

Clinical Guidelines for Ibogaine-Assisted Detoxification

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Particularly, we are indebted to the dozens of therapy providers who have uncovered the knowledge that is found within these pages, and to those brave souls who have undergone life-changing experiences with ibogaine, who continue to inspire this project. There are too many to name.

It goes without saying that this therapeutic community is indebted, perhaps more than it can ever know, to the millennia-old use of iboga in traditional cultures, and to those who continue those vibrant traditions today.

i. Introduction

Ibogaine is a naturally occurring psychoactive substance found in a variety of plant species in the Apocynaceae family, principally *Tabernanthe iboga*. It has demonstrated efficacy in the treatment of various substance use disorders (SUDs), particularly for opioid withdrawal management and the reduction of the associated post acute withdrawal symptoms (PAWS) (Alper 1999, Brown 2013).

For this reason, ibogaine continues to generate a growing interest among medical professionals as well as the general public, especially those seeking alternatives to conventional modalities of treatment for SUDs.

This promise accompanies recognizable physical and psychological risks. Ibogaine has not yet been developed as a prescription medicine. However, some countries have explored or are exploring experimental prescription frameworks that make it available for use in detoxification. Other countries have implemented strict bans or restrictions against its use, largely without recognition of its potential medical benefit. In many other regions, ibogaine is used either under the auspices of “compassionate care,” or otherwise as an unapproved treatment.¹

It has been noted that the lack of standardized clinical practices in the experimental environments in which ibogaine therapy is generally practiced presents increased risks associated with its use (Alper 2007). The purpose of these guidelines is to consolidate existing knowledge gathered from thousands of treatments conducted since the early 1980s in a variety of medical and non-medical settings.

Most of the information presented in this document is supported by published research. Elsewhere, it is derived from various clinical experiences, synthesized through extensive interviews with therapy providers.

It is our hope that the information presented herein will prove useful for medical professionals who wish to understand current clinical practices; in the development of protocols for clinical trials; and to policy makers who wish to review potential avenues for effective regulation of ibogaine-assisted detox as a prescription medicine. Finally, we hope that it will eventually lead to the development of comprehensive minimum standards of care in the future.

Risk Management

This methodology of risk management used in these guidelines is based on the International Standards Organization’s ISO31001: Enterprise Risk Management. This document uses a definition of risk outlined in ISO Guide 73, which calls it “the effect of uncertainty on objectives.” This effect has the potential to be positive or negative, but its effective management is crucial for ensuring positive outcomes.

Scope of this Document

This document was produced in an effort to identify the known risks associated with the use of ibogaine, and to present guidelines to manage its risks in a clinical setting. Although some other applications of iboga and ibogaine – as well as its metabolite noribogaine, and an ibogaine analog, 18-Methoxycoronaridine (18-MC) – are being explored, the scope of this document will refer specifically to the medical use of purified ibogaine hydrochloride as an aid in detoxification for opioid use disorders.

While this guide will focus on ibogaine's ability to mitigate opiate withdrawal symptoms, it is practical to note that a significant number of patients with opioid use disorders are also polysubstance users and/or have other psychological or physical co-occurring disorders. Many of these issues are also discussed throughout this document.

A significant amount of further research is required in order to completely understand the nature of the ibogaine experience and its implications in the treatment of SUDs. This study will require comprehensive collaboration between various disciplines, including psychology, sociology, ethnopharmacology, philosophy, and comparative religion, amongst others. These guidelines do not function as a textbook or a manual, but the considerations outlined may be useful for medical professionals in the development of clinical protocols.

Context & Continuing Care

While the fields of psychiatry and allopathic medicine primarily focus on a biological foundation of SUDs, there are a complex bio-psycho-social phenomena affecting addiction that are crucial to consider in planning a complete recovery (Tatarsky 2007, Mate 2009, Alexander 2010).

Simply addressing a SUD with ibogaine can sometimes be enough to help a patient make desired changes in their lifestyle and drug use if and when they are ready and equipped for this change. But much more often further ongoing psychological support is necessary. There is evidence from observational research following ibogaine therapy that patients who were provided or sought out additional therapeutic support had improved rates in long-term sobriety from their primary substance of abuse (GITA 2014, Schenberg 2014).

Whenever possible, the guidelines outlined here should be considered as an elementary step in the larger SUD treatment process.

A Note About Traditional Use

The particularly narrow focus of these guidelines also takes into consideration that the ibogaine-containing plants – particularly the *Tabernanthe iboga* and *Tabernanthe manii* shrubs – have been used for millennia by traditional communities in Gabon and the surrounding regions of the West African rainforest as a sacrament in rituals of healing and spiritual initiation. In this context as well, adverse events and fatalities have been

reported. In recent years these situations may be exacerbated by the fact that iboga is potentially threatened in its natural habitat, and other plants are sometimes used, whether mistakenly or intentionally, as substitutes, sometimes for economic advantage (GITA 2014). In other cases, these events have been attributed to unskilled practitioners.

It is hypothesized that aspects of the ritual and music employed by traditional communities are complexly arranged to both increase iboga's effects (Mass 2006) as well as physical and psychological wellbeing (GITA 2014).

Use of iboga in these traditional contexts is protected by the 2007 United Nations Declaration on the Rights of Indigenous Peoples, which states in Article 24 the right of indigenous people, "to their traditional medicines and to maintain their health practices, including the conservation of their vital medicinal plants, animals and mineral."

These guidelines can serve as a supplement to genuine spiritual practice, but in general this is beyond the scope of the considerations for risk management presented here.

Summary of Psychological Factors

For its psychological effects, ibogaine is best classified as an oneirogen, a substance that produces waking dreams (Naranjo 1974). For many ibogaine patients these effects are visual, and this visionary content is deeply subjective and personal, unfolding much the same way as dreams. The experience has also been likened to watching a film projected on an inner screen – often with a level of emotional detachment, even when the content is very emotional or graphic. This overall effect can evoke a state of profound contemplation and self-reflection.

It has been suggested that the experience is analogous to the kind of neurological and psychological integration that happens during a state of rapid eye movement (REM) sleep, but while the patient is fully conscious (Goutarel 1993).

These effects are distinct from classic psychedelic compounds, both subjectively and also because of the complex nature of the physiological effects that accompany them.

Onset, Phases & Duration of Effects

The onset of ibogaine's effects is generally noticeable within 1-3 hours after administration. This includes both a marked decrease in physiological withdrawal symptoms as well as the subjective effects, which are divided by Dr. Kenneth Alper into three distinct phases (Alper 2001). The duration of the effects mentioned below will vary based on dosage, the timeframe over which the doses are administered, and also factors that affect individual metabolism, including CYP450-2D6 phenotype.

1. Acute

This phase generally lasts between 4 to 8 hours, and include the most intense and visual

part of the experience that was described above as oneirogenic. During this phase the physiological effects, especially the mentioned ataxia, will be most pronounced.

2. Evaluative

The “evaluative” phase of the experience can last between 8 to 20 hours, and consists largely of a cognitive and more or less emotionally neutral review of material that was experienced in the acute phase. Patients generally prefer to be left undisturbed, and to lie mostly still and quietly during this integration phase.

3. Residual Stimulation

The final stage of effects generally last for another 24 to 72 hours. This period is usually accompanied by some level of exhaustion, and in some cases continued difficulty sleeping. Cognitive and introspective processes begin to relax and attention returns to the outer environment.

Management of Psychological Risk Factors

Conventional academic theories in the fields of psychology and psychiatry do not generally discuss the types of experiences that are commonly reported by ibogaine patients. When they do they are categorized broadly as forms of psychosis. However, transpersonal psychology and other similar experiential fields have established clear frameworks for understanding the nature and therapeutic value of these and other similar states. Rather than pathological, the natures of these states have been described by Stanislav Grof as holotropic, or “oriented towards wholeness” (Grof 1992). This field draws from millennia of human experience in accessing these states of consciousness for healing and rites of passage, and provides invaluable insights for integrating these types of experiences into contemporary therapeutic contexts.

It has been noted elsewhere that research using psychedelics contains rare but significant psychological risks, mostly involving “overwhelming distress” caused by a “bad trip” and resulting in the patient leaving the treatment site, or, less commonly, the manifestation of long-term psychosis (Johnson 2008). Unlike many conventional medical treatments, under the influence of ibogaine or other similar substances, it is difficult to overemphasize the importance of the treatment environment and the relationship that is formed between the patient and the caregivers, especially those who are present during administration and the acute period of integration following.

There are rare anecdotal reports of patients who experienced an acute confusional state after ingesting ibogaine that persisted from less than one hour up to several days. There are other very rare reports of symptoms of prolonged psychosis (Houenou 2011, Dyer 2011), as well as others of individuals who were diagnosed with mania following ingestion (Marta 2015).

However, to the extent ibogaine shares similar psychological risks to other psychedelic medicines, the psychological frameworks and therapeutic tools used when administering psychedelic medicines may also prove useful guides for psychological safety. Grof wrote

that, “LSD subjects whose sessions terminate in a state of incomplete rebirth show all the typical signs of mania,” but that, “when individuals experiencing this state can be convinced to turn inward, face the difficult emotions that remained unresolved, and complete the (re)birth process, the manic quality disappears from their mood and behavior.”

A comprehensive psychological assessment (Ch. 2) should precede any administration of ibogaine. Further, during and after treatment, sensitivity on behalf of the practitioners to the nature of the oneirogenic state and focused attention to the set and setting (Ch. 1) should be paramount at all times.

Working with Spiritual Emergence

One of the most important implications of transpersonal psychology is the understanding that many commonly diagnosed psychiatric conditions are forms of “spiritual emergency” or psychospiritual crisis that can lead to personal breakthroughs (“emergence”) if properly managed. Stanislav Grof lists addiction itself as a form of spiritual emergency, healing through which has the power to be incredibly transformative for an individual.

It is important to be able to differentiate between a genuine spiritual emergency that may possess cathartic healing potential, and forms of psychosis and genuine medical emergencies that require acute medical interventions.

