

The students trained both on the HI-CPR and on the SCV-CPR HLR units. Outwardly the units look very much alike; but their methods of adjustment and operation were enough different to cause confusion. We spent a lot of
time just practicing basic techniques of switching from manual CPR to machine CPR with each machine. One glitch that took some time to fix was discovering that Mike and Frank Rothaker has miscommunicated on getting matching hose fittings on the oxygen tank. It took a bit of thinking and engineering to get that problem corrected so we could do any work at all.

Another problem was that the SCV-HLR machine appeared not to be working correctly. Simultaneous-Compression-Ventilation means that instead of the HLR performing five chest compressions and then one ventilation in imitation of manual CPR, it performs one ventilation with each compression, in order to increase the total pressure inside the chest cavity. Mike had requested from Michigan Instruments that our machine be modified to pause ventilating after every fifth breath to provide a chance for an exhalation. This was for safety's sake, since our patients could possibly be on HLR support as long as 4-5 hours. Unfortunately, no change was observed after the fifth ventilation, so the unit will have to be further adjusted.

We also spent quite a bit of time discussing medications and learning how to draw them up and prepare for IV administration. Naomi Reynolds recently completed a phlebotomy course (learning how to do venipuncture for drawing blood and starting IV lines) to go with her Emergency Medical Technician certificate, so we hope ANC can now handle the entire transport procedure themselves if necessary. (No one hopes they will have to do it alone any time soon; but let's face it: we are growing and it will happen sometime.)

There was also another unexpected benefit to the weekend. Keith Henson brought along the latest in miniaturization: a small, 20 pound, nearly-shoe-box sized air compressor which was used to power the HLRs during practice sessions. Bottled oxygen is somewhat expensive for practice sessions, but the HLR's require compressed air or oxygen to operate at all. After we had initially run out of oxygen, Steve and Mike made sound effects to pretend that the machines were still going, so we could continue the training. However, "thump-a, thump-a, thump-a, thump-a, thump, whoosh" does not really provide an adequate substitute.

Fortunately, the compressor performed well, and we have purchased a similar unit for use here at ASC. The one drawback with the compressor which we will have to overcome is that it generates water in-line, which could damage the machine. We are currently engineering a water trap and chemical drier which can be used to dry out the airstream and prevent damage to the HLR from water entering the gas path and corroding the internal mechanisms.

A special thanks to all the "students" who attended this class and who so patiently tolerated the foibles of the instructors and taught them so much! New ideas were flowing freely and it seems likely that other useful pieces of new transport equipment will result from the session. Both Mike and Steve were pleased with the enthusiasm, attitude, and abilities of the ANC members. Certainly we are on the right track toward forming the kinds of Transport Teams that will be increasingly necessary in the coming months and years.

Also an extra thank you to Naomi Reynolds and Roger Gregory who opened up their home for a reception/gab fest for us on Friday night.
THE FRENCH CONNECTION?

by Mike Darwin

It began innocently enough. Our order for hydroxyethyl starch (HES) simply didn't get filled. Weeks went by, then months. Inquiries to the supplier (DuPont Critical Care) were initially responded to with vague reassurances. Meanwhile, the supply of a chemical critical to performing cryonic suspensions and doing cryobiological research began to dwindle dangerously. We continued to use our stock for research, anticipating re-stocking within a few days to a few weeks at most.

Finally, our inquiries to the supplier got more confrontational. And their answers got more "honest." The denouement of the problem came one smoggy day while I was sitting in a car with Saul Kent talking with a DuPont representative on Saul's car phone. "Why won't you fill our orders? Why has this material become impossible to get? And, more to the point, what do we have to do get it?"

After the usual evasive run-around, I pressed for some hard answers. Finally, I was told that the company had learned that their product was being misused by drug dealers to cut cocaine and that they had put tight controls on supplies. Ahhh yessss, using $75-a-kilo pharmaceutical grade cornstarch to cut cocaine. Makes sense to you too, doesn't it?!!

After pressing further on how we could get access to more HES, I was told that it was in very short supply now and that it was going only to investigators with credentials they thought good enough, who submitted a detailed plan of the experiments they intended to conduct with the agent. Then DuPont would decide whether to provide the material: and then only in the quantity that was needed. Jerry Leaf had a similar conversation wherein he was told that they intended to limit supply only to approved investigators for the next two years or so. . . . A more believable explanation is that they sense that their product may have a lot more uses and have come up with a ploy for peeking into researcher's lab books.

The amazing part of all this is that HES is not some new experimental drug or some exotic, dangerous chemical. It is a modified form of cornstarch (and about as toxic) and is in widespread medical use (in the U.S. and around the world) as a plasma substitute or blood "volume expander" used to treat shock. We had been ordering the material from DuPont for years without problems. However, while HES is widely used clinically, it was now generally available only as a 5% solution for IV administration, at a cost of about $65.00 per 500 cc (the material we had been using came as a powder). To get enough to do a whole-body suspension would cost nearly $4000 and would further require a costly and time-
consuming dialysis procedure to wash out all the sodium chloride which was also dissolved in solution with it!

Nor were we alone in our hour of need. Everyone else we talked to was having the same problem: no HES.

When our supply dwindled to 5.5 kilos (enough for 100 liters of perfusate -- barely enough to do a whole-body patient), we had to bite the bullet and buy Dextran-40 and Dextran-75 -- two other far less satisfactory colloids. Dextran isn't cheap either; the tab for that was about $750.

Meanwhile the search for a source of HES went on. An appeal was put out in Cryonics (April, 1989) asking for help in locating a new source. Chemist Hugh Hixon looked into making it ourselves and this turned out to be not a possibility. Hydrolyzing starch to get the right molecular weight distribution is an art, the hydroxyethylation didn't look much easier, and the quantity of HES we required put it beyond benchtop chemistry. We tracked down leads that it might be available in England. It was, but only as a sodium chloride-containing liquid solution at a prohibitive cost (the same situation as in the U.S.).

We heard rumors that DuPont's supplier was a German company. Then we found out that the Japanese held a patent. . . . On and on it went. Up one blind alley and down another. Through it all, the man who kept looking was Jerry Leaf. From March of 1989 till March of 1990 the pursuit continued.

Finally, near the end of March of this year a container of 30 kilos of HES of the required molecular weight and purity arrived. We cannot tell you how we got it. Or who
got it for us. Or what byzantine route was taken to secure the supply. You may however, rest assured it was done legally (and yes, even morally!). We wish we could publicly thank all the people who helped, but we can't.

We now have HES again, and we will not have to do any suspensions anytime soon without it. Yes, it did cost us several times what we were paying for it. But the point is, we got it.

Special thanks to Jerry and to all those who participated in the search for the HES. You know who you are.

The irony in all of this is that if we had wanted to buy 30 kilos of cocaine we would have found it far easier and it wouldn't have taken a year! And at the rate cocaine prices are falling we probably could have gotten it cheaper.

It's a strange, strange world.

But there's lesson in all this, a valuable lesson. William Bennett, listen up: interdiction works, at least for cornstarch. Chances are, we'd have ordered 60 kilos by now if it weren't for the cost and the hassle.

A small price to pay to keep the coke supply of America clean, pure, and uncontaminated by pharmaceutical grade starch.
SCANDINAVIAN MAN PLACED INTO SUSPENSION

The international shipping unit developed by Alan Sinclair of Alcor UK recently got its first workout. Bredo Morstoel, an 89-year-old native of Baerum, Norway was transported from Norway to the facilities of Trans Time, Inc. in Oakland, California. Morstoel's grandson, Trygve Bauge, first contacted Alcor in an attempt to get his father into cryonic suspension in the United States. Unfortunately, the circumstances were such that we could do little for him and Morstoel had already been legally dead for three days at room temperature before being straight-frozen at -20øC.

Bauge then attempted to make arrangements with the Cryonics Institute, in Detroit, Michigan. After initially agreeing to accept Morstoel, the CI board decided against doing so. Ultimately Bauge selected Trans Time and his grandfather's body was shipped there in early February.

Bauge had also contacted Alan Sinclair of Alcor UK, and Alan generously allowed the UK shipping container to be used to transport Morstoel's remains (Alan is the owner of the UK equipment).

Alan reports that the unit performed very well, and despite the fact that they were limited to shipping only a modest amount of dry ice, Morstoel arrived adequately refrigerated with a significant amount of the dry ice remaining.

Good news for the growing contingent of European members who are relying on the unit.

BRIDGE OVER TROUBLED WATERS

by Mike Darwin

Our Midwest Coordinator is missing. From the Midwest, anyway! But rest assured that Steve Bridge is alive and well in California at Alcor's Riverside facility. Steve is here on a three-month sabbatical from his "normal" job as a children's librarian in Indianapolis, Indiana.

Steve's goals here are multifaceted; on a personal level he hopes to "get a mental break from doing the same job for 15 years, something that everybody should do."
For Alcor, Steve hopes to "take on and complete, or at least make some real headway on some of the tasks that the regular staff doesn't have time to do, such as a number of editing projects which have piled up and on which my library and teaching background will be of real value. For example, my experience as an EMT and my work on the transport phase of two suspensions gives me the background to edit the Transport Protocol manual. Besides the editing, my other main area of responsibility will be working to organize Alcor's archives. This includes a huge amount of material provided by Curtis Henderson documenting the history of the world's first working cryonics organization, the Cryonics Society of New York."

"In addition, I'll be helping with Alcor's presence at the Reanimation Conference and the Space Development Conference. I'll also be taking up some of the load by giving talks and interviews. The rest of the time (what little there is of it) I'll be sitting around missing my girlfriend Angalee Shepherd."

In commenting on his experience at Alcor to date, Steve says "it is both exciting and difficult to work with such intense and committed people, who are also such individualists." (Do I detect a hint of diplomacy here? -- MD.) "Another very good reason for being here for me is that I will be able to participate in all the training sessions for suspensions, which I haven't been able to do before."

Steve also notes that "there are a lot of tasks that people like me with specialized skills can do at Alcor. Your skills don't have to be medical or engineering or scientific. Many tasks require only patience and perseverance. I hope that other Alcor members will follow my example because Alcor is not only a fascinating place to work, it is also the thing that is going to save our lives."

Steve arrived in early March and will remain at Alcor thru June 9th, providing his health and sanity survive that long. Certainly he has been a big help since his arrival.

And besides, one of his other duties has been proofing Cryonics and that means that we have someone else to blame the errors on.

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ATOMSCRIPT
This little bit of news isn't really about any fundamental technological breakthrough, and besides, most of our readers will know about it already. But it is neat and it may have desirable effects unrelated to its technical significance.

As most of you no doubt know, the letters "IBM" have been written in xenon atoms with a scanning tunnelling microscope (STM). This major feat of literary miniaturization was achieved by cooling a nickel crystal and some xenon atoms to 4øK and then grabbing and dragging around the xenon atoms on the surface, arranging them to spell out "IBM." How do you "grab" an atom with an STM tip? Well to quote from the paper in Nature (344, 524 (5 Apr 1990) by D. M. Eigler and E. K. Schweizer describing their nanowriting exploits, this is done by "increasing the tip-atom interaction by lowering the tip toward the atom; this is achieved by changing the required tunnel current to a higher value, typically in the range of 1-6x10^-8 amps, which causes the tip to move towards the atom until the new tunnel current is reached." Is that clear?

