Robert C. Randall is the president of the Alliance for Cannabis Therapeutics and a legal medicinal marijuana patient. Marijuana can make the difference between years of functional vision or blindness for people afflicted with glaucoma. I know this firsthand. Marijuana has given me nearly 20 years of vision. Despite marijuana's therapeutic value in the treatment of glaucoma, only three glaucoma patients in the United States currently receive legal, medically supervised access to marijuana. In each instance, marijuana has dramatically improved medical control over glaucoma and provided safe, effective relief from progressive nerve damage.

If glaucoma were a rare disorder or current therapies were more successful in controlling the disease, marijuana's medical use might seem unnecessary. Glaucoma, however, is commonly cited as the leading cause of blindness in the United States. While conventional medical therapy is successful in dealing with the majority of cases, the failure of existing medical treatments compel significant numbers of Americans into risky surgical treatments.

These problems are not unique to the United States. Outside the postindustrial states of North America, Europe, Australia, and Japan, glaucoma is even less likely to be successfully treated. Data also suggest the incidence of glaucoma is racially disproportionate; the disease may occur eight times more frequently among people of color. This combination of increased incidence and restricted access to modern and expensive synthetic medical treatments suggests marijuana may have a very important role to play in glaucoma therapy.

Briefly stated, glaucoma occurs when elevated fluid pressure inside the eye, commonly called intraocular pressure, damages the optic nerve. While the precise mechanics of glaucoma remain elusive, all treatment for glaucoma, medical or surgical, centers on reducing intraocular pressure.

Marijuana and Reduced Ocular Pressure

Unlike many of the medical uses for cannabis that were identified in earlier times, marijuana’s role in the treatment of glaucoma is much more recent. While there are indications that ancient cultures were aware of cannabis’ ocular effects, all of the glaucoma-specific data dates from the early 1970s.
Early Studies

In 1970 the University of California in Los Angeles (UCLA) initiated a series of studies on marijuana's effects on various biological systems. One aspect of this study sought to address marijuana's impact on pupil size. At the time, it was commonly believed that marijuana caused pupillary dilation, and law enforcement interests wanted to determine if pupil dilation could be used as an indication of marijuana use.

Dr. Robert S. Hepler of the Jules Stein Eye Institute at UCLA was asked to conduct these studies. Hepler agreed and received a constant flow of research subjects from the umbrella UCLA project. From photographic and other evidence it quickly became clear that the theory of pupillary dilation was wrong. In fact, smoking marijuana actually causes a slight pupillary constriction.

Pupillary constriction is a common consequence of several drugs that lower intraocular pressure and are frequently used in glaucoma therapy. This effect is particularly obvious in short-term and long-term miotics like pilocarpine and phospholine iodide. Hepler made this connection and began to systematically check the intraocular pressure (Kw) of research subjects in the UCLA program. He quickly discovered that marijuana induced a rapid, very significant decline in IOP in both the normal and glaucomatous eye and that the effect was dose-related. Hepler filed the first report of his finding in a letter to Journal of the American Medical Association in September 1971 (Hepler and Frank 1971).

For the next five years Hepler and his research technician, Robert Petrus, continued to explore marijuana's Kw-reducing utility. Briefly summarized, they reported marijuana significantly reduced IOP in approximately 80 percent of their subjects. The reduction in ocular pressure was quite significant, usually between 25 percent and 50 percent of baseline pressure. The IOP-lowering effect of marijuana occurs approximately 45 minutes to one hour after smoking and lasts from three to five hours.

The prospect of a plant that is inexpensive to produce and that could significantly improve the medical welfare of people afflicted by glaucoma was not, however, aggressively pursued by the federal government or the ophthalmic establishment. Federal law specifically defines marijuana as a highly dangerous, medically useless substance. Given a choice between upholding this legal fiction or working to meet the real treatment needs of seriously ill Americans, bureaucrats at the FDA, DEA, and the National Eye Institute (NEI) preferred fiction.
Glaucoma: A Patient's View

Written by Robert Randall

Personal Experience

I was oblivious to these events when I was diagnosed in September 1972 as having glaucoma. At the time of diagnosis glaucoma had already destroyed the central vision in my right eye and had greatly eroded peripheral vision in my left eye. My IOP was 42 mm Hg, more than double the highest "normal" pressure of 20 mm Hg. Prognosis at diagnosis was three to five years of remaining vision. I was 24 years old.