Implementing this model of practice in the case of an acute confusional state can be time and energy intensive for therapy providers, but, when fully understood, the benefits for the patients are difficult to overlook. In all known cases, those who experienced an acute confusional state following ibogaine administration and were cared for throughout the entirety of this episode later reported that the process was personally important and ultimately beneficial.

The most important implication here is in the strategy in regards to the use of prescription medications. In the treatment of spiritual emergencies, clinicians should avoid use of psychiatric medications that may interrupt the psychological process underway. Anti-psychotic medications such as halperidol or chlorpromazine, which are used to interrupt the effects of other psychedelic substances have been known to cause prolonged negative reactions in patients who have taken ibogaine. When necessary, and in instances where insomnia threatens physical and psychological wellbeing, the judicious use of benzodiazepines can slow subjective effects and help to facilitate relaxation and sleep.

Treatment of spiritual emergencies is a field too vast to summarize in these guidelines, and strays from our intended focus. However, there is a sufficient amount of existing material to inform clinicians on the foundations of this work (Grof 1989, 1993, 2008).

Summary of Medical Factors

While ibogaine's mechanisms of action are not fully understood, some aspects of its complicated pharmacodynamics have been summarized in scientific literature (Alper 2001). Here we will review some of the relevant aspects of ibogaine's effects and neurotransmitter activity, as well as documented risk factors. These types of considerations are not common amongst psychedelic medicines, highlighting the unique physiological characteristics of ibogaine and the importance of its particular requirements for screening and monitoring.

Basic Pharmacodynamics

In addition to the psychological effects noted in the previous section, ibogaine presents some powerful physiological effects, including ataxia, tremor, nausea, vomiting, slowed breathing, heightened sensitivity to sensory stimuli, as well as bradycardia, hypotension and other changes to heart rhythms or blood pressure, including QT interval (time between the beginning of the Q-wave and the end of the T-wave on the cardiac cycle) prolongation, as well as T-wave morphology changes. As a result, unless necessary, patients generally prefer to be lying comfortably without a lot of agitation or movement.

Ibogaine's physiological effects, particularly its cardiac effects, can present significant and potentially life threatening risk factors even within the therapeutic dose range in patients that have certain pre-existing heart conditions, electrolyte imbalance, or who are detoxifying from alcohol or benzodiazepines (Litjens 2016, Alper 2012).

The QT interval prolongation associated with ibogaine may have several causes. The primary factor is changes to the way that cardiac cells utilize potassium to repolarize their electrical charge. This repolarization reserve is also affected by bradycardia and the blockage of the hERG channel, which modulates the bioavailability of potassium. As addressed later, this presents concerns in patients who are hypo- or hyperkalemic.

Other factors also play a role in depleting repolarization reserves and causing QT interval prolongation. These include low levels of magnesium; other QT prolonging medications, foods and supplements; withdrawal from cocaine, alcohol, or amphetamines; and many of the risk factors outlined in Chapter 2.

In many cases, during the acute period of ibogaine metabolism, bizarre T-wave morphology may be noted. These changes may include flattening of the T-wave, biphasic t-waves, and initial decrease in the anterior slope of the T-wave. These changes have been postulated to be attributed to changes in intercellular potassium exchange caused by blockage of the hERG channel (Thurner 2013, Alper 2015).

Ibogaine is metabolized via liver enzyme CYP450-2D6 into its primary metabolite, noribogaine. There may be clinically significant variability in the initial phases of ibogaine's effects based on a patient's CYP2D6 metabolism phenotype, particularly at the extreme ends of the spectrum for poor and ultra-rapid metabolizers (Glue 2015).

Ibogaine is also known to cause a level of restlessness and sleeplessness in the days following administration in some cases. This is especially the case for patients who use benzodiazepines or other sleep inducing medications.

Seizures under the influence of ibogaine can induce lethal arrhythmias, and have led to fatalities as well as permanent injury. Ibogaine itself has not been known to induce seizures, however, this is a concern in regards to withdrawals from alcohol or benzodiazepines, as well as for epileptics.

Clinical Pharmacokinetics

Ibogaine saturation peaks at 2 hours after administration and has a half-life of up to 7 hours in human plasma (Koenig 2015). It is metabolized into noribogaine, which, it is believed, is stored in fat tissue and released over the course of the following weeks or months. Noribogaine possesses some of the same effects as ibogaine, which may account for the prolonged reported benefits.

In those patients who experience T-wave changes they generally last between 12-14 hours, but can persist for as long as 24 hours. Cardiac concerns decrease after T-wave stabilization.

Some adverse events have been reported as late as 76 hours after administration (Alper 2012), however the factors in these instances generally can be addressed with proper screening and preparation.

Neurotransmitter Activity

In animal models, ibogaine has demonstrated action at various neurotransmitter sites, including the glutamate, opioid, dopamine, serotonin, and acetylcholine systems (Popik 1999). Its effects may be the result of a complex interaction between these systems, rather than the result of an effect on a single neurotransmitter system (Alper 2001).

The accumulation of these effects has been shown to mitigate withdrawal symptoms from opioids. Some suggest this to be up to 90% effective based on subjective reports. Ibogaine has also been shown to reduce the accumulated tolerance to opioids, and to increase the central nervous systems sensitivity to opioids (Parker 2001), which has implications in the case of relapse post-treatment. Patients need to be made acutely aware that their tolerance is reset to that of an opioid naïve person in order to avoid accidental overdose.

Ibogaine has also been shown to potentiate the effects of opioids and stimulants, with some gender variation in the case of cocaine. It is important for clinicians to be aware of these interactions in order to effectively facilitate detoxification. Especially in cases where long-acting opioids are considered, potentiation of opioid analgesia can result in simply delaying the onset of withdrawals until the effects of the ibogaine have subsided.

Methodology

Prior to this publication, the most significant precedent document to outline strategies for use of ibogaine in detoxification was the Manual for Ibogaine Therapy (Lotsof and Wachtel, 2006).

The current guidelines were drafted and edited by a panel of authors with extensive clinical and research experience. It was refined through a series of proposals and extensive interviews with medical professionals, practicing ibogaine therapy providers and researchers.

Proposals were presented during a series of public consultation periods, during which input was collected through in-person, electronic interviews, and written correspondence with members of the broader community of ibogaine therapy providers and medical professionals. The first completed version of this document is scheduled for publication in September 2015.

1. Context of Care

Informed Consent

The World Medical Association's Declaration of Helsinki – Ethical Principles for Medical Research Involving Human Subjects outlines specific requirements for informed consent when working with an experimental treatment. Whether or not patients explicitly demonstrate the presence of risk factors listed in these guidelines, they should be provided complete and accurate information regarding:

- The legal status of ibogaine in the region where the treatment is taking place,
- The experimental status of ibogaine-assisted detoxification in scientific literature,
- The risk factors involved in ibogaine treatment,
- The level of experience and qualifications of the caregiver that will be present during ibogaine administration,
- A realistic assessment of any possible benefits,
- Possible limitations of the particular method and protocol to be used,
- Information about alternative options that may be available,
- And, The Ibogaine Patient's Bill of Rights.

This information should be available to the patient at least two weeks prior to arrival, if possible, and should be reviewed and signed in person with a staff member prior to treatment.

Care Providers

The Johns Hopkins University research team's Human hallucinogen research: guidelines for safety suggests that the most important quality in a therapist, rather than medical or psychological credentials, is empathic skill (Johnston 2008). We carry forward the same recommendation, however even if the primary care provider is not medically credentialed, in order to effectively manage medical risks a minimum level of medical supervision should also be present.

The John's Hopkins team and others in the psychedelic therapy community widely recommend the presence of a male/female therapist dyad (Johnson 2008, Grof 2000). In the absence of this, we recommend, at minimum, a same gender therapist. This has therapeutic value, but also practical considerations. The ataxia brought about by ibogaine makes it difficult to walk, and a therapist may be needed to assist the patient in the bathroom.

Medical Supervision

Whether as a primary care provider, or in a supportive role to another therapy provider, medical supervision ensures preparedness in the rare case of cardiac emergency, even after thorough screening has been conducted.

It is highly recommended that all care providers have a minimum of a Basic Life Support (BLS) certification, and are well versed in the treatment of status epilepticus seizures and maintaining an airway.

In addition, Advanced Cardiac Life Support (ACLS) trained and equipped medical staff that are knowledgeable about the pharmacodynamics of ibogaine and appropriate emergency responses should be available on hand throughout the treatment episode. Preference is given to medical staff certified through an ACLS for Ibogaine Therapy course offered by GITA.

The equipment necessary to minimize the risks as outlined in these guidelines is listed in Appendix B.

In cases where medical professionals are the primary care providers, we strongly recommend formal training in working with patients who are undergoing altered states.¹

Emergency Preparedness

Unless the treatment is conducted in a hospital setting, the treatment location should take place within a 15-minute range for fully equipped emergency responders, and ideally no further than 30 minutes from a fully equipped 24-hour hospital.

A thorough emergency plan should be in place, and staff should be well versed in their role in carrying it through.

Set & Setting

Since early days of psychedelic research attention has been drawn to the factors of set & setting (Leary 1964). It is crucial that the therapeutic team understands and displays an acute sensitivity to the development of therapeutic relationships and a therapeutic space.

Set

This term refers primarily to the internal preparations, the state of mind and the intentions of the patient. It can be helpful to review expectations, concerns and hopes prior to the session, as well as to review the general psychological and physiological terrain that might emerge during the experience so that the patient feels a sense of openness and preparedness for what they are about to undergo. Developing a therapeutic relationship can be integral to supporting the patient in this process.

Setting

This refers to considerations in the immediate environment, and can be extended to include such things as decoration, incense, and music selection. It is important to ensure that the space is as comfortable and clean as possible, with a bed, easy access to a bathroom, and that it will be free of interruption for the duration of the treatment episode.