Just what does writing on this scale really mean? Well, as Alcor's Dr. Mike Perry pointed out, if you took a nickel from your pocket and increased its size until it had the same diameter as the Earth, the letters "IBM" written in xenon atoms would be about 10 feet high. How's that for miniturization? An example of more relevance to the cryonicist is the one which we show on this month's cover; the STM images produced by Eigler and Schweitzer shown on the same scale as the biological structures which comprise our cells: DNA, cell membrane, ribosome, hemoglobin. . . .

The technical tour de force demonstrated by Eigler and Schweitzer isn't the kind of breakthrough achievement that cryonicists and nanotechnologists constantly chatter about -- at least not technologically. But it has had other effects. For many technically and scientifically sophisticated people this is graphic, understandable proof that you can really do nanotechnology! Several friends and acquaintances have commented to me "My god, you were right; you really can move atoms around! Just think about it!" Nanotechnology maven Ralph Merkle of Xerox PARC reports the same kind of phenomenon.

In the long run, the widespread press coverage associated with the publication of "Positioning Single Atoms With A Scanning Tunnelling Microscope" should not be underestimated. Just seeing "IBM" spelled out in letters 50 high, made up of easily resolvable individual xenon atoms makes it all seem so real.

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Nevada Cryonics Association: Inception Notes

by Russell E. Whitaker

My home, Las Vegas, Nevada, has many attractive features. It is a city of almost a million people and it is among the fastest growing in the nation. And it is only four hours' driving time from Alcor's Southern California facility.
The easy drive, and my own drive, impelled me to visit Alcor's facilities a little over a year ago. I was immediately taken by this irresistible appeal to my sense of adventure, and asked the "how do I sign up?" question. In short order, I received The Big Stack of Paperwork. Many months later, I bit the bullet and actually completed the requirements for membership.

I've never been satisfied "just being a member" of anything, and much prefer to "take big bites" of life (thank you, Robert Heinlein). After attending a couple of Alcor board meetings, and making a business-related decision to stay in Nevada, I decided that what this locale needs is a local emergency response capability.

Aah, but I'm getting ahead of myself, aren't I? My first thought was for a simple discussion group, supper club style. After all, doesn't Alcor have same-day world-wide response?* They don't really need the help (hindrance?) of neophytes.

Not so. To quote Jean-Louis Gassee of Apple Computer: "A novice is a beginning expert." I borrowed a stack of Cryonics back issues, did a little research, and quickly discovered that people involved as Alcor Coordinators were not strictly medical people, as one might expect. These people operate on the "volunteer fire department" principle of preparedness.

I telephoned everyone listed in my local database and invited each to the kickoff meeting of the "Nevada Cryonics Association." We held this exploratory gathering at a local Denny's restaurant, in a conference room in the back. No charge to us, just the expectation of the manager that we were paying customers.

* In theory we have 24 hour world-wide response. In practice this is likely to be far from the case. It could take 24 days just to get our "emergency" equipment through customs in some countries. If you live outside the U.S. you'd better start working on other scenarios -- this is why we now have a facility in England. It is also important to point out that even within the U.S. 24 hours is a long time to wait for basic help if your heart isn't beating! -- Eds.

Alcor President Carlos Mondragon attended the meeting. Though we had at most 14 people present, Carlos seemed quite happy with the turnout. Whereas I have usually attended gatherings of well-established groups, such as Toastmasters, Carlos has had much more experience with fledgling enterprises. Two of us locals were bracelet-wearing Suspension Members. To my surprise, several of the rest had already sold themselves on the idea. About half those previously familiar with cryonics expressed the desire to sign up with Alcor. Of the remainder, several asked for more information. . . . something I had more or less guaranteed by encouraging them to sign the sheet I had provided.

Carlos brought Bluebooks -- Darwin and Wowk's "Alcor: Threshold to
Tomorrow" -- and a stack of the new tri-fold brochures. We passed around some of these, and the ensuing studious silence bought us a little time to eat dinner with our mouths closed. Most of the rest of the four-hour meeting was "Q&A" format, with Carlos and I providing "A's" to the many "Q's." Questions ranged from "what is cryonics?" to "how can Alcor suspend me here when they are in another state?" Those of us who are already signed up discussed the need to find out about the local situation, such as hospice policies (nurses can pronounce death in these facilities), potential membership markets, and media opportunities.

The largest surprise of this meeting was the level of informed interest of most in attendance. Apparently, the interest has been here all along, but no one had taken the initiative to get things going. If just some of those present sign up soon, we're well on our way to starting an Alcor chapter. Many of the prerequisites are present: For starters, I took Arthur McCombs' March 25 training class for Alcor agents. I'm not "blooded" yet -- haven't signed anyone on -- but I've been told by local prospects that they are far less daunted by the sign-up requirements now that they have a local contact.

Additionally, I have a daytime job as a medical laboratory technician for a small clinic. I have my Emergency Medical Technician certificate with additional training and some experience. In May, I take the Alcor Transport Technician training course in Riverside. I plan to teach what I know to those new Suspension Members in Vegas who wish to take one of the EMT courses taught locally. Interestingly enough, some of the best EMT training in the country is taught at a local private ambulance company for under $300... books, materials, and test fees included.

One thing I emphasized at our meeting was that I invite the active participation of anyone interested. My experience with different organizations has shown me that no successful mutual aid society endures when one person takes all the initiative.

People from several radio stations on both bands have offered to provide public announcements of cryonics-related events. That, plus a couple of offers for local magazine and newspaper interviews, promises interesting times ahead.

On Saturday, July 14, Saul Kent and Mike Darwin will be featured speakers at an "introduction to cryonics" seminar to be held at a location to be chosen soon. This will give us time to set up interviews and send out press releases. A lead time of several months will also allow time to set up a mailing address and pick up a voicemail account for the local chapter. The latter is necessary to provide the curious with a means to contact us, and to provide updated recorded announcements of upcoming events.

We're also putting out a newsletter, initially a broadsheet which will answer the "what is cryonics?" questions and announce upcoming events. Later issues will still include outreach, but will shift some of its emphasis to local material such as synopsized minutes of previous meetings and other matters.

All new leads generated by personal contact and unsolicited inquiry will be sent to

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Alcor. I hope to speed turnaround on this in particular by sending all my correspondence to Riverside by way of CompuServe electronic mail, presently through the account of Steve Bridge.

Our next meeting is 7:30 PM, May 7 at:

Vegas Bayou restaurant
1290 E. Flamingo Road
Las Vegas, Nevada

We have the use of the Bayou conference room indefinitely for meetings on the first Monday of every month.

I hope to receive the opinions and suggestions of those who have traveled this road before. Anyone visiting Las Vegas is welcome to give me a call and set up a time we can talk in person.

Russell E. Whitaker
1350 E. Flamingo Rd. #247
Las Vegas, NV 89119
(702)366-7958
CompuServe: 71750,2413
Internet: 71750.2413@compuserve.com
GENie: R. WHITAKER4

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IS HALSTON FROZEN?

by Mike Darwin

Roy Halston Frowick, a.k.a. "Halston" was a man who made his fame and fortune by exploiting his pre-occupation with the beautiful. If you don't think you know who Halston was, you really do. Remember Jackie Kennedy's controversial (and ultimately wildly popular) pillbox hats: that's Halston. During the 1970's he dressed every woman who was anyone in the public eye: Liza Minnelli, Martha Graham, Elizabeth Taylor, and many others. He was also the first fashion designer to sell his "empire" for a phenomenal sum and grow rich. Sadly, he has become a victim of AIDS.

To a man as concerned with the aesthetic, the beautiful, AIDS must have been an almost intolerable blow. His unhappy recent years and his banishment from the fashion industry left him embittered and reclusive. On March 26, Halston died of Kaposi's sarcoma and related problems in San Francisco.

A few days later CNN and some of the Los Angeles radio stations began carrying stories that Halston, or part of him, had been frozen to await future revival or cloning. These rumors seemed to imply that

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As reproduced here, Avi Ben Abraham of the American Cryonics Society, denying that Roy Halston Frowick was placed in cryonic suspension by that organization.

We can only speculate that an effort of some kind by either Halston himself or someone close to him may have been under way to facilitate his suspension. It apparently didn't happen.

An AIDS death isn't really much of a news item. It's the kind of thing that increasingly happens all the time with people across the U.S. and even around the world -- people whose names you probably wouldn't recognize. Frankly, we wouldn't even have noted Halston's passing in these pages if it hadn't been for the buzz of rumors about cryonics surrounding his death.

Halston lived a wild and fantastically productive life. His death is a pity. But it was not the first and it will not be the last. It does however point out an important lesson: all the fame and wealth and admiration and productivity in the world don't mean a thing if terminal illness catches you unprepared.

What was the real story of Halston's last days? We may never know. But we do know the phone will ring here and elsewhere bringing similar stories of last-minute desperation. Most such calls will unfold to the same, dreary, hopeless ending. The real tragedy is that it needn't have ended that way.

How many more Halstons are needed before everyone who wants cryonics gets the message and gets signed up?

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MEMBERSHIP STATUS

Alcor has 169 Suspension Members, 383 Associate Members, and 13 members in suspension.

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Mormons Live Longer?

An eight year study by UCLA researchers indicates that Mormons who abstain from alcohol, smoking, and caffeine and who exercise regularly live longer -- a lot longer!

The study was conducted by Dr. James Enstrom of the UCLA School of Public Health and involved 10,000 religiously active Mormons and their wives. Males in the study could anticipate a mean lifespan of 85 years, compared to a mean life expectancy of 74 years for the white male population in the U. S. An additional benefit is reduced incidence of cardiovascular and cancer mortality: middle-aged Mormon men had only 14% of the rate of cardiovascular mortality and 34% of the rate of cancer mortality for middle-aged white men in the U.S.

The females in the study fared similarly, with average life expectancy being 86 for the Mormon females versus 80 for white females in the general population. Similarly, the women in the study experienced only 55% the rate of cancer death and 34% the rate of cardiovascular disease-related mortality of their counterparts in the general population.

Longer Lasting Transplants?

Fujisawa Pharmaceutical Co. of Japan, working in cooperation with University of Pittsburgh transplant maverick Dr. Thomas Starzl, has developed a new anti-rejection drug which is reportedly more effective than cyclosporin A. Starzl is responsible for the increasing use of liver transplantation around the world and was instrumental in introducing cyclosporin A into clinical use. The new drug, FK-506, is derived from a soil fungus first isolated in Japan. So far the drug has demonstrated superior control of rejection and has not demonstrated the troubling kidney toxicity of cyclosporin A. The drug is in preliminary trials at the University of Pittsburgh, and it is not anticipated that it will be released for widespread use for at least another 3 to 5 years.

Reconnecting Severed Spinal Cords

Exciting progress continues in the central nervous system repair and regeneration department. The latest is the success of University of Zurich researchers Dr. Lisa Schnell and Dr. Martin E. Schwab in inducing functional regeneration of severed cortico-spinal nerves of two six-week-old rats. New fibers grew by as much as 11 millimeters in three weeks; about 10 times the rate of regrowth that occurs without the new treatment. Nerve regeneration was achieved by the use of a genetically engineered monoclonal antibody called IN-1. The researchers discovered that IN-1 inactivates a naturally occurring inhibitory factor which normally blocks nerve regeneration.