My physician, noted ocular pathologist Dr. Ben Fine of Washington, D.C., honestly discussed my various treatment options, and I was immediately placed on conventional glaucoma control drugs, beginning with pilocarpine. The visually distorting effects of pilocarpine were so severe that I became temporarily disabled. Within a year of diagnosis my use of pilocarpine escalated dramatically. Initially I was prescribed 0.5 percent pilocarpine once a day. By September 1973 I was using 4 percent pilocarpine four to six times daily.

Unfortunately, as Fine had predicted, even this rapidly accelerating use of pilocarpine failed to adequately control my elevated IOP. I was continuing to lose my vision. Additional medicines were prescribed, including topical epinephrine and oral diuretics. While each of these new medicines provided short periods of IOP reduction, my ocular pressure quickly escaped medical control.

Then in the fall of 1973 someone provided me with two marijuana cigarettes. I had smoked marijuana frequently while in college. And I had noticed already then that marijuana helped ease visual symptoms my doctors at the time referred to as "eye strain." Upon graduating from college, however, I moved to Washington, D.C., and stopped smoking marijuana.

After having been diagnosed with glaucoma, I learned that these visual symptoms were, in fact, indications of dangerously elevated IOP. These symptoms typically began with a slight blurring of vision. If my IOP continued to increase, this blurring was followed by the appearance of tricolored haloes around sources of light. In extreme but not uncommon circumstances these haloes were followed by an onset of "white blindness," a condition in which IOP is so elevated it causes the light coming into the eye to diffuse. When this occurs IOP is very elevated, and one can become functionally blind, and additional damage to the optic disk often follows. In effect,
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one is watching oneself go blind.

I was experiencing tricolored haloes that evening in 1973 when I smoked one of the marijuana cigarettes I had been given. I had no great expectations and was certainly not smoking marijuana because of my glaucoma. Forty-five minutes later, however, I noticed that the tricolored haloes were gone. Then I remembered my college experiences with "eye strain." I was suddenly overwhelmed by the fact marijuana was medically helpful.

The next morning I reconsidered. It is, after all, difficult to believe that an illegal weed one associates with pleasure is somehow going to save one's sight when all the modern pharmaceutical drugs have failed to work. It took me six months of careful trial and error evaluation to accept marijuana's important role in my medical care. By the summer of 1974, however, it was very obvious that marijuana was having a profound and beneficial impact on my disease. For the first time my IOP came under stable medical management. And for the first time visual field analysis indicated that I was no longer losing vision.

Illegal Treatment

Against this promising backdrop, I confronted a stark problem that still plagues most patients who need marijuana for medical purposes. It is illegal for physicians to prescribe or for patients to smoke marijuana. So I was forced to purchase unregulated supplies of marijuana at illegal, prohibition-inflated prices. Trying to afford enough marijuana to meet my medical need—about three cigarettes daily—was ruinously expensive. In addition, the criminal market in marijuana means that supplies are not always available.

In an effort to compensate for these difficulties, I decided to grow enough marijuana to meet my medical needs during those times when I could not afford or find marijuana in the street market. In 1974 I lost my first few marijuana plants to spider mites. In 1975, however, I succeeded in growing six beautiful marijuana plants on my sun deck. These were coming along well when I was arrested in August 1975 in the District of Columbia for the crime of marijuana cultivation. I immediately informed my attorneys that I was smoking marijuana for medical purposes, to control my glaucoma. They said I would have to “prove it.” So that is what I set out to do.
Building a Case

Within weeks of my arrest I discovered that the United States government also knew of marijuana's important TOP-reducing properties. Then I became aware of Hepler's extensive studies of marijuana's use as a glaucoma control agent. In December 1975 I spent 13 days undergoing controlled medical test at the Jules Stein Eye Institute. The first stage of this controlled medical evaluation assessed the utility of conventional glaucoma control agents. In very quick order Hepler concluded that if left on standard glaucoma control drugs, I would become blind. Even with the use of all available glaucoma control drugs my MP was consistently elevated for much of the day. In the evening my IOP often spiked above 40 mm Hg, Hepler then tested my IOP response to oral, synthetic delta-9-THC pills similar to those now marketed under the brand name Marinol. The synthetic THC failed to reduce my IOP.