With other psychedelic medications, psychotherapy is often conducted in direct conjunction with administration. However, with ibogaine it is strongly advised that during the acute phase of the experience conversations be limited only to necessities, such as checking in quietly, or coordinating physical movements or medical assistance. It may be appropriate to offer minimal suggestions if prompted, but otherwise, therapeutic conversations should be limited to pre- and post administration, and patients should be given as much space as possible to benefit from the internal process.

Patients become extremely sensitive to light and sound during the course of treatment. The external space should be quiet, and if necessary aids such as earplugs or music through earphones should be available. It should also be easy to darken the space. Comfortable eye masks (such as Mindfold Relaxation Masks) can be effective at blocking all or most of the external light. Ideally, lighting in the room should be ambient and non-intrusive, such as candles or soft lamps.

There are various schools of thought regarding the timing of sessions. Some therapy providers have preferred late afternoon and early evening because there are naturally fewer distractions. However, it is generally easier to access medical support or emergency services during daytime hours. Risk reduction should be a primary consideration.

Music

In psychedelic therapy, music has widely been regarded as beneficial both therapeutically, as well as in reducing psychological risks, by helping the internalization of the process (Grof 2000). Researchers from the Maryland Psychiatric Research Center observed that, “Music appears to be involved significantly in the crucial extra-drug variables of both set and setting” (Bonny 1972).

In general, considerations about the music in psychedelic sessions are also relevant for ibogaine therapy. It is strongly advised that appropriate high fidelity music is available through comfortable headphones.

It is highly recommended that the music selection playing during the peak effects is non-verbal, or that it be in a language not understood by the patient. Appropriate music can be found throughout cultures in which there were efforts to use music meditatively, or in order to access transcendental spaces. Examples include Indian ragas, Sufi chants, Mongolian throat singing, etc. There are also extensive libraries of music by traditional pygmy and Bwiti villages that can be very useful to incorporate. The polyphonic singing of some pygmy communities is one of the most advanced known examples of natural overtones. However, the patient’s music preference, and their response to what is played should be a main concern.

Adjunct Therapies

Ibogaine can be a powerful tool in detoxification and treatment of substance abuse disorders. Both ibogaine (He 2006) and noribogaine (Carnicella 2010) have the capacity to increase the expression of glial cell-derived neurotrophic factor (GDNF), a protein that stimulates the growth of new dopamine neurons. It has been shown to produce a valuable state of neuroplasticity that can improve ability to learn new information (Popik 1996), and potentially to ingrain new habits and patterns of thinking.

Neuroplasticity can also be an opportunity to further ingrain negative patterns if positive ones are not enforced. There are practices that in conjunction with ibogaine can help to ensure that the lifestyle changes it initiates are positive and health affirming. This is a non-comprehensive list of adjunct therapies that many have incorporated into their treatments with positive results, including:

Psychotherapy

Various psychotherapeutic techniques provide valuable guidance after ibogaine therapy. This can include CBT, which has been shown to have strong efficacy in the treatment of addiction (Hofmann 2012). Others such as Holotropic Breathwork, Gestalt therapy, and others have been shown to be valuable tools when applied in conjunction with psychedelic therapy.

Nutrition

Nutrition not only promotes overall health, but can also be a source of important precursors for neurotransmitters that can be very helpful in the recovery process. Diets are often extremely deficient when people are struggling with SUDs and not tending to their basic needs. The period following ibogaine detox can be an important time to re-learn an appreciation for proper nourishment.

Bodywork

Bodywork and physical therapy not only can provide relaxation, as well as relief for people who suffer from discomfort or chronic pain, but it can help people to connect with their body in a new way, which can be grounding and affirming.

Exercise

Exercise has been shown to increase beta-endorphin levels, which has a positive effect on many underlying conditions that are common amongst those with SUDs.

Meditation

Simple mindfulness practices have been shown to help balance brain hemispheres, and generally promote clarity, focus and relaxation. It is not necessary to encourage any elaborate practice, but the foundations of simple meditation techniques can be helpful both during and after the ibogaine experience.

Neurofeedback & Brain Stimulation Devices

Some brain stimulation devices have been FDA approved to treat a variety of conditions, including anxiety, insomnia, depression and chronic pain.

Caution Regarding Detoxifying Therapies

Detoxifying therapies can include colonics, enemas, ozone therapy, castor oil, etc. These methods have all been included for various reasons, some in order to deal with constipation or compaction for opioid users, which constitute a risk factor during treatment.

It should be noted that any therapy that has the potential to deplete electrolytes should be avoided. However, after any major procedure that might affect electrolytes, blood tests and liver panels should be re-taken.

Furthermore, therapy that might release toxins into the body should be avoided in close proximity to ibogaine therapy as this might have an overall effect on energy metabolism.

Record Keeping

Working with an experimental medication requires rigorous record keeping for many reasons. Most obviously, detailed records can provide useful statistical information on how successes were achieved.

In relation to risk management, however, it is much easier to learn and to share information about well documented cases in which treatment goals were not achieved or the treatment episode resulted in an adverse event. Without records, important details can be easily missed and these mistakes are liable to be repeated.

It is highly recommended that extra care be taken in documenting every aspect of the treatment process described in the rest of this document.

2. Patient Criteria & Considerations

Inclusion Criteria

The therapeutic relationship begins during the initial contact with the patient. Whether in person, by telephone, or other means of correspondence, it provides an important opportunity to begin to build a foundation of trust, as well as to assess the patient's eligibility for ibogaine therapy.

The most basic considerations for inclusion in therapy are establishing a significant degree of consent. Ibogaine has been generally observed to be ineffective in patients who are not personally committed to therapeutic process. That excludes all forms of intervention-based treatment, state-incentivized treatment models, or any other way that patients may be coerced to participate in the therapeutic process.

The basic considerations for inclusion are as follows:

- Has understood the basic scope of the treatment and what it involves.
- Has a strong desire to take ibogaine and realistic expectations about possible outcomes.
- Is willing to be a part of a process that includes adhering to provider and clinicians' requests to attend appointments, submit a comprehensive application, and complete other preparation protocols thoroughly and honestly.
- Takes the responsibility of engaging with other services of the options that have been referred to, and continues with ongoing self-care.
- Accepts the Responsibilities in the Ibogaine Patient's Bill of Rights.

Application

Careful screening is the most important factor in minimizing risks associated with ibogaine-assisted detoxification. The considerations below reflect the known psychological and biological risk factors, and should be taken into careful consideration for every patient prior to intake.

Sufficient personal information and medical history, particularly that regarding psychological diagnoses and drug or medication use, prior to intake is crucial in making an informed assessment. Interpretation of this information for screening is discussed throughout the rest of this chapter.

Personal Information & Medical Overview

Basic personal information should include: name, personal contact information, emergency contact person information, height, weight, assigned and preferred gender identity, a complete history of accidents, hospitalizations, and surgical procedures, as well as a complete history of previous illnesses, heart conditions, and other diagnoses,

including psychiatric diagnoses.
Drug Use Profile

Clinicians should review a complete history and current use of all medications, prescriptions, over-the-counter drugs, street drugs, vitamins, supplements and herbs. This should include dosages and usage patterns. Information about the nature of use including overdoses, at-risk behavior, and any previous experience with psychotropics should also be taken into consideration.

Medical Tests

The following medical tests should be considered in relation to the medical history:

- Resting 12-lead electrocardiogram
 - Taken while not wearing metal objects, while not under the influence of stimulants (including coffee), and while not in withdrawal, etc.
- Thallium (nuclear) stress testing, echocardiogram or 24 Holter monitor
 - In the case of arrhythmias, extrasystoles, ST segment changes indicating myocardial ischemia/infarction, significant cardiac history, or cardiac risk factors.
- Complete Metabolic Panel (Chem 12 or Chem 18)
 - Including creatinine and electrolytes, sodium, potassium, serum magnesium, and liver functions.
- Complete Blood Count (CBC)
- Complete Thyroid Function Test
 - In the case of a history of thyroid disease or currently prescribed thyroid medication, a thyroid-stimulating hormone (TSH) test is not sufficient.
- Complete Physical
 - Conducted when necessary to consider the context of certain pre-existing heart conditions.

Cytochrome P450 2D6 Phenotype

Some preclinical data suggests that dosing regimens should be altered according to CYP2D6 phenotype, especially for Poor Metabolizers and Rapid Metabolizers, the extreme outliers on the spectrum (Glue 2015). This is later addressed in the discussion about dosing (Ch. 14).

Absolute Exclusion Criteria

In the presence of any of the following criteria patients should be excluded from ibogaine-assisted detox due to the gravity of the associated risks. The nature of some of these risks have been previously discussed; others are explained here for clarity. Where possible, these issues should be addressed, and then reassessed, prior to intake.

Certain psychiatric conditions

- Schizophrenia
- Bipolar disorder for which patient has been hospitalized or medicated
- Depersonalization and/or Derealization Disorder
- Cerebellar dysfunction
- Epilepsy
- Rule out the possibility that benzodiazepine or alcohol withdrawal related seizures have been misdiagnosed as epilepsy.
- Psychosis or acute confusional state
- Organic brain disease
- Dementia

Certain pre-existing heart conditions

- Prolonged QTc Interval
 - The FDA considers prolonged QTc to be: > 450 milliseconds for males and > 470 milliseconds for females
- History of heart failure, enlarged or hypertrophic heart
- Active blood clots
 - Pulmonary embolism
 - Deep vein thrombosis

Certain major respiratory conditions

- Low oxygen levels and required steroid treatments lead to excessive risks.
- Emphysema
- Chronic Obstructive Pulmonary Disorder
- Cystic Fibrosis

Severe or chronic gastrointestinal issues

- Bleeding ulcer
- Leaky gut syndrome

Other criteria

- Abnormal blood test results:
- If potassium or magnesium are outside normal ranges, they should be corrected.
- Impaired Kidney or Liver function
- Any patient with liver enzymes greater than 2.5 times normal levels, on dialysis for kidney failure, or with abnormal Blood Urea Nitrogen (BUN) or creatinine levels may not be able to metabolize ibogaine properly, and might experience toxicity.
- Active infection or abscess
- Within 6 months of major surgeries. Get physician approval.
- Pregnancy

Cardiac Risk Factors to Consider

In the presence of any of the following conditions, a risk benefit analysis should be conducted by a cardiologist that is knowledgeable about ibogaine's pharmacodynamics if patients are to be considered.