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The discovery raises the possibility of an effective cure for those suffering from recent spinal cord injuries. It also raises as a possibility the transplantation of the intact eye and of the brain: two organs considered untransplantable as a consequence of the inability of the spinal cord and other CNS tissues to regenerate. Successful transplantation of the brain might well lead to pressure for developing brain cryopreservation techniques in a weird reversal of the usual situation wherein an organ is needed until it can be matched with a recipient's body. In the cryopreserved brain scenario, it would be a person waiting to be "transplanted" into a new body.

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A EUROPEAN TUTORIAL

by Mike Darwin

As we announced last month, Alcor USA and Alcor UK will be sponsoring a cryonics conference and grand opening of the new UK facility from October 26-28, 1990.

I want to go on record here in saying that I think this will be one of the most exciting conferences ever held. And I want to urge each and every one of you to attend.

I am sure that an awful lot of you are thinking what I used think: Europe! That's so expensive! Well, it's true that it isn't as cheap as a day trip to the local amusement park. But it needn't be that expensive either. I'm guess I'm kind of a zealot about this conference and in trying to encourage you to go. Not just because of the cryonics angle, but precisely because it is Europe. My trip there in February of 1989 was a high point of my life. It was also a very educational and emotional experience. I stood on Charles Darwin's grave and touched the slab covering Isaac Newton in Westminster Abbey. I toured the Vatican museum and saw the Sistine Chapel ceiling in Michelangelo's original technicolor! When I return in October, I intend to see the Hunterian Medical Collection at the British Museum; visit Down House, Darwin's home; see the site of the Telford Bridge where the industrial revolution began; and visit Berlin to be witness to the collapse of communism and gaze upon the face of the woman from my past: Nefertiti.

Some of you will have visited Europe in the past, but all too many of you have never been there. Technophile, bibliophile, biologist, or lawyer, Europe will offer you places where you can be in the presence of the roots and beginnings of the disciplines you love. It may be almost mystical, but there is an emotion of the moment which comes when you smell the sickly sweet smell of Westminster Abbey and you realize that with each breath you inhale atoms that once comprised the heart and soul of Newton, Darwin, Ben Johnson, Dickens, Kipling, Samuel Johnson, Chaucer, and countless others.

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In short, Europe is a feast for the intellectual, the man or woman in love with the history of ideas as well as the world. It offered me experiences which come as close as I can come to a sense of the religious: to be in the presence of the history which gave birth to both me and the civilization I am in large measure the product of.

But enough of the sales campaign. The intention of this article was to "de-intimidate" those of you who might be hesitating to go.

Cost is always a big deterrent. It needn't be. Thanks to Luigi Warren the cost of my ticket to Europe was only $500 round-trip. This was possible by purchasing my ticket from a discount ticket firm called Airkit. The only restrictions were that I could not purchase my ticket more than 30 days in advance and that I could not order any special meals (a modest handicap in my case since I am a vegetarian and the flight lasts 11 hours). Other people who have checked ticket prices recently have still been able to find regular flights for October in the range of $600.00 round trip.

Once in London, travel via the "Underground" was both easy and economical. I had no trouble finding my way. This from a spatial cripple who can't find his way across town! Weekly passes to the Underground are available and these cost only a few pounds (currently the English pound equals $1.60 US).

Unlike in the U.S., trains and subways go EVERYWHERE, and they are clean, economical and efficient. There is also an incredibly inexpensive way to get around Europe going 1st class and doing it dirt cheap. It is called a Eurail Pass: It is available only to non-Europeans and it gives unlimited, first class travel anywhere the trains go, which is just about anywhere in Europe. There are several levels of pricing; one of the most popular is "Flex-Pass" -- if you travel by train only 5 days out of a 15-day period, the charge is merely $198.00; 9 days out of 21 -- $360.00, etc. A "Youth" pass is available for people under the age of 26 for one month of unlimited second class rail travel for $380.00. You can get a Eurail pass from your local travel agent.

In the off season, such as October, fairly nice hotels, can be secured for about $35 to $45 a night in almost all European cities. And keep in mind if you schedule your travel right, you can cut your hotel bills by traveling at night and getting a sleeping car (a chechette) for a modest extra sum. This also allows you to use your travel time for sleeping time.

And the trains are G-R-E-A-T. They are so neat, especially the sleepers, that it's hard to shake the feeling that you aren't in some wonderful old black-and-white movie. You half expect Bogart to turn the corner at any moment. It's a wonderful, romantic feeling.

Food prices are comparable to here and you can shop at grocery stores. Unless you want to pay an arm and a leg, it is wise to shop for things to eat. A cheese sandwich may cost as much as $4.00 at a food stall! Trains are NOT economical sources of nourishment. Coca Cola should also be avoided in Italy: it's $2.00 a can, but other food was inexpensive and delicious.
All in all, you can go to Europe inexpensively. And if you don't believe me, just buy some of "Europe On $40 Dollars A Day" guides available in any bookstore.

I think this conference is going to be a good one. I think that London and the rest of Europe can also be a wonderful, life enhancing experience.

See you in London!

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LETTERS TO THE EDITORS

To the Editors:

In response to the Richard Liebee problem, of course any member who has made the necessary arrangements to be suspended and through no fault of his own has ended up in a dire situation, we must do something for. I feel the best way at the moment is to establish a Richard Liebee Suspension Fund, funded by voluntary contribution. The reason I say this is because the arrangements we are establishing in the U.K. would not help you if we adopted "A" [Establish a general suspension emergency fund to be paid for by increased dues/suspension charges. See Cryonics, March, 1990, page 1 for details. In view of the heavy expenditures to establish a suspension facility in the U.K., most of the dues of the U.K. members are being returned to the U.K. group. -- Eds.], and for my part I will be willing to contribute $500.00 Unfortunately I am unable to offer more at this moment in time.

I feel on the long term, if it looked as though various members were slipping through the net, it would be nice if we could establish an emergency fund to cover any eventualities; as you quite rightly point out, it is very difficult without hindsight to establish problems that may occur. However, if we just allowed anyone in our organization to rot when they have done everything in their power at a given time to be suspended, this could give cryonics a bad name, and after all, it could be any one of us tomorrow.

Alan Sinclair
Wannock, United Kingdom

* * *

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Alcor,

Suggestion:

I have noticed a time or so some vulgar expressions were printed -- used for emphasizing the point to be made --

I myself at age 70 years have a very
bad habit of cussing like a sailor when talking to myself - stress is a habit. Vulgarity turns some "off." They tend to judge the whole group or all to be less than desirable. I hope you don't turn away some people who might become members.

I have absolutely marveled at the work done on the production of this magazine you print. I find the hard work you all do inspiring. Never say die! It has meant a lot to me -- kept me informed on problems and progress made and more informed on scientific expectations and or possibilities.

Faith, hope, & charity -- the love of life is greatest.

Bye for now,
Alice Leaf

* * * * * *

The Alcor Life Extension Foundation operates the finest cryonic suspension facility in the world in Riverside, California. There are currently 13 cryonics patients being cared for at this facility and 175 Full Members who have made financial and legal arrangements to be placed into suspension should the need arise, and many others who are in the process of doing so.

The Alcor Facility has received worldwide media attention. It has been featured in major publications such as "People," "Longevity," "The Los Angeles Times," and "The Boston Globe," and on national TV shows such as "Good Morning America," "Larry King," "Oprah Winfrey," and "Lifestyles of the Rich and Famous." A recent episode of "L.A. Law" was based entirely upon Alcor's precedent-shattering legal action.

Now you can tour the Alcor Facility yourself under the expert guidance of the Alcor staff. The Alcor Facility is open to small groups (of no more than 15 people) who wish to learn how terminal patients are placed into suspension within Alcor's high-tech cryonics laboratory and how they are cared for at -320 degrees F. in tall, stainless-steel cylinders.

The Alcor Tour also features a discussion of the scientific evidence that patients in a cryonic suspension have a realistic chance of being restored to life, health, and youthful vigor as well as a fascinating exploration of the fantastic advances coming in the 21st century and beyond. The Alcor Tour provides an invaluable opportunity for you to have all your questions answered about cryonics, the prospect of extended human lifespan, and the shape of the future.

This article presents some thoughts based on the growing, but still incomplete, understanding of human thinking now being developed by neuroscientists. It's all tentative. What I aim to do is more to focus on experimental issues involved in this question. The answers seem to me to move over slowly into the statement of the title; but after all, everything has turned around more than once and we won't really know until the game is over.

Yet the notion of uploading incorporates a complete metaphor about how we think, remember, and exist. The idea is that we are (very complex) computer programs, running in more or less identical machines. This is not an unreasonable idea, and it's had a lot of use. And in fact it would imply that we can take this program and run it on more powerful machines.

Yet even scrutinizing the computer program metaphor, any honest hacker would raise problems to an easy porting of any arbitrary program. We can't, after all, simply take the very same Macintosh program in 68020 code, load it onto a DOS 3.3 80386 machine and expect it to run. It doesn't even follow that programs written for 80386's will run on every machine using that chip. That means we can't move it without changing it. If we try to move it to more exotic computers, say from an 80386 over to an Ncube, the needed change becomes far more violent. Any honest hacker would wonder if it remained the "same" program in any useful sense at all.

In some of these exotic cases, porting isn't really even a serious problem. Some of them use (but very differently) the same kinds of chips we have in our own computers. They may even run special versions of Unix. Fine, so we can move the program. But then we smash into a second issue: so near and yet so far. Sure, we can run the program on this machine. It doesn't run any better than it did before, though, because it's quite incapable of using ANY of the extra power. (Apple people, by which I mean not Mac but Apple, see this everyday. An Apple IIGS will run every Apple II+ program ever written. If you were a II+ program wanting to see the world in high-resolution color, this would be cold comfort. I'm sure the Mac world sees the same problem).

Even this consequence of the analogy should tell us something important. Our minds are adapted to run in one particular computer, with a particular speed and peripherals. It's not enough just to make it run in another computer; it might even fail to work if we simply increase the speed. (Game programs give a simple common example). If programs (or minds) are ported, they often have to go through extensive changes. The more resources available to the target computer, the more changes needed.

Many people in cryonics, and (if you allow longer time spans) even myself, think one kind of technology or another will someday let us achieve things people only dream of now. Yet we do and will learn that some things are impossible: just like a technological optimist of 1790, firmly convinced that someday everyone will buy and use bottled phlogiston. Uploading may very well end up like phlogiston.

So far we have accepted the program metaphor. And someone could always
say: well, what about upgraded versions of programs? Isn't it reasonable to say that they are developments from the original parent, at least as much identical as you now and you when you were ten years old?

Yet in some very important senses we may not be programs at all. One fascinating

fact about brains is that they change, all the time. Neuroscientists have examined individual neurons in living (animal) brains, and seen their dendrites and axon move about within the brain. Some major genes activated with adult learning are those activated during growth and development. One major question about memory, still unanswered (basically because we just haven't worked up to it yet) is that of really long term memory. Forget LTP (long-term potentiation). LTP, involving chemical changes to synapses, with structural changes following on closely as a consequence, very likely does encode memory; the question comes from the obvious fact that we have no reason to think that these changes will last for more than a few months at most. How is it, then, that I can still remember playing in the snow at age 10? Or again, in terms of skills, I haven't ridden a bicycle for three years but have no doubt that I could ride one immediately if I wanted.