Finally, Hepler tested my IOP response to prerolled marijuana cigarettes prepared by the National Institute on Drug Abuse (NIDA). Initially, a dose of one cigarette failed to reduce my IOP. Both Hepler and I found this puzzling. In my experience I usually smoked less than a complete cigarette to achieve IOP reduction. Perplexed, we agreed to increase the dose. I was told to smoke as much marijuana as I cared to. I smoked seven cigarettes in an hour. My IOP decreased dramatically.

Over the next few days Hepler worked to establish a marijuana dose that would effectively reduce my IOP on a consistent basis. By the conclusion of the UCLA study, it was clear that I would need between eight to ten NIDA marijuana cigarettes daily to achieve desired levels of IOP reduction throughout the course of a day.

After reviewing the UCLA data, my physician, Dr. Fine, recommended a second controlled medical experiment to confirm his and Hepler's conclusion that my IOP was beyond the reach of conventional glaucoma therapies. This second, confirmatory study was conducted over a period of six days at the Wilmer Eye Institute at Johns Hopkins University.

At the end of the six-day, in-hospital evaluation, Wilmer ophthalmologists agreed with the UCLA findings and concluded that even under maximal medical treatment, employing all available glaucoma control drugs in combination and at the highest recommended dosages, my disease could not be adequately controlled. The Wilmer physicians concluded that if I were left on these therapies, I would quickly lose my remaining vision. They recommended immediate surgical intervention to save my sight.
Nearly all credible ophthalmologists would agree that surgery for glaucoma entails very real risks. In many instances, such surgery fails to reduce IOP. Glaucoma surgery can also trigger the development of cataracts or result in sight-destroying infections. One study suggests that nearly one-third of patients suffering from "end-stage" glaucoma are blinded as a result of surgery.

Fine agreed with Hepler that surgical intervention would probably destroy the small island of healthy optic nerve that gave me functional vision. After carefully reviewing my medical history and the results of the controlled medical studies conducted at the Jules Stein Eye Institute and the Wilmer Eye Institute, Dr. Fine concluded it would be medically unethical to deprive me of therapeutic access to marijuana.

Medical Necessity

In July 1976 I went on trial in the District of Columbia accused of the crime of growing marijuana. In defense, my attorney, John Carr, argued that any sane man who knew marijuana could help to retain his sight would break the law to obtain the marijuana he medically required. In November 1976, the court agreed and ruled that my use of marijuana was not criminal, but an act of "medical necessity." I was found not guilty. In the same month, federal drug control agencies began providing me with legal, medically supervised access to marijuana for use in the control of my glaucoma.

From November 1976 through January 1978 I received marijuana through Dr. John C. Merritt, an ophthalmologist at Howard University in Washington, D.C. Merritt ignored my previous medical records and started from scratch. First, I was tested on conventional glaucoma control drugs. These failed to adequately reduce my IOP. Next, Merritt tested my IOP-response to orally administered synthetic THC pills. These also failed to reduce my ocular pressure. Finally, Merritt tested my IOP-response to smoked marijuana. Marijuana significantly reduced my ocular tensions to below 20 mm Hg. After fine-tuning my dose, Merritt determined that I required daily between eight to ten 0.9 gram prerolled NIDA cigarettes of 2 percent THC potency or greater to control my elevated IOP.

Merritt acted as my treating physician for a period of 14 months. During this period I routinely
visited his office for evaluation of my IOP-response to marijuana therapy. In addition, my IOP was monitored at home using a Schiotz tonometer. At times this monitoring was very extensive and involved IOP checks every half hour. After 14 months of therapy, Merritt concluded that marijuana effectively lowered my IOP and that the resulting decrease in ocular tension was preventing further damage to my optic nerve.

Despite Merritt's findings, federal agencies disrupted my medical access to legal supplies of marijuana in January 1978. In part this was in retaliation for my refusal to keep my medical access to marijuana a secret. It was also when Merritt left the Washington area. It should be noted that this disruption in access to marijuana occurred despite the fact that another board-certified ophthalmologist was ready and willing to monitor my medical use of marijuana.

In May 1978 my legal access to marijuana was restored as the result of an out-of-court settlement. On the basis of this out-of-court settlement I was guaranteed legal, medically supervised access to government supplies of marijuana of medicinal quality. Under this arrangement my private physician monitors my condition and writes prescriptions for marijuana that are honored by a designated pharmacy in the Washington, D.C., area.