Certain pre-existing heart conditions

- Borderline QTc interval
- The FDA considers borderline QTc to be: 430-450 milliseconds for males and 450-470 milliseconds for females.
- Ibogaine also extends the QT interval. This should be carefully monitored to make sure that it does not become prolonged. Monitor electrolytes.
- Irregular heart rhythms (Arrhythmias)
- Atrioventricular heart blocks
- Any history of ventricular arrhythmias
- Atrial Fibrillation (clot risk)
- Childhood congenital heart defects
- Heart attack (Myocardial Infarction, Coronary Vasospasm)
- Murmur
- Use echocardiogram to rule out significant Valvular Heart Disease (Valve Stenosis, Regurgitation, Prolapse).
- History of Pericarditis/Endocarditis
- Call for an echocardiogram.
- Family history of heart attack/sudden cardiac death before 50 years of age.
- Major heart/vascular/pulmonary surgery i.e. transplant, Coronary Artery Bypass Grafting, artificial heart valves, surgeries for Coronary Heart Disease, other surgeries.
- Internal or External Pacemaker
- History of blood clots
- Stroke or transient ischemic attack should only be accepted with sufficient time since event and pre-clearance.
- Pulmonary embolism or deep vein thrombosis should only be considered if these previous issues have been sufficiently resolved.
- Abnormal Heart Rate/Rhythm
- Resting heart rate of ≥ 120 beats per minute or higher; or ≤ 50 beats per minute.
- High/Low blood pressure
- 170/105 or higher or 90/60 or lower, while not taking blood pressure medication.

Other cardiac disease risk factors

Including: Hypertension, hypotension, diabetes, nicotine use, high cholesterol, peripheral vascular disease, chest pain/shortness of breath with or without exertion, frequent indigestion, and unexplained fainting.

Other Risk Factors to Consider

The rest of this list includes non-cardiac-related risk factors that should be considered during the application phase by a knowledgeable physician. In some cases preparation protocols can help to resolve these issues prior to treatment, and success with these protocols should be considered prior to acceptance.

Certain psychiatric conditions

- Without proper therapeutic support, ibogaine may exacerbate or re-traumatize patients with some conditions.
 - Bipolar disorder
 - Post traumatic stress disorder (including sexual or violent trauma)
 - Borderline personality disorder
 - Beck depression inventory score ≥ 24
 - Suicide attempts, ideations and/or intents

No accessible veins for IV port access

In this situation a central line should be inserted prior to treatment. Although other options exist for emergency meds, a central line is the only way to provide fluids, which are primary to several emergency interventions (Ch. 14).

Irregular Thyroid

Hypothyroid leads to increased bradycardia, while hyperthyroid leads to increased risk of tachycardia. Make sure that thyroid medication has stabilized thyroid function.

Major respiratory conditions

- Sleep Apnea
 - Sleep Apnea makes it difficult for people to breath when they are in a sleep state. Saturation or flood doses of ibogaine put people into a sleep-like state, and apneic breathing can cause oxygen saturation to fall dangerously low, which in extreme cases could put the patient at risk for life-threatening hypoxia.
 - Patient should use their prescribed respiratory device during ibogaine administration, and oxygen levels should be monitored closely.
- Asthma, COPD (Chronic Obstructive Pulmonary Disease), emphysema, history of smoking more than 30 packs of cigarettes per day for 30 years
 - Check for severity of the condition. If the patient has been hospitalized or has had multiple severe attacks in the last 2 years, they should be considered a poor candidate.
 - Check for preparation requirements with prescribed steroid inhalers (Ch. 8).

- Review Asthma Attack Intervention (Ch. 15).
- Pulmonary Fibrosis
 - Typically patients will be oxygen dependent. Should be considered a poor candidate.
 - Check for preparation requirements with prescribed steroid treatment (Ch. 8).
- Sarcoidosis

Metabolism, Diet, and Gastrointestinal Issues

- CMC or Metabolic Panel results outside of normal ranges
 - Preferred ranges for potassium (4.5-5.5 mEq/L) and magnesium (1.5-2.5 mEq/L) are ideal.
- Constipation or Impaction
 - Movement of the diaphragm can be compromised, leading to lower blood-oxygen saturation, and subsequently hypoxia and left ventricular dysfunction. Hypoxia may precipitate angina or tachycardia. Observe for dizziness and hypotension.
 - Can impede metabolism of medications.
- Obesity
 - In addition to significant concerns about energy metabolism, ibogaine is stored in fat tissue. Obesity will greatly affect metabolism of ibogaine.
 - At a BMI of 35+, risk of blood clot and stroke are dangerously high. These patients can be put on mini-dose heparin or enoxaparin sodium – blood thinners that don't change coagulation factors – during treatment.
- Eating disorders
 - It can be extremely dangerous to treat with anorexia or bulimia. These patients must be stabilized with electrolytes prior to treatment, and must remain under constant observation to make sure they are not purging or bingeing prior to administration.
- Malnutrition
 - Noribogaine is stored in fat tissue and released back into the blood plasma, and by some accounts this may have an effect on lasting benefits.
- Crohn's disease, Irritable bowel syndrome, diverticulosis, diverticulitis
 - Patient should be cleared by a gastroenterologist if the condition is stable. Discuss considerations with medication.
- Pancreatitis
- Other considerations
- Chronic infectious diseases (i.e. Tuberculosis, Hepatitis B or C, HIV)
- Overdose history
- History of head trauma with loss of consciousness for a significant amount of time
- Age 60+
 - Recommended to obtain a stress ECG test.
- Family history of certain psychiatric conditions, which may have a risk of expressing, primarily in patients under 30 years of age

- Bipolar disorder
- Schizophrenia
- Depersonalization and/or Derealization disorder
- Cerebellar dysfunction
- Epilepsy
- Psychosis
- Organic brain disease
- Dementia

Drug Interactions to Consider

Common prescription drugs and drugs of abuse which pose major complications, or for which treatment is frequently sought, are listed in the following chapters:

- Chapter 3: Opioids
- Chapter 4: Benzodiazepines
- Chapter 5: Alcohol
- Chapter 6: Stimulants
- Chapter 7: Antidepressants
- Chapter 8: Steroids
- Chapter 9: Other Medications

Here are listed broad classifications of other medications and their considerations:

QT prolonging medications, foods and supplements

Ibogaine causes bradycardia, hypotension, and prolongation of the QT interval. Each of these symptoms may be in conjunction with or independent of the others. Other QT prolonging drugs exacerbate these effects, which can put a patient at greater risk for lethal cardiac arrhythmias. All other medications that prolong the QT interval should be assessed.¹

Diuretics

Can lower potassium and sodium levels. It is important that serum electrolyte levels are checked after discontinuing diuretics.

CYP2D6 Metabolism Interactions

This enzyme is the channel by which ibogaine is metabolized in the liver. Other drugs in the body that are metabolized by this enzyme² could interfere with a patient's ability to efficiently metabolize Ibogaine.

Centrally Acting Drugs

All centrally acting drugs should be avoided or managed with extreme vigilance. Interactions with ibogaine are not well understood. This classification includes blood pressure medications, benzodiazepines, non-benzodiazepine hypnotics (i.e. Z class drugs), barbiturates, muscle relaxants, antipsychotics, anticonvulsants and general anesthetics.

Serotonin Increasing-Medications

Examples of medications that increase the level of serotonin in the body include: SSRIs, SNRIs, NRIs, MAOIs, buspirone (anxiety tx), trazodone (depression & insomnia tx), certain migraine medications, certain pain medications (fentanyl, meperidine, pentazocine and tramadol), dextromethorphan (cough suppressant), certain anti-nausea medications (granisetron, metoclopramide, ondansetron), cocaine, and some dietary supplements like St. John's Wort.

Ibogaine also causes an increase in serotonin, and extreme levels can cause serotonin syndrome with symptoms of acute confusional state (see Ch 7 for Antidepressants).

Calcium Channel Blockers or Beta Blockers

Calcium Channel Blockers are prescribed for high blood pressure and for arrhythmias. Beta Blockers (see Ch. 9) are primarily prescribed for high blood pressure and tachycardia. Carefully consider these underlying conditions, and whether the medications are necessary. These medications can lower blood pressure and heart rate dramatically, and alter electrical conduction through the heart.

Anti-Arrhythmic Medications

Patients with arrhythmias that require medication should be considered poor candidates.

Birth Control

Some forms of female birth control are slightly higher risk for blood clots. This should be noted wherever other clot risks are present.

A Note About the Calculation of Half-Lives

Throughout the rest of this document, when calculating the period of time to recommend that people stop taking certain medications prior to treatment, calculate from at least four to seven times the listed half-life of the medication. This is variable depending on the drug and how critical it is. In some cases more specific considerations will be noted.

3. Opioids

Background Information

Abuse of opioids such as heroin, morphine, and prescription pain medications has been widely recognized as a large-scale global health problem affecting all levels of society. The UN Office on Drugs and Crime World Drug Report 2015 estimates that 48.9 million people worldwide use opioids or opiates.

The most effective existing treatments for opioid use disorders are medication assisted treatments such as methadone and buprenorphine. These substances have been shown to help dramatically to reduce the severity of SUDs and to help people to significantly reclaim their mental and physical health, as well as livelihoods and family relationships. However, for those who wish to stop opioid maintenance treatments, there are few options, and withdrawal symptoms are at least as difficult and greatly prolonged compared with shorter-acting street drugs or prescriptions.