The implication (I don't want to say this is fact because it hasn't reached that status and may never) is that our learning itself is a kind of development, continuous with What we went through as children. That is, something grows and changes. That would mean that at some level what we learn affects our brain anatomy. It is because of this effect that the memory stays with us so long. This would mean, of course, that we would all differ from one another quite significantly in our wiring, looked at closely enough. I could not think your thoughts because I am only hooked up to think my own thoughts.

If learning and processing change our actual anatomy, and our anatomy strongly affects how we respond to learning and processing, the fundamental idea of a program vanishes like an ancient genie. The fundamental idea is that the program is separable from the machine on which it runs. We have a computer, and then on this computer there is a program, which could certainly run just as well on another computer of the same kind. Suppose though that the program itself, from the moment it began, started rewiring (and changing chips on!) the computer on which it was running, in response to its input data so that it would work better and better on the incoming data. It would very soon happen that each such system becomes quite incompatible, even if they had begun as twins in infancy.

Some computer languages, such as LISP, don't enforce a strong distinction between the program and the data, so computers are writing self-modifying programs right now. It's not even surprising that such programs can start to show a glimmer of intelligence, even if only a faint flicker. Yet programs that physically rewire the computers in which they are running take this self-modification off into another dimension. The suggestion (still only a suggestion) about brains is that this is the way they work. And brains with biological circuitry certainly show a kind of machine which could very well work this way. That is, the hardware to build such a computer certainly exists, even if it turns out after all that these capacities aren't used in our brains.

Uploading ourselves into another more powerful computer assumes, just like the idea of a program itself, that we are separable from our brains.
If we use this rewiring in any essential way, conceivably even if we only use it in core areas, any simple ideas about uploading find themselves in severe trouble. You are your brain, you're not just a program running on your brain.

I don't want any mistakes here. It remains clearly possible, and someday it must even become easy, to store the complete structural information about a brain in a computer. The issue in uploading is not that, but of somehow making a functioning real version in that computer. Storage encoded in some kind of media, in multiple copies, will someday become an ultimate form for cryonic suspension.

I would like to spend the rest of this article raising some ideas about how we can respond to this. For after all, when somebody wants to "upload" they have aims in mind that uploading seems to them a way to achieve. I do not intend this article at all to argue against these aims, which I share myself. My arguments so far only mean to raise problems with some methods proposed. Certainly it is right and proper to want to grow, dealing mentally and physically with more and more of the world, and with deeper and deeper understanding.

Please understand: we come from a long evolution, which has pressed us to optimize ourselves for our current way of life (I don't mean paleolithic, I mean now. Evolution didn't stop when we just became human; bone shapes and strengths have changed between paleolithic men and ourselves). The same evolution acting on Homo Erectus acts on us now. Evolution (and economics) will both apply to immortal superbeings. And this evolution works regardless of the origin of the changes on which it acts. But it is NOT static. We don't live now even as people did 200 years ago. (We don't die as soon, among other differences!) One way to see immortality itself is that we are trying to use technology to hasten our adaptation to the new way of life we've already adopted.

How could we do this? One way might come from using ideas from nanotechnology to allow us to expand the number of processors in our brains. The idea, of course, would be to miniaturize the processing and wiring still more, possibly to allow multiplication of neurons too. The process would move by an extension of existing forms of growth and development.

The advantage of miniaturization is that we can remain mobile in more or less our present form. Clearly, though, the amount of brain power we can keep inside our skulls is limited. But we don't have to keep our brains inside our skulls! Nothing keeps us from having peripherals. Just as our eyes and ears are IO ports, we might develop other kinds of IO ports: special senses to link to the pieces of our different brains. Perhaps we'll migrate into these peripheral brains, with bodies like our
own turning into the peripherals. Perhaps not. Someday we will know how far that may go and what kind of creatures we've become. And I propose an alternative to uploading: metamorphosis.

WISEGUY?
"Let Them Eat Cake"

Television Review And Commentary
by Mike Darwin

"There will come a time in the not too distant future, when it will be impossible for anyone living in this culture for more than a week to pick up a newspaper, turn on the television, or look at a magazine without confronting cryonics."

--Mike Darwin, circa 1978

Well, it's happening. My prediction is coming true. But what I didn't predict is how painful it would be. Once, in response to a criticism that 90% of science fiction was "crap," writer Theodore Sturgeon replied, "Yes, but 90% of everything is crap." Well, he was right.

It is now virtually impossible to turn on the television or pick up a popular magazine or newspaper without seeing or reading about cryonics. The sad fact is that most of this material is shoddy, inaccurate, or even slanderous.

Beginning on February 14, 1990, the television "suspense drama" Wiseguy began a series of five episodes which dealt in part with cryonics. The climactic episode reviewed here, which aired on March 28, was entitled "Let Them Eat Cake" and was written by Clifton Campbell and Robert Engels. (Other episodes were aired on March 7, 14, and 21.) First some comments on the show itself, then some thoughts about how cryonics is treated here.

I had not seen Wiseguy before the March 21 and 28 episodes and these were viewed on videotape. (If anyone has the first three episodes on tape, please send them along to us for the Alcor video archives.) Several people I talked to about the general quality of the program told me that it was both "good" and "popular." All I can say is, if this is what passes for entertainment in this culture, we're in even more trouble than I thought. (Although even TV Guide called this final episode "bizarre" and further commented, "This is one story arc that's gotten completely out of hand.")

The acting was forced, melodramatic, and pathetic. Mark Volcek, the cryonicist/maniac in these episodes, was played by Steven Ryan, (whom Steve Bridge claims had a fine reputation as a stage actor several years ago at the Indianapolis Repertory Theatre). Apparently, forgetting everything you've ever known about acting is a prerequisite for breaking into television. The performances in this insipid two hours of programming make "Gilligan's Island" a tempting alternative. In fact, "The Donna Reed Show" was running on cable at the same time I was watching "Wiseguy" and, given a
choice, I would have without hesitation chosen Donna Reed!

The plot of "Wiseguy" is not simple, even if the show is simpleminded. A tyrannical man, one Mark Volcek, is running a small town (Lynchboro) as his personal fiefdom in an almost cultish fashion. For reasons not made clear in the two episodes I saw, he wishes to get a hospital built in Lynchboro and is willing to bribe the state permit-giver if that's what it takes (isn't that how it's always done?). Meanwhile, the Federals, in the nerdish personage of McPike (played by Jonathan Banks) have been investigating Volcek for his corrupt activities and are now setting up to topple him and to nab the crooked health services fellow.

Apparently one of the reasons Volcek wants the hospital is to facilitate the revival of his father and grandfather from cryonic suspension. He has his paternal relatives in slick-looking capsules tucked away in the obligatory creepy mausoleum in his back yard.

Meanwhile we get to see Volcek for what he is, an emotionally crippled, mean-spirited, twisted human being who was treated in a degrading and domineering fashion by his father and suffered from the weak/absent mother syndrome (there's more pop Freud in this thing than a two-hour Joyce Brothers special). He hangs out most of the time in the town brothel, which he appears to control, and spends the rest of his spare time acting ugly and intimidating people.

Cryonics enters the scene because of his morbid fear of death, and it exists not just to rescue him from death but to allow him to perpetuate his groveling servitude to his father and grandfather -- even after their death. The most visible manifestation of this morbid twist to his personality is Volcek's pathological fascination with the old black-and-white grade D horror movie Mr. Sardonicus (Castle Films, 1961), a film whose plot competes with this one for stupidity (but which was at least better acted). Volcek is constantly screening this film (and subjecting his guests to it!) and has adopted mannerisms and a speech cadence similar to that of the central character.

The supporting characters can't even support themselves, let alone the load of garbage represented by the Volcek character. The dialogue consists of pseudointellectual gibberish referring to things like "Walden's Pond" and "skinner boxes."

Finally, in a twist so bizarre and unbelievable it's hard to imagine, let alone actually expect us to believe, the town cop, Federal agent McPike, and one of Volcheck's prostitute girlfriends contrive to re-enact the end of Mr. Sardonicus and change the ending so that the central character doesn't end up being punished for disturbing his father's grave.

Miraculously, all of Volcek's psychological problems are cured by this simple maneuver. He turns over a new leaf. The following dialogue tells us how, and moves us onto the issue of how cryonics is treated in this pitiful excuse for entertainment:

Prostitute Girlfriend (PG): I knew that this belief in cryonics and your father's dream of a dynasty -- that was your vulnerability. I
saw that vulnerability and I exploited it because it kept me strong.

Mark Volcek (MV): Julie, you've helped me. The lessons that I've learned from you are too great for anger. I have the power now not to fear death, but to celebrate life.

PG: (laughing) You've always had that, Mark.

MV: Yes, yes, I can see that now. I don't want to waste another minute, there's something I've got to do. (He runs into the snowy outdoors in his bath robe and makes his way into the crypt where the dewars holding his father and grandfather are kept.)

MV: Honor thy father! All right I'll do it. I'll continue to do it as I've done all these years. But you know what Daddy, I'll never make a son of mine honor me like this. (He picks up a screwdriver and begins prying his own nameplate off the dewar that was to ultimately receive his frozen body. Meanwhile, PG has followed him to the crypt.). You were presumptuous enough to think that you had a right to eternal life. Why?! You were a domineering son of a bitch! And I allowed you to perpetuate your dominance from inside this nitrogenized cigar tube. (MV then walks over and turns up the temperature on the unit. The camera cuts to the control panel and we observe the LED temperature readout start to rapidly climb.)

MV: Begone, Daddy! (No, I am not making this dialogue up, it's for real!) Begone, Gramps! (he says this while thawing out his grandfather.)

MV: He wouldn't let my mother be preserved since she wasn't a Volcek. The dynasty flowed through the men, he said; the bearers of the sons were immaterial, one just as good as another. How strange it is to be reborn while standing in your father's tomb. How strange -- and how -- wonderful! (Shrieking sounds are made by the equipment as the patients thaw -- or maybe that was me shrieking. Volcek suddenly drops his head.)

PG: What is it?

MV: I -- never grieved for them. When they died I didn't grieve because I knew I'd be seeing them again. Reverend Adams congratulated me on the strength of my religious convictions, but I didn't have the heart to tell him that it was, really, CRYONICS. (This line is delivered with as much hokey histrionics as you can imagine.) Now they're truly dead, and at my hand! Patricide!

PG: You didn't kill them! Death can't be cheated. Not even by Volceks. But life can be, and they cheated you of yours! They can't do that anymore.

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MV: McPike playing Crawl! (the Igor-like character from Mr. Sardonicus)

MV: What day is this?!
PG: It's Sunday, the 17th.

MV: There's still time, there's still time! (He exits the crypt running. (This bit of dialogue is cribbed from Dickens' A Christmas Carol and is uttered by Scrooge when he banishes the ghosts and decides to mend his ways.)

Volcek then heads off to attend an "event" which he is responsible for: it seems that in order to get a job in law enforcement in Lynchboro you must first be beaten senseless by a mindless brute who is apparently kept on retainer for just this purpose. A young man who was not beaten quite senseless enough on a previous episode is now about to be beaten again.

Volcek arrives just in the nick of time and subjects himself to the beating. When he is beaten bloody he stands up and declares that Volcek blood is red, not blue, and further states:

You all grew up believing that you owed the Volceks. Well, the fact is, that we owe you, and starting today I'm going to start paying you back. Volcek Mining and Logging will henceforth be known as Lynchboro Mining and Logging. It will no longer be a private company. And stock options will be divided among all of its employees. . . . We're all family. . . .