The legal settlement specifically prohibits federal agencies from manipulating my medical care and from forcing me into unethical research programs. This legal settlement has provided me with FDA-approved medically supervised access to marijuana as a glaucoma control drug for nearly two decades. Employing marijuana as a mainstay drug, I have been able to eliminate several far more dangerous glaucoma control agents, including timolol and oral diuretics while still retaining stable control over my ocular pressure.

The resulting therapy has afforded me continuous control over elevated IOP for nearly two decades. My IOP now usually falls in a range from 12-18 mm Hg. Computerized visual field analysis consistently demonstrates that there has been no significant progression of sight loss since I began legally receiving marijuana. By any standard of glaucoma assessment marijuana has successfully stopped the rampant progression of sight loss.
I would be the first to admit surprise at this outcome. In 1976 when I was fighting for the legal right to smoke marijuana, I anticipated that like other glaucoma control drugs, marijuana would work for a brief period of time and then fail. But that has not been the case. As I finish this chapter in the spring of 1996, marijuana is providing the same degree of IOP-reduction as it did in 1976. Moreover, this therapeutic benefit has been achieved without any obvious mental or physical side effects.

Official Denial

While a marijuana-based therapy has been highly successful in helping to retain my vision, the FDA and other federal agencies, including the National Eye Institute (NEI), have virtually ignored marijuana's important role in glaucoma therapy. In the late 1970s the NEI poured nearly $2 million into efforts to develop synthetic THC-based topical eyedrops (Colasanti et al. 1984a, b). The resulting product was an abysmal failure. Not only did the NEI's THC-based eyedrops fail to reduce IOP, but the oil-based eyedrops caused severe ocular irritation. The NEI dropped additional research in this area in the early 1980s. Significantly, the NEI and FDA have refused to aggressively pursue marijuana's IOP-reducing properties.

The failure of the THC-based eyedrops could have easily been predicted. As early as 1974, Dr. Mario Perez-Reyes at Research Triangle Park in North Carolina reported that at least five chemicals in marijuana are responsible for the plant's IOP-reducing actions. Significantly, delta-9-THC is not the most impressive IOP-lowering agent in marijuana.

Other researchers, including Keith Greene, report chemicals not soluble in marijuana may also significantly contribute to reduced ocular tensions. Quite obviously, marijuana smoke contains numerous ingredients that are responsible for the plant's IOP-lowering effect.

Current Situation

At the present time only three Americans afflicted by glaucoma have legal, medically supervised access to marijuana. Significantly, the other two patients, Elvy Musikka and Corrine Millet, also report that marijuana has effectively lowered their ocular pressures and successfully prevented further erosion of their vision.
Despite these promising facts, United States law still prohibits marijuana's prescriptive medical use. Over the past decade, while United States researchers have ignored marijuana's important role in glaucoma therapy, investigators in the West Indies and elsewhere have been less reluctant to explore the plant's therapeutic usefulness. In particular, the work of Dr. Manley West in Jamaica has resulted in the creation of topical, cannabis-based eyedrops (Canasol) that appear to have some value as an IOP-reducing agent.

Over the years I have spoken with several individuals who have explored Canasol as a therapeutic agent. They consistently report the topical marijuana eyedrops can successfully reduce IOP. However, they also report concern over the eyedrops' long-term use. In one case the patient reported Canasol worked well for several months, then abruptly failed.

While I find West's work exciting I am not prepared to risk my vision on Canasol. Speaking personally, I already know that smoking marijuana successfully, consistently, and safely reduces my IOP. After more than 20 years of stable and reliable treatment, I would be foolish to abandon a therapy that I know works well and replace it with one that might or might not work. In short, I do not have enough remaining eyesight to risk the stability of my current care for a treatment that is uncertain and, therefore, risky.

Clearly, however, Canasol may have an important role to play in glaucoma therapy. And there may be very real advantages to a topical, marijuana-based agent. Some people, for example, do not wish to smoke. Others may find eyedrops less troublesome to use. In a rational world the decision to use marijuana or Canasol or some future cannabinoid-based synthetic to lower IOP should be made by treating physicians and their patients, not by vice cops and bureaucrats.

Finally, it is important to remember that the goal of successful glaucoma treatment is the preservation of sight. If this medical objective can be successfully achieved through the use of smoked marijuana, then smoked marijuana should be legally available, by prescription, for use by those patients who choose to use it.

References