Many addiction professionals do not see a need to stop a presumably successful treatment protocol, but according to the World Health Organization, patients who are receiving treatment for substance use disorders should be helped to withdraw from opiates if it is their informed choice to do so (WHO 2009). The increasing number of patients of ibogaine therapy who have sought treatment in order to withdraw from methadone or buprenorphine is a demonstration of the difficulty that patients have in withdrawing from these medications when they wish to terminate the therapy.

Of all of ibogaine's therapeutic benefits, one of the most impressive is in the treatment of opioid use disorders. For these patients, and for others who are properly informed and still choose not to undergo maintenance therapy, ibogaine provides a valuable treatment option.

General Considerations

In addition to ibogaine's ability to mitigate withdrawal symptoms, it also has the potential to potentiate the analgesia of opioids if they are co-administered. The accurate calculation of half-lives and of timing in dosage in order to avoid the risk of accidental overdose, or the prolongation of residual withdrawals is critical in ensuring that the treatment is both safe and effective.

This is especially a concern for long-acting opioids, which remain in the system long after the initial onset of withdrawal symptoms.

Also, in order to prevent accidental overdose, it is very important that as part of the regular process of informed consent clients are made aware that if they use opioids during treatment they are at immediate risk for overdose. Further, it is important to reiterate at discharge that if the patient relapses and uses opioids after their treatment, they must take

the same dose as an opioids naïve user, due to the fact that whatever tolerance they accumulated during the course of their previous use, has been reset.

Preparation

Patients who are using opioids by any other means of administration (smoked, insufflated, IV, IM) should be advised to switch to oral opioids under medical supervision prior to arrival. Oral morphine is preferred.

Long-Acting Opioids

If ibogaine is administered while long-acting opioids are still present in the blood there is an aforementioned risk of analgesic potentiation. However, after the ibogaine treatment, as this analgesia subsides, patients may experience residual withdrawals and PAWS. To avoid risk and ensure that detoxification can be completed successfully, patients who are attempting to withdraw from long-acting opioids should switch to shorter acting opioids, such as morphine sulfate, prior to treatment.

It is important to accurately calculate the elimination half-lives and conduct an effective switch-over. There is some controversy about the best way to calculate this timeline, but a general guide is to ensure that there is a residual dose of no more than .125mg of buprenorphine, or 2mg of methadone, prior to treating with ibogaine.

Some clinicians believe that the half-lives for both of these medications be calculated at 24 hours. In this case, for example, a patient that is taking 4mg of oral buprenorphine would have a residual dose of 2mg on day 2, 1mg on day 3, .5mg on day 4, .25mg on day 5, and would be cleared for treatment by day 6.

Likewise in the case of methadone, a patient taking 100mg per day, would have a residual dose of 50mg on day 2, 25mg on day 3, 12.5mg on day 4, 6.25mg on day 5, 3.125mg on day 6, and would be cleared for treatment on day 7.

Other clinicians prefer to ensure that the elimination is calculating using a longer half-life: up to 72 hours for buprenorphine, and up to 48 hours for methadone. These considerations may be especially useful in certain situations, such as for poor metabolizers, those who have been taking these medications for many years, or simply to avoid some of the complications when the switchover cannot be completed under close supervision.

Using these calculations, a patient with a 4mg daily dose of buprenorphine, would be recommended to switch for at least 18 days. A patient taking 100mg per day of methadone would be recommended to switch for at least 14 days.

It is very important that regardless of the half-life calculations done in advance, the switch-over be conducted carefully and under medical supervision. Some of the reported

instances of residual withdrawal may result because of improper calculation of the necessary dosages of short-acting opioids.

It is important to calculate the appropriate conversion dose, and to build up of this dose gradually as the long-acting opioid are eliminated. For example, a patient on 4mg per day of buprenorphine may require 100mg of oxycodone in order to stabilize. However, if the patient stops taking buprenorphine, by the end of the first half-life cycle they have a 2mg residual dose. If the patient is given 100mg of oxycodone right away, they will be over-sedated, which will result in discomfort during the switch-over process and an increased opioid tolerance prior to treatment. The switch-over must be conducted gradually, and in order to be done effectively, should be done under medical supervision and with dosages just sufficient to treat observed withdrawals. Application of the Subjective and Objective Opioid Withdrawal Scales (SOWS & OOWS) is standard.

The challenge with long switch-over times is that in many places it is very difficult to obtain the necessary short-acting medications due to legal restrictions. Under no circumstances is it preferable for a patient to switch to short-acting illicit drugs. During long unsupervised switch-over periods there is an increased risk that patients will engage in illicit activity, and that increased tolerance will develop.

As an additional safety concern, patients should not be instructed to taper their use of any other medications (including benzodiazepines) during the switchover process. Benzodiazepine use, for example, should be stabilized prior to the opioid switch. Under no circumstances should it be recommended that benzodiazepines or other potential drugs of abuse be used to counteract discomfort or withdrawal during the switch-over process.

These recommendations provide a guide for preparing a patient prior to intake, however, testing for presence in the urine can verify that the drug has been sufficiently eliminated, and it is possible that in some cases treatment may be started sooner. Note that buprenorphine and some synthetic opioids will not appear in the standard ELISA lab tests (see Ch. 11).

Other Considerations

Some clinicians have attempted to treat patients who are withdrawing off of long-acting opioids after less time by doing initial treatment 24 hours after the last dose and keeping the patient under supervision for 7 to 10 days after, using booster doses to treat residual withdrawal symptoms.

This method is not recommended for reasons previously mentioned, but especially in the case of methadone. Large doses of methadone have been shown to prolong the QT interval (Kornick 2003) by hERG channel inhibition, and there is some evidence that suggests lower doses also have this effect. In rare cases, QT prolongation from methadone alone has led to torsades de pointes (Vieweg 2013).

Intake

Ibogaine has been shown to decrease accumulated tolerance to opioids, and to increase the central nervous systems sensitivity to opioids (Parker 2001), increasing their effects when co-administered. This must be taken into consideration throughout, as well as after the treatment, when opioids are administered.

Short acting opioid, preferably morphine sulfate, should be used to stabilize the patient as long as necessary prior to ibogaine administration, or in the case that it is not possible to administer ibogaine for medical reasons. Stabilization dosage should be sufficient to manage withdrawal symptoms as identified on the SOWS and OOWS.

Extreme vigilance should be taken to ensure that the opioids dosage administered is not higher than the dose necessary to manage withdrawal symptoms.

4. Benzodiazepines

General Considerations

Benzodiazepines are a class of drugs that enhance the effects of the neurotransmitter GABA, producing sedative, hypnotic, anxiolytic (anti-anxiety), anticonvulsant, and muscle relaxing effects. These drugs have been a factor in several fatalities temporally associated with ibogaine administration due to the onset of seizures related to withdrawals (Alper 2012).

Ibogaine has been shown to have no effect on the GABAergic system (Popik 1998), and does not reduce withdrawal symptoms from benzodiazepines. In fact, hERG channel blockage, or syncope episodes caused by QT prolongation can increase the likelihood of the onset of seizures in general.

Benzodiazepines may dull some of ibogaine's psychoactive and psychologically therapeutic effects. This should be considered, especially in the case of non-benzodiazepine dependent patients. However, no other negative interactions are noted, and use should be continued throughout treatment to avoid withdrawals. This effect can be a hindrance to the therapeutic process, but risks and benefits should be weighed accordingly. This effect can be beneficial in cases of overwhelming effects or prolonged insomnia.

If a patient is tolerant of a very high dose of benzodiazepines, or if questions arise about the accuracy of a patient's reported dose (some users have been known to develop amnesia about the quantity and frequency of their dosage), this should be considered a high risk factor and the patient should be considered a poor candidate unless their use can be stabilized.

Preparation

Under no circumstances should benzodiazepine dependent patients be directed to stop benzodiazepine use before or during treatment. It is suggested that, if necessary, a long-term medically supervised taper be completed prior to intake, or conducted after discharge.

During Treatment

If patients are taking short-acting benzodiazepines, they should be switched onto a longer-acting benzodiazepine, such as oral diazepam or clonazepam, prior to arrival in order to avoid peaks and valleys of anxiety and withdrawal symptoms during treatment.

The Ashton Manual, and other sources, provide a clinical protocols for facilitating benzodiazepine detoxification, as well as for calculating half-lives and equivalent doses of different benzodiazepines.

It is usually possible to observe withdrawal symptoms from short acting benzodiazepines within 24-72 hours. As mentioned, if there are questions about the accuracy of a benzodiazepine dependent patients dosage, an observation period of 72 hours should be observed. In the case of longer-acting benzodiazepines, withdrawal symptoms may not be visible for up to 3 weeks. Patients should be considered a candidate only if the dose is justifiably stable.

In cases where patient is not alcohol or benzo-dependent, or benzo-naïve, use short acting benzodiazepines such as alprazolam.

Also it is important to note that if administering benzodiazepines as a sleep aid to benzo-naïve patients, note that a very small percentage may experience a mild waking dream state.

5. Alcohol

General Considerations

Alcohol withdrawal can be a serious and life-threatening process that can occur in patients after weeks, months or years of regular drinking. Like benzodiazepine withdrawal, alcohol withdrawal has also been a factor in several fatalities temporally associated with ibogaine administration due to the adverse health effects of withdrawal such as cardiomyopathy, delirium tremens and seizures related to withdrawals (Alper 2012).

Acute alcohol withdrawals are usually between 24 to 120 hours after the last drink. The peak seizure risk is generally between 48 to 72 hours.

Preparation

All patients should refrain from drinking alcohol for at least 3 days before ibogaine administration, and for at least a week afterwards.

Patients who are at risk for alcohol withdrawal syndrome need to be completely detoxed from alcohol dependency under medical supervision for 5 to 7 days prior to treatment.

Intake

If during the intake process or later it is suspected that a patient has been drinking, or is a regular drinking, detox and stabilization should be completed under medical supervision. Stabilization should continue for at least 5 to seven days prior to the administration of ibogaine, or until cleared of seizure risk by attending physician.