And then he announces that he will send all the prostitutes to college!

Yes, folks, this is for real. And as a last bit of improbability, since Volcek has promised to be a nice guy, it appears that the Federal agents are going to drop their investigation of him and forgive him his past corrupt activities. This must be an alternate universe.

So, the take-home message here? Simple, really: only pathological personalities are cryonicists. They are egotists who have the nerve to want to live forever and if they have to be thawed out and, incidentally, killed in the process ("Patricide!") so much the better.

One person who sat through this miserable two hours with me disagreed when I labeled this kind of show vicious. I stand by my assessment of it. Any work which sends the message that suspension patients can be callously destroyed "because they are domineering and have the arrogance to want to live forever" is vicious, deathist propaganda in its purest form.

I defend strongly the right of people to disagree with cryonics, and even to make movies like this one. But I will not concede any ground whatsoever when it comes to analyzing what this movie really articulates. The quote that opens this piece never for a moment said how the world would talk about cryonics.

Rest assured much of it will be in this vein: hate-filled, reactionary, and defensive of the status quo. But don't let that discourage you for a moment. Just think about the situation objectively. Here we are, less than 400 people in the whole world signed up, and yet we attract major media attention, every day, every month, year in and year out. Our ideas have seeped deeply into the groundwater of this culture and they surface now not only in its "news" but in its art: its movies, its television, its tabloids, and its

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This is just the beginning of the most powerful revolution the world has ever known.

Who was it that said that vilification is the first step on the road to adoration?

If you are as incensed by this kind of garbage as I was, let CBS Television know how you feel. One of our readers, Mark Potts of Stillwater, Oklahoma, has done so and sent along the following address for you to do the same if you want:

CBS Television
Audience Services Department
51 West 52nd Street
New York, N.Y. 10019

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PROSPECTS OF A CURE FOR DEATH

by Thomas Donaldson

Of course you probably recognize that the title is a misnomer for the same reason an article about a cure for cancer is a misnomer. There are so many different varieties of cancer, and so many different varieties of death, that no one cure can exist. But since there is so much optimism about curing cancer, and so little optimism about curing death, the title begins by bringing out the parallels.

We will probably always have some form of death, and some form of cancer. Looking at cancer, of course, most people see this optimistically: "yes, but many forms will disappear." Death, however, is an "eternal verity" and the "proper" attitude is to concentrate on the deaths that remain rather than those that disappear. For cancers, the absence of any single overriding cure is something to regret as a man of the world. For deaths, it is one more proof of an eternal verity.

No short article can do justice to the problems of cures for death because there are too many kinds of death, each needing a separate cure. What I will do, when I become technical, is to discuss a few currently major types of death and prospects for their cure. But we ought not to become technical too soon, because that's a very good way not to see the forest for the trees.

Fundamentally, cryonic suspension isn't about freezing people whose conditions are clearly just a matter of time until we find a technology to deal with them. It's about freezing people whom we don't know how to cure or even if a cure will be possible. Someday almost certainly we'll have better means to preserve people, too. Freezing is only our current best means. But cryonics is about preservation, the need for which will always remain.

Right now, nanotechnology has become a popular idea. But nanotechnology only solves half the problem, that of manipulating life on very small scales. The other half, whether there is a patient there to be saved, isn't yet known. Probably it will never be known for all patients
in all conditions. We didn't need nanotechnology before to tell us that
cryonic suspension was right. We don't need it now, either. It is right
to keep people around in suspension because cures for their problems have a
way of turning up. We should not let ourselves be turned into fertilizer
just because we don't see how we can be fixed. Doing that isn't just
wrong, it is absurd. And so, nanotechnology is a proof of cryonics,

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but in a deeper way. You thought, before, that no possible way could
repair frozen people. And now, somebody has come up with a possible way.
See, you've just seen it happen: cures for their problems have a way of
turning up. (Didn't you hear me the first time?)

There's another fundamental point, put forward first by Robert
Ettinger. It is actually a technical point, so I will discuss it later.
But it isn't really a point about particular technologies or particular
knowledge. Watch for it.

Ischemia and hypoxia

The two deaths I shall discuss are closely related. They often happen
as the final outcome of many other conditions, such as cancer or heart
disease. When a person's heart stops for any reason, his brain suffers
ischemia, which means "absence of blood flow." If blood flow stops to any
particular brain region, we have local ischemia, which if it continues long
enough causes a stroke. If it only lasts a short time (perhaps a few
minutes) then people can recover. But they will have felt a transient loss
of control over one side of their body, or a transient loss of memory, or
other temporary brain problem. (If this happens to you, go to a doctor at
once!)

Hypoxia (literally: "low oxygenation") is a condition in which only
small amounts of oxygen reach the brain (or other organs). It can result
from suffocation, which in turn can result from emphysema, chronic
bronchial obstruction, severe asthma, or other conditions. If allowed to
continue, it often continues into ischemia, but it is quite damaging in
itself.

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Legal declaration of death usually occurs after ischemia has lasted for
more than four minutes. Usually, though, ischemia isn't the reason for
declaration of death. Attending doctors had already decided not to revive
the patient if his or her heart stopped. Brain ischemia just ratifies
their decision.

Cellular and biochemical events of ischemic death

For cryonics, the relevant events of ischemic death all happen before
anyone tries to restore circulation. Many neurologists have reported their
observations of what happens after attempts to restore circulation. These
reports all describe a slow (over a period of many hours) deterioration of
neurons into fragments of cells. We should see this deterioration as a
consequence of ischemic death rather than identical to it.
These observations are very important for anyone trying to cure ischemic death. Sometimes they also tell us indirectly about the events which happen during ischemia. But even only a short time after we attempt to restore circulation, our brain cells end in far worse condition than immediately after ischemia. The papers I discuss in Box 1 attempt to work out, through observations during and after ischemic death, just what happens in that time.

For ischemia and hypoxia, current technology can recover people from these deaths, if the incidents don't last more than about four minutes. Some studies have even claimed as long as 15 minutes for patients at room temperature.

Cryonics patients will usually follow a different course. But even for cryonicists, accidents or neglect can cause quite prolonged delay. Heartbeat can stop minutes or hours before a suspension team can reach the patient. Cures for ischemic and hypoxic death remain extremely important to us.

Curable in principle and curable in fact

Even if only a few scraps of skin are left, we can in principle recreate a whole human being from those scraps. That created or recreated person would remember nothing of what their forerunner had done, believed, or wanted. They would be twins of their original. Re-creation or creation of this kind may in fact become common. When both John and his friends want to hang on to continuity, they could create someone with no memories at all from any scraps of John remaining, and declare that recreated person to be John. Of course, they will say that John was "hurt so badly in his accident that he lost all his memories."

But this is philosophy. Such a re-creation isn't what we want. We want to come back with our memories, beliefs, and ambitions intact. If this isn't possible, we want to come back with as much as possible. In discussing cures for death we must therefore discuss just how much damage the different deaths do to our memories and character.

Events during brain ischemia

Twenty years ago, most neurologists thought that after four minutes of ischemia, patients followed an inexorable downward course. One very important scientific development of the last 20 years consists of a growing realization that this isn't so, that drug treatments can alter the sequence of events after ischemia. Ischemia deaths (and strokes, which are ischemia deaths of only a part of the brain) are now problems to be worked on rather than decrees of omnipotent gods. However, official neglect and many

Box 1: Injuries to circulation and support

Brains are complex systems. Ischemia injures much more than just their neurons. Without supposing any immediate damage at all to neurons, these injuries can alone explain a lot of brain damage after ischemia.
The major event after ischemia, and historically the earliest one seen, is swelling and blocking off of the capillaries. Attempts to restore circulation therefore fail and hours later the neurons themselves will die. Some papers observing these changes are:


Given that after ischemia, swelling of brain capillaries cuts off support for the neurons, we would like to know just how functional are the neurons themselves. One way to find this out consists of treating the brain to restore circulation. In 1969 K. A. Hossmann and K. Sato at the Max Planck Institute tried this with cats. They could show recovery of neurons after as long as one hour of ischemic death at room temperature. (Then and now, brains aren't thought to live past about five minutes of ischemia). Some papers describing Hossmann and Sato's work are:


**) accidents produce conditions identical to those of 20 years ago. I'll therefore begin by discussing what we know of as happening during ischemic death.

The most outstanding fact about ischemic or hypoxic death is that, so far as current experiments tell, most damage occurs after circulation is restored. Very few papers have studied what happens to brain cells if nobody tries to revive the patient. For cryonics this is very important. We're not trying to revive the patient in the near future. We're just trying to freeze them with as little additional cellular damage as possible. The problem of bringing them back is left to a technology far better able to deal with all the pathologies that happen after attempted revival.

A small amount of work does try to delimit events during ischemia. Furthermore, the damage happening with revival really happens because of groundwork for it laid down during ischemia. We can use this work to infer what has happened during ischemia. In Box 1, I have set out some kinds of events known to occur in the ischemic brain. Hypoxia is more serious because even if only a little oxygen gets through to the brain, the biochemical events involved can cause damage even while hypoxia goes on.

How reversible are ischemic changes?

The most direct way to show survival of neurons after ischemia would be to grow them in culture. Unfortunately, few neurologists have bothered to do culture studies of adult neurons taken from postmortem patients.

Even after an hour of ischemia, brain cells go through many structural changes. But cells aren't mechanical systems. Many dramatic changes in
cell structure completely reverse when we restore oxygen and nutrients. Cell cultures can tell us just how serious these changes may be.

From what little we know of memory storage, these changes don't involve outright destruction of memory. To try to evaluate this, we can look closely at the effects of ischemia on two parts of the cell probably involved in memory: the cell nucleus and the synapses.

One fundamental form of damage consists of damage to the cell's ability to make new proteins. This may happen in the nucleus, where the initial stages of making new proteins occur. But several stages equally critical happen outside. The machinery to synthesize a protein is very complex: the cell reads off plans for the protein from the genes onto RNA, which then moves to the ribosomes, which make the actual protein. We don't know which part of this machinery is damaged and which remains. A major point, though, is simply that this is machinery. It isn't just a single molecule which can be destroyed or not. This complexity suggests that evidence of memory will remain despite damage.

Few scientists have studied events within the cell nucleus during and after ischemia. Much more attention has gone into means to prevent its effects prior to the event. Alcor

Box 2: Neuronal cell culture

Some reports of neuron cell cultures taken from postmortem human patients are:


Kim and his coworkers successfully cultured human neurons from the spinal cord (the superior cervical ganglion from the neck) taken from adults between four and six hours after death. They point out "with surprise" that they could succeed even after so long a delay after death.


Retinas from five dogs, taken within one hour after death, survived in culture. The retina is an extension of central nervous tissue. I believe we may accept that neurons will definitely survive one hour of ischemia. No attempts to culture dog neurons taken after one hour are reported.


This is an example of an earlier paper, in which cell cultures from cadaver brains are established as long as six hours postmortem. The authors report the rare presence of cells which appear to be neurons.

Establishing a cell culture of adult neurons is very difficult. Neurologists only developed reproducible procedures as late as 1979. Furthermore, cultured cells can lose the characteristic shapes by which we know them in living animals. The meaning of earlier reports therefore isn't clear. I have not found any recent attempt to culture neurons taken from the cerebrum of adult human beings postmortem.
itself has devoted considerable attention to that problem, with some success. But that's a significantly different problem.