Care should be taken to ensure there are no residual alcohol-withdrawal related symptoms including QT prolongation, arrhythmia, hypertension, delirium tremens, abnormal psychological findings, confusion, etc.

6. Stimulants

General Considerations

Stimulants are a broad classification of psychoactive substances that induce temporary enhancements in cognitive or physical functions. However, use can present significant cardiac effects that can lead to “varying degrees of tachycardia, vasoconstriction, unpredictable blood pressure effects, and arrhythmias, depending on the dose taken and the presence or absence of coexisting cardiovascular disease” (Ghuran 2000).

In lower doses ibogaine itself acts as a stimulant, and if taken in conjunction with various stimulants, ibogaine can amplify their effects.

In addition to these general concerns, many specific stimulants present their own unique risks, which are further discussed here along with recommendations for how to minimize these risks prior to treatment. If there are doubts about compliance during intake or at any point during the treatment, a urine screen and any relevant cardiac tests should be re-administered.

Even after the terminating use for the periods described below, patients who have been using stimulants chronically may have higher instance of arrhythmias due to hyper excitable heart. Particular attention should be paid to the cardiac status throughout the treatment process.

Crack & Cocaine

In addition to the general stimulant considerations, crack and cocaine are known to block the hERG channel and lead to QT prolongation (Karle 2002).

Ibogaine has been shown to potentiate the effects of cocaine by sensitizing receptors that mediate its effects (Szumlinski 2000).

It is recommended that patients cease consumption of crack and cocaine for at least 7 days prior to administration of ibogaine.

Methamphetamine

Methamphetamine is known to prolong the QT interval even though it does not interact with the hERG channel or any other known cardiac channel (Haning 2007), as well as to affect CYP2D6 metabolism. Ibogaine has been shown to potentiate the effects of methamphetamine by sensitizing receptors that mediate its effects (Szumlinski 2000).

It is recommended that patients cease consumption of methamphetamine for at least five days prior to administration of ibogaine.

Prescription Stimulants

It is recommended that patients cease consumption of prescription stimulants, including methylphenidate (Ritalin) and amphetamine/dextroamphetamine (Adderall), for at least five days prior to administration of ibogaine.

Caffeine

Caffeine inhibits the metabolism of melatonin (Härtter 2003), which may alter the circadian rhythm. Caffeine has also been shown to induce hypokalemia (low potassium) (Tajima 2010), and may contribute to QT prolongation.

It is recommended that patients cease consumption of caffeine for at least 5 days prior to administration of ibogaine.

7. Antidepressants

General Considerations

The World Health Organization states that, “Depression is the leading cause of disability worldwide, and is a major contributor to the global burden of disease.” It estimates that as many as 350 million people worldwide suffer from depression (WHO 2012), and prescription of anti-depressants continues to rise dramatically, especially in developed countries (OECD 2013).

There are several different types of medications commonly prescribed to treat depression, most of which operate by increasing the level of serotonin. These medications may present a risk for serotonin syndrome if consumed in close proximity to ibogaine.

SSRI/SNRI Medications

Selective serotonin re-uptake inhibitors (SSRIs) and serotonin and norepinephrine re-uptake inhibitors (SNRIs) both increase the level of extracellular serotonin by inhibiting its uptake into the presynaptic cell. Ibogaine is also known to increase the level of serotonin, which when taken together may lead to an increased risk of serotonin syndrome, although the extent of this risk is not clear.

Additionally, cytochrome CYP2D6 plays an important role in the metabolism of ibogaine, SSRIs and SNRIs, which may slow metabolism through that enzyme and present significant risks for ibogaine administration as it does for other medications.

Presently, due to the significant half-lives and extended withdrawal symptoms, it is very difficult for people to stop taking most of these medications or to taper their dose without significant discomfort. As a result, many clinicians treat patients only 2 to 3 days after terminating their use, however this cannot be said to be an ideal treatment course in order to minimize risks.

Ideally, these medications would be completely tapered under the careful observation of a physician or psychiatrist. A psychiatric reassessment should be re-done after the taper.

MAOI Medications

Monoamine oxidase inhibitors (MAOIs) affect the metabolism of many drugs, and for this reason are usually only prescribed when SSRIs or SNRIs are not an option. However, MAOIs are sometimes prescribed as a first-line treatment for Parkinson’s disease.

Further, MAOIs interact significantly with other psychoactive drugs, including some hallucinogens, amplifying their effects. It is not known whether or to what extent MAOIs interact with ibogaine in this way.

All MAOI medications should be stopped 7 to 10 days prior to treatment.

8. Steroids

Corticosteroids

Corticosteroids, including cortisone, hydrocortisone and prednisone, are drugs that closely resemble the cortisol produced by the adrenal gland, and are commonly used in the treatment of asthma and other respiratory conditions, as well as arthritis and autoimmune disorders.

These drugs are known to prolong the QT interval (Busjahn 2004), and present increased an increased risk for arrhythmia.

It is recommended to calculate the half-life of steroid medication and for patients to cease consumption with sufficient time for the drug to be eliminated prior to treatment.

If corticosteroids must be used as an intervention (Ch. 15) during treatment, do so with caution.

Anabolic Steroids

Androgenic or Anabolic steroids closely represent testosterone and are available as prescription hormone treatments, however they are also often abused by body builders in order to facilitate rapid development of muscle tissue.

Patients who are taking anabolic steroids orally are prone to liver malfunction. Pay special attention to liver function tests.

For all anabolic steroids, get a prostate-specific antigen (PSA) test in addition to metabolism tests. Also, pay close attention to hemoglobin and hematocrit. A high blood cell count means patient is an increased risk for thrombosis, blood clots, heart attack. These patients also tend to have strong anxiety after treatment due to high blood pressure.

Patients should be counseled that this is a form of substance abuse.

If patient is taking testosterone correctly (shots, creams or gels), these should not be used during the day of treatment.

9. Other Medications

Thyroid Medications

If the patient is euthyroid then thyroid medication should be continued throughout the treatment.

Note that patients with hyperthyroidism that is not properly managed may subjectively interpret these symptoms as symptoms of opioid withdrawal. It is highly beneficial to make sure that symptoms are well managed prior to treatment, and to be mindful of this effect throughout the treatment process.

Beta Blockers

Beta blockers should not be stopped acutely, but should be tapered over the course of 7 to 14 days onto another blood pressure medication.

Psychiatric Medications

All psychiatric medication is centrally acting (Ch. 2), and, in communication with the patient's psychiatrist, should be tapered and stopped prior to treatment.

Patients who are taking long-acting psychiatric medications like a once a month shot, or who cannot stay off of their medications for 5 days, should be considered poor candidates.

If patient is stable, psychiatric medications can be re-started 24 hours after treatment.

10. Preparation for Treatment

The considerations in this chapter, and in any section titled “Preparation” in the previous chapters, are considerations for the period between receiving the patient’s application and receiving them in person for intake. In some cases, there are further instructions for these conditions noted in the chapters that follow.

Cardiovascular Pre-care

Hypertension

Patients should continue to take blood pressure medication that is not centrally acting. If patients are not on any medication, they should seek medical recommendation about a prescription for blood pressure medication. Patients should not demonstrate hypertensive crisis for at least three days prior to treatment.

Hypotension

Ibogaine may lower blood pressure in patients with hypotension and this could lead to serious complications. Check fluid intake and recommend that patient hydrate with at least 1 fl. oz. per kg of body weight throughout the day, more for physically active people. A good indicator of sufficient hydration is light or clear urine.

This intake should be with fluids that contain electrolytes such as coconut water, or other electrolyte preparation, and not with plain water or other fluids. If blood pressure does not increase with hydration, patient should be considered a poor candidate, and should seek further medical attention.

Limited Vein Access

If patient has limited vein access, they should be informed about this risk factor and should attempt to follow the following vascular pre-care protocol for at least 10 days, or if possible, until veins become easily accessible.

- Stop all intravenous substance use.
- Take 1 aspirin per day to thin the blood.
- Take 2g oral Vitamin C per day to help with vein repair.

Cleansing/Fasting

Patients will be encouraged not to engage in any cleansing regimens, to severely restrict diet or proteins (example: juice fasting, master cleanse), or any other diet that may otherwise deplete physical energy level or electrolytes from the time of application until after discharge.

Except when appetite is suppressed by ibogaine during treatment, patients will not be encouraged to cleanse or fast. Patients should be advised to eat healthy, whole meals in order to develop strength.

Nutrition & Supplementation

If patients are below the normal or preferred range of potassium and magnesium (Ch. 2), they should be instructed to supplement until normal blood test results can be returned. If results are not lower than normal, then no supplementation should be recommended. Levels higher than the preferred range present a similar risk as deficiency.

Psychological Preparation

Therapy providers should work with patients prior to arrival to maintain realistic intentions, to prepare mentally for the experience, and to begin to develop plans for continuing therapeutic care after treatment.

Informed Consent

A written copy of informed consent should be provided prior to arrival. This document should clearly explain the risks involved in taking ibogaine, as well as the treatment methods and expectations of the center.

Additionally, major potentially life-threatening risks should also be discussed prior to intake. This includes information such as:

- Stopping benzodiazepines or alcohol suddenly or within 72 hours prior to treatment may cause seizures during treatment and can be life threatening.
- Using substances such as stimulants, opiates, or others that interact with ibogaine during treatment, or misrepresenting current or previous use of these substances, greatly increases risk of ibogaine potentiating an overdose.
- Malnutrition or dehydration prior to treatment increases risk of complications which can be life threatening.
- If choosing to use opiates, stimulants, or other substances that interact with ibogaine after treatment, account for the fact that tolerance has been significantly reduced to that of an opiate naïve person. Otherwise, there is an acute risk of overdose.
- Having an active infection, abscess or inaccessible veins is an absolute exclusion criteria. If the patient has any either of these conditions upon arrival they will need to undergo further medical care. Patients should instead follow the vascular pre-care protocols mentioned above.