Yanigihara reports experiments which study the binding of ATP to proteins within the cell nucleus. ATP is a common form of energy storage for all cells. Both during and after ischemia, some high molecular weight proteins in the cell nucleus lose their ability to bind to ATP. Since the nucleus needs ATP to make new protein, this change may explain the failure of protein synthesis after ischemia. Failure to bind to ATP happens to some quite specific proteins with molecular weights about 60,000 times that of a single hydrogen atom. Since the nucleus contains many different kinds of protein, we have no reason to believe this protein encodes for neuron memories.

Synapses may also carry memory. Ischemia damages all cell membranes, and so also the cell membranes of the synapses. The damage consists of chemical reactions which destroy phospholipids, one characteristic component of the cell membranes. These components are unlikely to carry memory, especially because they occur not just at synapses but everywhere in cell membranes.

Box 2 covers some papers on the culture of brain cells. The points to be made from these experiments are:

-- Brain cells, and therefore brains, may be essentially viable for up to six hours at room temperature. We urgently need to repeat and expand upon the work on culturing brain cells taken from adult brains after death. Few scientists have even tried to do this, but their results are very suggestive. Up to six hours, also, brain cells retain their structure very well. Changes at the microscopic level, and even at the electron microscopic level, are relatively small.

-- For up to six hours, repair probably won't require any advanced nanotechnology. At its low end, of course, nanotechnology shades into ordinary pharmacology. Many changes in brain cells during ischemia are changes in levels of crucial chemicals such as calcium ions. Drugs which sequester these should cause a distinct improvement (and do). Other events such as destruction of membrane phospholipids may require activation of systems to rebuild membranes. Since brain cells already tear down and rebuild membranes constantly, drugs only need to protect and enhance this ability, not reproduce it artificially.

One problem with current drugs may be that they act on too many different chemistries. Designer drugs with much more specific action, capable of entering brain cells, should help this problem a good deal. One system a step beyond our current abilities might consist of an interrelated family of drugs, activated only in specific circumstances, and controlling one another's action much like enzyme systems do in normal biochemistry. Systematic modification of existing enzyme systems is one road to making such systems.

Not in all cases. . . .
How, then, would we go about repair of ischemic brain? One point Robert Ettinger made 20 years ago still stands, and is quite profound. In The Prospect Of Immortality, Bob made the comment that brain cells after ischemia (or freezing) simply are not universally destroyed. Even in adult human beings, significant areas of brain can survive lengthy periods of ischemia. What does that mean? Bob described a column of soldiers after a machine gun attack. If none of the soldiers ever gets up or shows any sign of life, then they’ve probably all been killed. But if only a few get up afterward, then many more are probably wounded but still alive.

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Box 3: Reversible ischemic changes

As an illustration of changes known to reverse, a detailed study of ischemic changes in brain neurons is:


After one hour of ischemia and under light microscopy, the neurons showed slight swelling. Under electron microscopy, mitochondria were swollen, the myelin sheath surrounding the nerves had begun to disintegrate, and the nucleus underwent changes (chromatin in the nucleus had clumped together). The Golgi bodies (characteristic bodies within neurons) swelled up "remarkably." The cell matrix also lost chemicals, so that it seemed to become lighter in the electron microscope. These changes were gradual and progressive for up to 10 hours.

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What this analogy argues is that brain cells after ischemia aren't grossly disrupted. They are probably completely normal cells except for a very small number of metabolic faults. Almost 20 years of neurological research lets us go much farther to specify exactly what has happened to ischemic cells. This tells us just what kinds of medical technology we need to repair them. Here is a list of faults and specific suggestions for repair technologies.

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Box 4: Failure of protein synthesis

Another event occurring during ischemia has many consequences for neuron's ability to recover afterward. For reasons not yet clear, the neurons lose their ability to make new proteins. Since even minimal repairs of cell damage require making new proteins, this loss virtually determines everything afterward.

The following papers report that this failure occurs and examine its reasons:


Yanagihara's paper is particularly interesting. He found that certain specific proteins in the cell nucleus changed their binding to ATP, the cells' energy transfer chemical, after ischemia. He may have located the problem to changes within the cell nucleus.

-- Failure of protein synthesis.

We don't currently know what links in the protein synthesis chain have been disrupted. Only one link, of course, breaks the entire chain. To solve this problem we must identify the wounded molecule, not individually for every cell but by research into chemical events generally happening in ischemia. A properly tailored enzyme, perhaps with a delivery system to inject it into the cell, will deal with this molecule.

-- Membrane destruction.

Normal cells maintain their own cell membranes. If injury to membranes is too great, the cell cannot maintain its own internal composition. What is needed here is a chemical system which can arrive from outside, carrying the needed energy, and bringing with it much the same enzyme systems cells currently have for membrane construction. An elaborate system isn't needed.

Reports of success with drugs like verapamil are very significant here, because they suggest that after ischemia the structural parts of membranes still remain. What may happen is that the cellular pumps which constantly work to maintain chemicals within the cell are deranged. If true, then very much simpler strategies than the above should work.

Box 5: Cell membrane changes

Loss of energy sources by the cell causes it to degrade its own membranes. The entire membrane isn't destroyed outright. Instead, selected components are. The following paper reports experiments on destruction of cell membranes during ischemia:


Fatty chemicals, the phospholipids, form the basic structure of cell membranes. Initial ischemia causes destruction of the one specific such chemical, the phosphatidylinositol, in brain cell membranes. Arachidonic and stearic acids result from this. Later, in a slowly progressing reaction lasting many hours, other fatty chemicals making up the membrane
are gradually degraded.

Unfortunately, once the cell makes arachidonic and stearic acids, other chemical reactions then make even more damaging chemicals out of them. Among these are prostaglandins, thromboxanes, and leukotrienes.

One issue very important to us is damage to the synapses. Very few papers look specifically at synapses. However, the following paper does:


Damage to synapses consists of cell membrane damage much like that of other parts of the cell membrane. No special damage to synapses seems to be involved.

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Box 6: Chemical imbalance within neurons

The most outstanding changes to neurons during ischemia come from changes in their chemical balance. First, without oxygen or glucose, the cell has no fuel. Cells aren't passive bags. They use energy constantly to maintain a different concentration of chemicals inside than outside. Cell membranes contain pumps constantly working to keep calcium outside and potassium inside. Without energy, these pumps fail. Calcium enters the cell, where it poisons the ability of the cell to produce energy. Calcium also causes breakdown of the cell's membrane, which of course allows even more calcium to enter.

A review describing all of these events is:


**Imbalances of calcium, sodium, and potassium within the cells.**

A specially designed enzyme system which simply sequestered these atoms, and only worked within the cell, would repair this problem. It's possible that repairing the pumping ability of the membranes would solve this problem too.

**Release of prostaglandins, leukotrienes, and other chemicals causing swelling.**

This only happens after ischemia, and causes swelling which cuts off blood flow to the brain. Any drug or other simple chemical system which deactivated these substances (and any others like them which may be also be released) would solve this problem. Repair after revival would consist of immediate provision of this drug system.
Box 7: Free radical damage

Free radicals are damaging chemicals made when the cell burns its food. Because of their role in aging, free radical damage is a very popular hypothesis. For ischemia, though, the situation is cloudy. Neurologists have disputed for the last 10 years about existence of free radical damage.

Two papers taking opposite sides in the dispute are:


One new class of chemicals, the lazaroids, may prove that free radicals are involved and provide a treatment. A paper on lazaroids is:


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Release of free radical chemicals.

Just like the drugs which sequester calcium ions, a simple drug system to deactivate these free radicals will answer to this problem. *                        *                        *

Devices able to carry out these repairs are not advanced instances of nanotechnology. One problem easy to neglect here, though, is simply that we must work out very specifically just what the problem is before we repair it. This takes time. Research will have to explore many different possible injuries before it finds a very small number, maybe only one, of actual injuries. Nanotechnological devices as research tools let us do much more sophisticated probing of cell injury. We may still need many years to probe the problem.

It might easily happen (remember aspirin and heart disease?) that we discover effective treatments which are simple, even banal. Yet we may have needed all our nanotechnology to make this discovery.

What about after six hours?

For ischemia up to six hours duration, 20 years of research has taken us far along the road to a solution. Reviving someone after six hours of ischemia simply isn't a problem within the ken of today's research. Right now in 1990 we know very little about what the injuries are. Ideas about repair are bound to change.
Still, we do know that cell membranes are seriously damaged. At some stage the brain cells simply won't support piecemeal additions and metabolic helps. A repair device would have to bring along its own genetic apparatus and protein synthesizing machinery. We can envision these as resembling bacteria. Their problem isn't really to recognize the damage and how to repair it. All of that can be done from our preexisting understanding of cell reactions combined with an ability to read off information from the target cell's own DNA. They will need their own genetic and synthetic machinery because the target cell can't provide any help on its own.

The first act of the repair cell would consist of rebuilding the target cell's membranes and membrane pumps. It would then rebuild, clear out, or even replace the target cell's mitochondria. By this time the target cell could start to function on its own. The repair cell could withdraw, leaving behind a repair system like that we've seen earlier.

Other damage

The preceding was an account of events during brain ischemia. The brain is not quiescent. The neurons are frantically trying to adjust to many damaging events. But they fail and cause even more damage by doing so. All of these events set the stage for more damage after attempts to restore circulation.

It is only after circulation returns that blood vessels and glial cells swell up to cut off circulation. Starved of energy, the neurons go into seizure activity, which uses up even more energy and leaves them in a worse state. Because they can't make more proteins, even normal metabolism wears them out and they slowly die.

Of course, by intervening in these events we can expect to prevent or counteract them completely.

Cell repair and cell technology

Death has many forms, ultimately each needing its own repair. Looking at only one major form, ischemic, we find it consists of a series of events lasting many hours and still only partly understood. During ischemia, more and more cell damage progressively occurs, to an increasing number of specific sites within a cell. Practical and real repair technologies will become sophisticated along a gradual range. At the near end they would look simply like special designer drugs, exactly the kind of drugs current pharmaceutical chemists invent. (The only distinction is that these drugs will act on novel sites not yet known). From single drugs, repair technology passes to interacting systems of drugs, from there to viruslike systems with some capability of directed response... and so on.

One major lesson of these ideas is of exactly how important gradual improvement is and should be to any serious plan to cure any kind of "death." We should not think of repair as a matter of fully sophisticated nanocomputers or nothing. Indeed, not to take the first elementary steps with elementary nanomachines (drugs, drug systems, gene transfer viruses) guarantees that advanced devices will never come, ever. Seeds never
planted never grow, no matter what technology exists elsewhere.

A second lesson makes the point that we need to focus not just on the technology needed for repair, but also on technology needed to find out how to repair. Electron microscopes haven't yet directly cured anyone of any illness. Their value lies elsewhere. Whenever we look specifically at some kind of death, the first and major step consists of exploring the problem and its causes. Before we find these causes, we must sift through fantastically many possible causes, only one of which will turn into reality, every one of which is equally real prior to our search. Once we find cures, all but one becomes forgotten history. Any nanotechnological tools helping our search have immense value even if they never come near a patient.