Travel Medical Insurance

If receiving patients who are traveling from another country, it is very important that patients obtain a travel medical insurance policy that covers emergency medical care

through the entire duration of the patient's stay. There are many suitable insurance brokers that offer affordable policies that can be purchased online.

11. Intake

The considerations in this chapter, and in any section titled “Intake” in the previous chapters, are considerations for receiving the patient in person and the beginning of the treatment process.

Informed Consent

As mentioned before, informed consent about the risks present is one of the most important ethical considerations when administering ibogaine. A copy of the written informed consent document already provided to the patient should be reviewed and signed in person with the patient and one staff member as a witness.¹ The primary emergency contact information should be confirmed again for use in case of emergency.

Secondary Screening

Electrocardiogram

Another ECG should be taken upon arrival to confirm the results of any that were submitted during the application process. Throughout the treatment process, the same exclusion criteria and risk considerations will be followed before any administration of ibogaine (Ch. 2)

Urinalysis

A urinalysis test should be administered to screen for pregnancy and all substances, including benzodiazepines, buprenorphine, and EtG and EtS (test for alcohol), to verify information submitted via the application. Analysis via GCMS or GCLS is generally available within 24-hours.

It is important for clinicians to be aware that many synthetic substances do not appear on the standard ELISA lab test. Oxycodone and fentanyl will not appear as opiates, and neither will buprenorphine unless specifically requested. Clonazepam and lorazepam will not appear as benzodiazepines.

Additionally, qualitative testing reveals the presence of a substance, while quantitative testing can reveal the concentration of that substance in the system. Sometimes slow metabolizers return positive for drugs after the recommended preparation periods mentioned in the previous chapters. Quantitative testing provides sufficient detail to verify if the patient’s reporting is accurate.

Personal Belongings

Patients should be asked to turn over any and all medications, food, or drugs, including nasal sprays, diarrhea medications, or others. Also, many therapy providers prefer that

patients report or turn over any electronics that they have with them, as well as anything else that may provide major distractions for them or other patients during the treatment process.

Patients are known to try to hide unmentioned substances in their luggage for many reasons. Many people are concerned that the treatment won't work as planned and want to be prepared to alleviate their withdrawal symptoms if they get uncomfortable. These types of situations should be expected.

Conducting a baggage search in the presence of the patient to look for any item or substance that could cause harm to them or anyone else during their stay is one way to try to avoid drugs from entering the treatment environment. However, even with a very thorough search it is not always possible to ensure that drugs aren't present, and conducting a search at the outset of the treatment may, for some patients, create an environment of distrust if not introduced with great care.

A less invasive alternative is to simply observe the patient carefully during an extended stabilization period. This time can also be beneficial for developing a therapeutic relationship.

Medical Examination

A physician should conduct a physical exam, and a review of clinical history and preparation protocols. It is advisable to re-administer blood tests if they are older than 14 days, and especially if the patient has done any other detoxifying therapy. Up to date blood tests and a current ECG should be reviewed prior to receiving clearance for treatment.

Stabilization

Patients should be stabilized on short acting opiates, preferably morphine sulfate, for at least 24 hours in order to observe the patient's psychological condition and to build a therapeutic relationship.

Refer to preceding chapters for instructions on stabilizing for opiates (Ch. 3), benzodiazepines (Ch. 4), alcohol (Ch. 5) and other drugs (Ch. 6-9).

12. Treatment

Ongoing Observation

The following observations should be considered on an on-going basis from the time the patient has cleared the intake process until the patient is ready for discharge. Detailed record keeping throughout this entire process is important (Ch. 1).

General Observations

General observations of breathing, behavior, skin color and elasticity should be monitored. Fluid and food intake should be noted consistently. Patients should be instructed to notify supervisors if they experience any palpitations, chest pain, shortness of breath, or faintness.

Hydration

All patients will be well hydrated prior to administering ibogaine. About 1 fl. oz. per kg of body weight throughout the day is recommended, or more for physically active people. All hydration done during 24 hours prior to treatment, during treatment, and for at least 72 hours after treatment, should be done with fluids that contain electrolytes such as coconut water or other electrolyte preparation, and not with plain water or other fluids. This will help to maintain electrolyte levels.

Screening Tests

Further electrocardiograms or other tests should be administered as needed, or before the beginning of any follow-up treatment episode. At all times, the same exclusion criteria and risk considerations should be observed (Ch. 2).

Pre-Treatment Diet

Prior to treatment all intensive fasts and cleanses should be avoided. Patients should eat healthy whole foods in the days prior to treatment, and should make sure to eat well the day before. However, during treatment, in order to avoid nausea, patients should have an empty stomach. It is advised to avoid eating for at least 8 to 12 hours prior to dosing in order to minimize the risk of vomiting and medicine loss.

Immediately Prior to Dosing

The guidelines laid out in the rest of this chapter supervision include considerations for the acute phase of ibogaine administration. Some clinicians have made exceptions only when risks are assessed as minimal and the measures cause extreme discomfort.

Intravenous Port Access

Dehydration and loss of electrolytes is a serious risk with ibogaine treatment. Although it is possible to hydrate with fluids orally, this can be problematic in cases of chronic vomiting, especially when cardiac concerns become acute enough to make IV anti-nausea medication an added risk factor (Ch. 13).

In order to avoid these risks, and to provide access for emergency medicines in case of arrhythmia or seizure, an intravenous port should be inserted until the physician has confirmed cardiac risks have passed.

In the absence of sufficient vein access to establish an IV port, a central line can be inserted on the chest to the same effect. This procedure, as well as its subsequent removal post-session, should be done in a hospital unless sufficiently equipped and sterilization can be ensured.

Preparatory Fluids

Administering 1 liter of lactated ringer or normal saline, along with 1 ampule of magnesium sulphate produces a beneficial fluid overload that decreases hypotension and protects the patient from developing torsades de pointes. This should be administered beginning 1 hour before a flood dose, until 2 hours after the dose is administered.

Anti-Nausea Medication

In patients with extreme physical tension, vomiting can provide a release and help the patient to relax. However, in general, most patients find this experience to be uncomfortable and physically tiring. In some cases it can also influence the oneirogenic effects.

Administering anti-nausea medication prior to treatment can help to counteract this experience. It is important to note that many common anti-emetic medications, including metoclopramide (which is also centrally acting) are known to prolong the QT interval, or like palonosetron, cause a drop in heart rate after IV administration. Pay particular attention to the cardiac effects of anti-nausea medications.

The preferred intervention is 25-50mg of diphenhydramine (IM) administered prior to ibogaine.¹

In some cases it may be sufficient to use milder remedies, such as ginger extracts or tea, and only to use anti-nausea medication when there is a significant risk of dehydration from repeated vomiting, or at the suggestion of the attending physician.

Supervision

The supervision periods recommended here are considered to begin after the administration of a single large dose of ibogaine hydrochloride. For recommendations about supervisors see Chapter 1.

Close Supervision (First 12-24 hours)

In order to minimize cardiac risks, patients should be connected to a 3 lead heart monitor and under close supervision with at least one ACLS trained staff member present to monitor cardiac changes, and a second ACLS attendant closely on hand to respond in case of emergency. The quality and rate of respiration should also be monitored closely.

Patients should remain under close supervision until the ACLS attendant has confirmed that the t-wave morphology changes have normalized for at least 1 hour. This generally takes between 12-15 hours, but can take as long as 24 hours.

Regular Supervision (First 72 hours)

After the period of close supervision, patients should remain under regular but less intensive supervision for at least 72 hours after the initial dose.

During this period, although the patient may be alert and mobile, there are still remaining cardiac risks evidenced by reports of adverse events (Alper 2012). In order to minimize the remaining risks, blood pressure, pulse and blood oxygen levels should be taken at least every 4 hours while the patient is awake, and whenever possible between sleep. At least 1 ACLS trained staff member should remain on hand in case of emergency.

Acute Confusional State

In rare instances where clients experience an acute confusional state in which they seem to have a psychological break from reality that persists after the acute effects of the ibogaine have subsided, the patient should be kept in a secure environment and under constant regular supervision until the effects of the acute confusional state have passed. Refer to the section on acute confusional state intervention (Ch. 15).

13. Dosing

Life Contract

Some providers have found it useful to make a “life contract” with patients prior to the administration of a saturation or flood dose. The contract might contain simple agreements such as that the person will not leave the space for the duration of the ibogaine’s effects, etc. but can also include the agreement that if the patient has the opportunity to permanently leave their physical body then they will come back and continue their life.

Manner of Administration

In some schools of psychedelic therapy for the practitioner to place the medicine in a container on the table rather than delivering it directly into the patient’s hand or mouth. This sensitivity provides the psychological context for the patient to accept to enter into their experience of their own free will. It is beneficial to consider this level of attention to set and setting (Ch. 1).

Test Dose

A test dose is a single threshold dose (usually 2 to 3 mg/kg) of ibogaine hydrochloride that is administered at least two hours prior to the remaining dose in order to monitor for allergic reaction, and to observe metabolic reactions to the medicine³. With sufficient clinical experience, observations should inform any further decisions about dosing.

There are varying schools of thought around the administration of a test dose prior to ibogaine treatment. With sufficient preparation and screening this is not necessary.

Extreme Weight Considerations

It is very important to consider that dosing in mg/kg might not be literal in cases of extremely over or underweight patients. In these situations it may be necessary to take into consideration the relative size of the person’s digestive system. Careful monitoring of dose response in extreme cases is highly recommended.

Hepatic Conditions

Many who seek ibogaine are diagnosed with liver conditions, or have elevated liver enzymes caused by Hepatitis C, HIV, or other conditions. Although more clinical research is needed to confirm, there are case reports of reduced viral loads after ibogaine treatment (Lots of 2006).