Finally and ultimately, no one else will bring us cures for conditions that only we consider as diseases instead of Theological Events. Not just the suspension itself, but every stage along the way will come not from faceless Scientists but from cryonicists and their own work. But then, we don't just have five minutes but five CENTURIES in which to find these cures. Perhaps even five millennia. Welcome, everyone, for your ride into history.

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THE GREAT GRAVE ROBBERY

by John Minahan

Book review by Mike Darwin

This was a very strange book for me to read. It is not poorly written, and it is not particularly the style that made it peculiar to read (although the author's style is a bit unusual); rather, it is the content of the book, or, more precisely its world-view that is the problem.

The Great Grave Robbery is of the mystery-thriller genre. The chief protagonist, a New York Police Department detective named "Little John," is a character who has appeared in others of Minahan's 18 or so books. In The Great Grave Robbery Little John is assigned to crack the mystery of the "largest case of industrial espionage on record." It occurred in 1966 when secret documents and 36 vials of cell repair viruses were stolen from the International Cryogenics Corporation in New York.

The thief's identity was known, but he disappeared at the time of the theft and did not reappear until 17 years after the statute of limitations for prosecuting the crime had expired. And where had he been during the interval between the theft and his reappearance in 1988? Why in cryonic suspension, of course. For, shortly after he absconded with the goods, he developed a then-incurable heart disease and was frozen. But now he is back, revived with the protocol stolen from International Cryogenics and repaired with the stolen cell-repair viruses and equipped with a new heart. Is it for real? Has cryonics really worked? Minahan weaves an interesting tale in classic whodunit style.

What was peculiar for me in reading this book (and what may not be peculiar for you) is that it evoked memories from childhood* and an eerie sense of disorientation that I can hardly describe. Perhaps the closest I
can come to communicating the feeling is to ask you to imagine yourself waking up one morning and going to the mirror only to find that you don't show up in it! Furthermore, everywhere you go, no one knows or sees you and every bit of evidence that you have ever existed has vanished.

Minahan's book affected me in that way because it gives a fictional history of cryonics right up to almost the present. It is a history which blends fact and fiction in a peculiar way and the sensation is almost one of stepping into an alternate universe. There is a Cryonics Institute run by Mr. and Mrs. Robert Erickson. There is a Cryonics Society of New York, there is a Trans Time/American Cryonics Society, and the Segall Beagle article from People magazine is reproduced (sans the picture of Miles) in toto.

But there is no Mike Darwin, or Jerry Leaf, or Hugh Hixon. Cryonics is still carried out with mortuary equipment. Indeed, Minahan's book is littered with complex technical descriptions of the perfusion process (circa 1968), the hardware, its operating principles, and even the Arrhenius Equation which describes the relationship between the rate of chemical reaction and temperature. The description of Erickson's basement cryonics facility in Oak Park, Michigan sounds as if it was lifted from the article which was most responsible for getting me interested in cryonics. "The Deep Freeze Scheme For Immortality," by J. F. Wilkinson appeared in the October, 1967 issue of True Man's Magazine and described one Robert Ettinger's basement cryonics facility in Oak Park. My father brought it home for me from work, and I have it still.

* Mike Darwin became acutely interested in cryonics at the age of 12 in 1967 and actively involved in cryonics in 1968. He participated in a human cryonic suspension when he was 17 years old. Thus, much of his childhood and all of his adult life has been spent as a cryonicist.

But in Minahan's story there is no Alcor. And consequently cryonics is marooned in time, at least from our perspective. The use of medical procedures never arrived for cryonics in Minahan's world. There is no high impulse CPR, no field extracorporeal support of patients with blood pumps and membrane oxygenators. There are no cerebral preservation medications, no bypass technology, no monitoring of cryoprotectant concentration or computer modeling of perfusion. Nothing Alcor has done, technically, legally, or scientifically shows up. It is a universe where Jerry Leaf and Mike Darwin did not introduce basic medical technology into cryonics. Indeed, it is a world where Alcor's founders, Fred and Linda Chamberlain, do not exist, with their perfusion machine and many other technical upgrades which came in the 1970s!

There is also no nanotechnology per se. The idea for tissue repair apparently came from a paper by Jerome White presented at the Second Annual Cryonics Conference held in Ann Arbor, Michigan in 1969. Consequently there is also no sign of the scenarios for repair by Thomas Donaldson, Eric
Drexler, Brian Wowk, Mike Darwin, or Ralph Merkle. Modesty aside, this means that the issue of repair seems more than a little deus ex machina, especially when compared to the detail present in the rest of the book.

At first, reading this book made me frustrated, and, yes, a little angry. It is not a good feeling to look into a mirror and find you don't exist. No doubt many of his loyal readers will buy Minahan's book. I think they will be in for a bit of surprise; this book will be rough sledding and probably far too technical for the typical reader of mystery-thrillers. It is sad that these readers will not get a more complete and, I might add, more optimistic picture of cryonics (not to mention more technically plausible).

It was especially frustrating for me to read this book because it was obvious that Minahan put a lot of time and effort into getting the technical details right and into trying to offer the most plausible scenario for repair he could come up with. This is rare in an author of fiction. Extremely Rare. Take it from one who knows from hard experience in dealing with novelists who want to write about cryonics.

After some reflection, I decided I didn't feel hostile about this book. A little wistful for what might have been, perhaps. But really, on balance grateful. Yes, grateful. Because, like Jimmy Stewart in It's A Wonderful Life, I got to see what the world might well be like if I and all the fine men and women of Alcor had never existed.

I'm glad we do exist. What's more, I'm proud that we do.

I have heard it rumored that the original manuscript for this book was written in 1968 or '69. This may explain much of the "eerie" datedness. A brief note in the front of the book explains that Minahan and Robert Ettinger are long-time friends and it is apparent that Minahan has relied heavily on Ettinger and The Immortalist for his "updating."

The Great Grave Robbery is pricey entertainment at $18.95 for 211 pages in hardcover. Because of its heavy technical emphasis I doubt if this will be one of Minahan's big sellers, so don't expect to see it in paperback (although in the publishing business you can just never tell!). Nonetheless, it was enjoyable reading and, for those of you Alcor members who have been plugging away at making cryonics work for even half as long as I have, you may well find it worth your $18.95 to find out what the world would be like without you.

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SPARSE DISTRIBUTED MEMORY

by Pentti Kanerva, MIT Press, 1988
book review by Thomas Donaldson

Many people, even those not working with computers, have noticed by now the growing interest in neural nets. "Neural net" is a general term for a special design of both computer and the software running on it which imitates, in some respects, the behavior of a brain rather than a computer. They are important to computing because brains so far have vastly outperformed computers in some simple tasks.
You can recognize that a faded, scratched photograph taken from an odd angle is a photograph of your brother. You do so in less than a second, so quickly that you don't even have to (consciously) think about it. Yet this ability to recognize has defeated armies of programmers with Cray computers for 30 years. How can that be, when 20 years ago all the Authorities were proclaiming how simple it would be for a good enough computer to imitate and surpass human beings? No one yet has built a robot able to walk about in a natural setting for 5 minutes without getting itself into a ridiculous fix: trying to climb the nearest tree in the belief that it is the sidewalk, for instance. Something has been very wrong.

Devices based on neural nets have begun to solve these problems now. Along the way they have told us a good deal about computing and about ourselves. More is almost certainly to come. But they work very differently from "normal" computers.

A normal computer consists of one special (by now highly evolved and complex) chip, the CPU, together with a block of memory chips (there are other support circuits, of course), keyboard, disk drive, and other input-output devices. When you turn on (the jargon for this is "boot") the computer, the CPU starts reading memory like a book, at one fixed location. Assuming all went well with the design, that location contains commands which tell it to "load" other series of commands ("programs"). It does this by reading them into its memory; once complete, it starts reading those commands and executing the ones it has read. At that point it may read in other material ("data") which do not act as commands, but the passive objects of these commands. Think of a word processor: first it must read in the word processing program, then all the text it is to process. That text doesn't ordinarily issue commands to the computer. It is the material on which these commands work.

This paragraph sketches the general design of a generic "normal" computer. One major element is that "memory" is always a kind of clean slate, onto which any set of valid commands can be written. (Just like us, the CPU only understands a particular "language" of command. We call this language the "assembly language" of the computer. By the time most people use their computer, programmers have provided means to translate the people commands into the "assembly language" of the particular chip. That translation is called "programming"). And onto this memory, too, any set of valid data may be written.

Another generic trait of the "normal computer" is that there is only one CPU chip reading data and reading, then executing commands. Among other results, this means that a "normal computer" cannot work faster than the electrical circuitry on its CPU. This is fast but not infinitely fast.

A neural net proceeds quite otherwise, and so differently that some may wonder if it even qualifies as a computer. A neural net consists of many processing chips (often the same kind of CPU chip used normally), each one given a small bit of memory. But most important, each one is linked to the others in a network, so they can send messages back and forth to one another. Sometimes the amount of memory an individual CPU in the neural net can directly read (without having to ask another one to read its memory and send it the result) is quite small compared to ordinary computers. Sometimes, in fact, these processing chips would not qualify as CPU chips at all. They may not have any general ability to read and respond to commands, but only a fixed ability, nonchangeable or only slightly
changeable, to respond to all the inputs they receive from the other chips to which they are connected.

Network? Processing chips? Where have we heard this before? In barest terms, I am describing the structure of brains. Neurons are the processing chips, connected in a network, which we call the axons and dendrites of each neuron. A neuron sends messages through its (usually but not always single) axon, and receives them from its dendrites. It changes due to the messages it receives and their frequency.

At this point, we begin to see how "neural net" is just as generic as "computer." Its designers can choose individual processors with many different kinds of response to incoming messages. The behavior of the entire neural net always results from the action of all of these processors combined, not their individual response. Just like our brains seem to do.

Kanerva, the author of the book under review, has written a small, elegant exposition of one particular way to design a neural net. Be warned: it's full of mathematics, even though Kanerva has written it far more clearly than most books containing math. It's not for every cryonicist. Yet Kanerva comes through it not as a judge but a student, honestly trying to understand his subject fully and completely. And then this student tries to explain what he has learned.

What makes this book interesting to cryonicists is that Kanerva also proposes that his version of neural net might explain the working of our brains, not just generically but specifically. It is exactly at that point that I personally felt very unhappy with his ideas and his results.

My unhappiness came from both my understanding of how a real, true neuron responds, and from Kanerva's theory. In Kanerva's version of a neural net, many messages coming through one particular message channel (i.e. synapse) will increase the response of the neuron even to messages on other channels. That does not seem to be true at all. We can notice changes with learning in the synapses through which messages pass, but no sign of a general increase in response to ALL incoming messages.

And though Kanerva shows himself honest and careful, within his limits, he also suffers terribly from a malady I have seen far too often among computing people who try to understand our own brains. If this book seriously proposes to explain our own learning, where is the biology, the biochemistry, the comparison with facts? The book contains very little, which is exactly the point. Its high point in this regard is to allude to the connection pattern of nerves in the cerebellum, which may indeed work like Kanerva's neural net (I hope this idea works out, too). Yet our brains contain many other regions, lacking that pattern of connections, with apparent algorithms for learning which do not match Kanerva's.

This malady has many serious consequences. Where else have we heard of a group of people who decided that they understood the world well enough that they needn't bother with ever checking their ideas against it? The classical Greeks (but then, they were only the first). And that malady comes with tremendous arrogance and outright refusal to listen to ideas from other quarters, from the barbarians. Why bother to understand those odd, ignorant, unwashed people, with their incomprehensible language and strange ideas? Such arrogance leads on, as it must, to a great collapse.
From his book Kanerva does not seem personally arrogant at all. I'm sorry that he got infected, quite unconsciously, with a vicious meme. And for those willing to face his math, he's written a very good exposition of how at least one kind of neural net can work. There must be others, we know, not only from the work of other people but from our own brains. As for brains, his ideas about the cerebellum are very interesting. But his biology deserves pickling in salt before consumption.

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SCIENCE UPDATES

by Thomas Donaldson

NEURON LEARNING AND MEMORY DISTINGUISHED IN CHEMICAL FORM

The phenomenon of long term potentiation (LTP), ever since its discovery, has received intense attention by the small group of neuroscientists trying to unravel the biochemistry of memory. LTP refers to the fact that neurons in slices taken from a variety of brains will react differently to a prolonged stimulus than to a short one. Afterwards, for hours and perhaps days, the particular synapse carrying this stimulus will respond more easily to a single stimulus.

The suggestion that LTP may help explain neuron memory comes to mind at once. That suggestion has led to tremendous effort on the part of several neuroscientists, who are still working on it. As it stands, LTP is a phenomenon rather than an explanation.

Among the points discovered are: for LTP, the neurotransmitters activate one particular receptor chemical, the NMDA receptor, in the cell membrane at the synapse. The activation involves changing the structure of the NMDA receptor to let calcium ions into the cell. At least two enzymes which react to calcium, calmodulin and PKC (protein kinase C) will then change the conformation of some other enzymes (the kinases). These kinases do something still unknown to create LTP (narrowly speaking, we do know what the kinases do. They add phosphate to other proteins. But which proteins and with what effect??).

We know these kinases are involved because LTP cannot occur if we put other chemicals into the synapse to turn them off.

Recently another row of bricks in our understanding of memory was laid by a group from Stanford University headed by Richard Malinow (Science, 245, 862 (1989)). I have described above, in very schematic form, some of the reactions involved in producing LTP. Fine, so these reactions are involved in producing LTP. But do they play any role in the process by which LTP happens, once it's been produced?

Just as with other papers on LTP, the authors used a technique capable of measuring voltages and delivering biochemicals to an individual neural synapse. Malinow and his coworkers used this technique to answer the question asked above. They could measure voltages (and thus the existence of LTP). They could also deliver test chemicals very precisely. Given this technique, they used a variety of inhibitor chemicals to stop this
memory chain from working at one point or another. This would tell them exactly what biochemical is needed to achieve what effect. The inhibition here took place at the receiving end of the synapse (the postsynaptic cell).

Here is a summary of what they found. Two enzymes are involved in producing LTP: PKC (protein kinase C) and CaM-KII (calcium-calmodulin dependent kinase II). Both are needed to make LTP (if they inhibited only one, LTP could not happen).

But very interesting: if the particular synapse has already acquired LTP, then we could stop both PKC and CaM-KII from acting and the LTP would remain just as before. At least as interesting: if we inhibited these kinases on both sides of the synapse rather than just one, the synapse would no longer show LTP. Even more interesting for cryonicists: yes, we could turn off LTP by bathing both sides of the synapse with at least one inhibitor chemical, H-7. But that inhibition would be reversible.

(If LTP is not a red herring!) these experiments and many others on neuron memory tell us a lot about mechanisms by which it is first produced and afterwards expressed. As mentioned before, the synapse structure on the receiving side contains 20% to 40% CaM-KII, which must play a major role (whatever that role is). But these experiments also suggest that none of these chemicals actually is the mode by which LTP persists. They answer about "potentiation" but not the "long term" part at all. But patience, that too will come.

PICTURES OF MEMORY

We have already seen that complex biochemical processes underlie memory formation. These processes involve several known chemicals and others unknown: calcium, PKC (protein kinase C), CaM-KII (calmodulin-dependent kinase II), calmodulin, c-fos, zif/268, and the unknown others. But how does this chemistry inside single neurons relate to the global brain memory which is our consciousness and our self? Theories lie about in every book and journal, but experiments are scanty.

A recent paper in Science, (245, 866 (1989)) by Daniel Alkon and others at the NIH stands out in this way because it attempts some global measurement of changes occurring with memory. Out of their attempt come some pictures, showing distribution of chemical changes in rabbit brains with learning.

The specific memory they used was classical conditioning of rabbits to close their nictitating membrane to respond to a specific tone. The chemical in question, PKC (protein kinase C, already implicated in memory). Before Alkon and his colleagues did their work, they knew that with learning PKC would move from the cytoplasm of the neuron to the actual cell membranes. They had chemicals they could use to distinguish these two different forms. By labelling these chemicals radioactively they could show just where the PKC was distributed, in cytoplasm or on the cell membranes, before and after this learning event.

The most interesting issue, here, was of course the physical
distribution of PKC after learning. With their dyes, the difference after conditioning as shown by their color pictures was dramatic. In brain slices taken before conditioning, much less PKC existed on the cell membranes all through the CA1 to CA3 regions of the rabbits' hippocampus. Three days afterwards, their pictures show a dramatic rise. It's very clear, among other points, that millions of hippocampal cells were involved in this simple classical conditioning. The changes do show a pattern, which someday we will specify with much more precision than we can now.

Alkon and his colleagues themselves point out how the hippocampus is only a small part of the brain. They have not even tried to measure changes elsewhere. They think changes in the cerebellum would be particularly important for showing the exact locations were coding for the conditioned stimulus took place.

Some theories of memory have attempted to guess our capacity for memories by simple measures which try to make a translation between computer bits acquired and rate of acquisition. Results such as these of Alkon and his colleagues do suggest problems with such a simple view.

But it's easy to criticise the first steps at anything. These results are much more significant because they start to work out, even on a very global, low resolution scale, not only WHAT but WHERE the changes take place when we acquire a memory. Even now we can begin to guess ways to someday measure these changes neuron by neuron, and eventually to read off, reversibly, all the changes, marked by location, into a database. The number of questions equals or exceeds the number of neurons.

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ADVERTISEMENTS AND PERSONALS

The Alcor Life Extension Foundation and Cryonics reserve the right to accept, reject, or edit ads at our own discretion, and assume no responsibility for their content or the consequences of answering these advertisements. The rate is $5.00 per line per month (our lines are 90 columns wide). Tip-in rates are $45 (already printed); or $90 (printed one side) or $135 (printed both sides), from camera-ready copy. Advertisers in tip-in material must be clearly identified.

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Life Extension Fiction: Including such authors as Lee Corbin, Thomas Donaldson, Cameron and Leigh Rockwell, and other cryonics society members. Send for a free sample issue. LIFEQUEST; P.O. Box 18690; South Lake Tahoe, CA 95706.

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MEETING SCHEDULES

Alcor business meetings are usually held on the first Sunday of the month. Guests are
welcome. Unless otherwise noted, meetings start at 1 PM. For meeting directions, or if you get lost, call Alcor at (714) 736-1703 and page the technician on call.

The MAY meeting will be at the home of:

(SUN, 6 MAY, 1990) Saul Kent and Jo Ann Martin
16280 Whispering Spur
Riverside, CA

DIRECTIONS: Take the Riverside Freeway (Hwy 91) east to Riverside and get off going south (right) on Van Buren Blvd. Whispering Spur is south of the freeway four miles, and 1.0 miles beyond Mockingbird Canyon Rd., on the left. 16280 is the second house on the right, at the end of the white fence.

This meeting will be the same weekend (May 4-6) as the Reanimation Conference at the Clarion Hotel, Ontario Airport, Ontario, CA.

The JUNE meeting will be at the home of:

(SUN, 3 JUN, 1990) Brenda Peters
8150 Rhea
Reseda, CA

DIRECTIONS: Take the San Diego Freeway (Interstate 405) north into the San Fernando Valley, to Roscoe Blvd. Go west (left) on Roscoe 3-4 miles. Rhea is 2 blocks past Reseda Blvd. Turn south (left) on Rhea, which has a geodesic dome church on the corner. 8150 is the second house in the second block, on the left.

The JULY meeting will be at Dave and Trudy Pizer's, in Wrightwood, CA, concurrent with the Venturists' Cryonics Conference

* * *

Alcor members in the San Francisco Bay area have formed an Alcor chapter, and are aggressively pursuing an improved rescue and suspension capability in that area. Meetings are generally held on the second Sunday of the month, at 4 PM. Meeting locations can be obtained by calling the chapter's Secretary-Treasurer, Thomas Donaldson, at (408) 732-4234 (home), or at work, (415) 593-3200 (ask for Thomas Donaldson).

The MAY meeting will be held at the home of:

(SUN, 13 MAY, 1990) Roy Yowell
12 Skyline Crest
Monterey, CA

The JUNE meeting will be held at the home of:

(SUN, 10 JUN, 1990) Keith Henson and Arel Lucas
1794 Cardel Way
San Jose, CA

* * *
The New York Cryonics Discussion Group of Alcor meets on the third Saturday of each month at 6:30 PM, at 72nd Street Studios. The address is 131 West 72nd Street (New York), between Columbus and Broadway. Ask for the Alcor group. Subway stop: 72nd Street, on the 1, 2, or 3 trains.

The meeting dates are as follows:

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If you live in the New York, Philadelphia, New Jersey, or Boston areas and would like to participate in the rebirth of New York cryonics please contact one or more of the following people:

Gerard Arthus (516) 474-2949  
Curtis Henderson (516) 589-4256

A Las Vegas, NV chapter of Alcor is in the process of organizing. The next meeting is at 7:30 PM, May 7 at:

Vegas Bayou restaurant  
1290 E. Flamingo Road  
Las Vegas, Nevada

We have the use of the Bayou conference room indefinitely for meetings on the first Monday of every month. Anyone visiting Las Vegas is welcome to give organizer Russ Whitaker a call or a drop a note to:

Russell E. Whitaker  
1350 E. Flamingo Rd. #247  
Las Vegas, NV 89119  
(702)366-7958

Other Events Of Interest

-- The Reanimation Foundation will hold a Reanimation Conference May 4-6 at the Clarion Hotel at Ontario Airport, Ontario, CA. See the February, 1990 Cryonics, or call 800-841-5433 for details.

-- Alcor will be present at the Space Development Conference, Memorial Day weekend (May 25-28) in Anaheim, CA. Call Alcor for details. Volunteers are also needed to man our exhibit.

-- A July 4 Cryonics Festival sponsored by the Venturists will be held at the Mountain View Lodge in Wrightwood, California. The weekend features outdoor activities, speakers from Alcor and the American Cryonics Society and noted science fiction author Gregory Benford. For information contact The Venturists, P.O. Box 458, Wrightwood, California 92397, phone (619) 249-3553.

-- Conference On Biostasis And Reentry sponsored by Lifepact will be held August 24-26 at the Asilomar Conference Center, near Monterey, CA. For
information, contact Linda or Fred Chamberlain at (916)542-1331 (days) or
(916)577-4746 (eves); or P.O.Box 18698, South Lake Tahoe, CA 95706.

-- There will be a European Cryonics Conference October 26-29 at Gatwick
Airport (London). This will include a tour of Alcor, U.K.'s new facility. See the April, 1990 issue of Cryonics for details and contact Saul Kent at
16280 Whispering Spur; Riverside, CA 92503; USA for additional
information.