However, during treatment, even slightly elevated liver enzymes or liver damage can result in drastic changes in metabolism of ibogaine and other medications. This can result

in very rapid, or greatly prolonged and/or intensified effects. This should be taken into consideration and patient should be observed on a test dose for at least eight hours before continuing.

General Considerations

Patients should not receive a full dose until all the conditions for close supervision (Ch. 12) are met, and their vitals, drug use and general condition are stable. Supervisors should observe that they are well rested, have had at least two regular bowel movements, and are sufficiently nourished and hydrated.

In order to avoid complications of co-administration and potentiation of on-board opioids, dosing should not begin until significant discomfort from withdrawal symptoms is noted. Objective Opioid Withdrawal (OOWS) scores of 3 to 7 are considered optimal.

However, dosing should be delayed if withdrawal symptoms are fully expressed. OOWS scores of 10 or higher are considered excessive. If full-blown withdrawal symptoms are present, patient should be re-stabilized on morphine sulfate and ibogaine-assisted detox should resume at the next best opportunity.

Once dosing has begun it can be unnecessarily distressful to be in the midst of withdrawal symptoms while under the effects of ibogaine. Symptoms should be assessed objectively, and sufficient material should be used to reach the therapeutic goal as long as it is safe to continue administration.

It should be noted that doses in excess of 12mg/kg have been reported to have high er instances of cardiac abnormalities, and should be considered higher risk. Dosing should never exceed 24mg/kg in a 24-hour period.

Booster Doses

Booster doses are threshold doses (usually 1 to 5 mg/kg) administered outside of the single large treatment dose. Optimally, in order to avoid cardiac complications, booster doses should not be given later than 2 or 3 hours after the initial dose, or at least 24 to 36 hours later. In the days and weeks following ibogaine therapy, judicious use of booster doses can help to treat residual withdrawal symptoms (verify with SOWS and OOWS) or other unresolved psychotherapeutic issues, and to prolong ibogaine's therapeutic effects.

In some cases discomfort similar to withdrawal symptoms may present because of dehydration, lack of sleep, or other conditions. In these cases, the basic treatment course interventions (Ch. 15) are preferable to booster doses.

Patients being treated for certain SUDs, especially SUDs of extended duration and involving long-time use of long-acting opioids, may require more material in order to manage residual withdrawals and cravings. An honest assessment should be made, as early as possible, about the anticipated treatment protocol needed to reach that goal.

14. Interventions

Basic Treatment Course Interventions

Anxiety / Panic Attacks

Evaluate whether there are corresponding physiological symptoms, and respond accordingly. Then:

- Work with simple breathing practices to cultivate a sense of calm.
- Supplementation with GABA can help to calm patients that are experiencing acute anxiety. Administer 750 mg every 30-45 minutes until the patient becomes calm.
- Supplement with other GABAergic natural health care products such as L-glutamine, valerian, passionflower, magnesium, can help to stabilize patients that experience frequent anxiety, especially stimulant users.
- If anxiety persists, benzodiazepines may be administered. Doses of 5 to 10 mg of oral diazepam may be used for benzo-dependent patients. For non-dependent or benzo-naïve patients, .25 to .5 mg of alprazolam is preferred. In some cases, use of alprazolam may provide more acute relief, even in benzo-dependent patients, but watch for peaks and valleys of withdrawals. Refer to previous notes about benzodiazepines (Ch. 4).

Chronic Fatigue

Refer to Depression below.

Chronic Pain

Ibogaine, in threshold doses, has been known to reduce pain. And generally patients do not experience pain during a full dose. However, it is common for pain to emerge afterwards, especially in patients who were treating chronic pain with opiates. Learning to work with pain management effectively is a critical segue to continuing care.

Long-term opioid use is known to cause hyperalgesia, an increased sensitivity to pain (Marion 2011). This may exacerbate chronic pain symptoms in people for whom it either is or is not an underlying condition. If hyperalgesia has been ruled out as a factor, a thorough assessment of pain management options should be explored in collaboration with the patient.

If patient has detoxified from opioids, but continues to experience symptoms of chronic pain, and/or requests for pain medication to be re-administered, opioids can be prescribed under appropriate medical care in minimal possible doses. Patients should be counseled about the risks associated with the reduced tolerance, then be discharged and instructed to return to a pain management specialist.

In addition to other adjunct therapies (Ch. 1) effective pain relief has been found from the following:

- Pay attention to diet and make sure that patient eats nutritious whole foods after treatment. Avoid inflammatory foods such as gluten and dairy.
- Non-opiate, non-steroidal anti-inflammatories and analgesics, such as ketorolac 30 to 60 mg (sublingual or IV), naproxen 500 mg (oral), 3 mg diclofenac (IM).
- Spasmolytics such as carisoprodol 250 mg to 700mg oral, tizanidine 2-4mg oral, or baclofen 20 to 40 mg.
- As well as natural health products such as 1 to 2g curcumin extract, corydalis, 300 to 600 mg calcium/magnesium powder, or a magnesium oil or spray.
- Analgesic heat rubs such as diclofenac gel, BenGay, or Tiger Balm.
- .5Mhz of cranial electrotherapy stimulation for 30 minutes, twice daily.

Constipation or Impacted Bowel

It is critical that patient has at least two normal bowel movements prior to administration of ibogaine. Check that bowel sounds are positive in all four quadrants. Make sure patient hydrates, exercises, and eats foods that are high in fiber as well as vegetables and fruits such as papaya. Avoid bananas or white rice for their binding effect. A stool softener such as sennosides can be administered, but avoid other bowel preps or medications that may cause diarrhea or electrolyte loss.

Dehydration

For instructions on how to deal with dehydration during treatment or for serious episodes of acute dehydration, see Dehydration listed under Acute/Emergency Interventions later in this chapter.

If patient becomes dehydrated during the normal course of treatment, attempt to rehydrate orally. About 1 fl. oz. per kg of body weight throughout the day is recommended, or more for physically active people. All hydration done during 24 hours prior to treatment, during treatment, and for at least 72 hours after treatment, should be done with fluids that contain electrolytes such as coconut water or other electrolyte preparation, and not with plain water or other fluids. This will help to maintain electrolyte levels.

Intravenous saline fluids should be administered to pre-emptively treat dehydration in patients who experience an inability to retain fluids orally.

Depression

Depression is a common underlying condition for many people who are looking for ibogaine treatment. Because of ibogaine's serotonergic activity, many patients experience an elevation in mood. However, occasionally, patients may regress into acute depression in the acute integration period following ibogaine administration, in which case it should

be worth noting that this is often part of a healing process. Adjunct therapies to treat depression can be extremely beneficial:

- Psychotherapeutic support should be available.
- If appropriate, repeated lower doses of ibogaine can be administered.
- Many other adjunct therapies have been shown to assist in treating depression (Ch. 1).

Gastrointestinal Conditions

Many people who are seeking ibogaine treatment experience various gastrointestinal conditions. Some providers have reported a decrease in vomiting, gut infections, and related problems (such as not being able to continue administration) through the use of probiotic preparations, and natural and pharmaceutical prophylactic anti-nausea (ginger, diphenhydramine, etc.).

Insomnia

Inability to sleep should be considered a normal response for the first 24 to 35 hours after treatment. Outside of this window however, without proper rest and recovery, it can be very difficult to integrate the ibogaine experience. Prolonged insomnia may precipitate anxiety, mania or acute confusional states.

- Supplementation with natural health products such as melatonin, GABA, valerian, tryptophan, 5-HTP, etc. can help to balance sleep patterns.
- Diphenhydramine can be administered to facilitate sleep, as can judicious uses of zolpidem, eszopiclone and zaleplon.
- If insomnia persists, doses of 5 to 10 mg of oral diazepam may be administered (Ch. 4), but not recommended for longer than 1 to 3 days.

Acute/Emergency Interventions

Acute Alcohol Withdrawal

As an aid in symptomatic relief of acute agitation, tremor, impending or acute delirium tremens and hallucinosis, administer 10mg diazepam IV (preferred) or IM (via shoulder), then 5mg to 10mg in 3 to 4 hours, if necessary.

If signs of delirium tremens persist after the first dose of diazepam, continue to administer and consider calling EMS for transfer to a hospital.

Acute Confusional State

Although very rare, acute confusional states (ACS) have been known to occur after ibogaine administration, especially after long sessions involving relatively high doses. This is characterized by a noticeable psychic break from reality, during which the patient may be fully alert and communicative. This has been known to persist for periods as short as 15 minutes, but sometimes longer than 24 hours. Extreme cases have been reported of up to 14 days. In each of these reported cases patient eventually returned to their normal state.

Avoid using antipsychotic medications to try to stop a bad trip. Halperidol or thiorazine will not function as they do to interrupt other psychoactive substances, and can precipitate a very difficult psychological experience that can last for 2-5 days.

Benzodiazepines may be implicated in causing or exacerbating the ACS. However, if this is not considered a factor, benzodiazepines (for example: 5 to 10 mg of diazepam for long-acting effects, or .25 to .5 mg of alprazolam for more acute short acting effects) may be administered very carefully in order to assist in sleep and relaxation.

The transpersonal school of psychotherapy considers that an abrupt chemical intervention may interfere with a natural psychological breakthrough or resolution that may be beneficial to the individual. Several patients have noted that in retrospect the experience was personally valuable. There are no reported cases of permanent psychosis that have ever resulted from experiencing an extended ACS.

The most important factor is to maintain a physically safe space. Patient should be under constant supervision for the complete duration, and should not be discharged until they are able to care for themselves. If care providers are not, or becomes unable, to provide this level of care, then a decision should be made in conjunction with the patient's emergency contact person about how to proceed, and into which professional services the patient should be discharged.

Allergic Reactions

In the case of mild allergic reaction, 50mg of diphenhydramine can be administered (IM).

For more serious reactions such as facial swelling, IM methylprednisolone is preferable.

For anaphylactic reactions (throat closing) an epinephrine auto-injector should be administered. Patient should be connected to a cardiac monitor, airway should be monitored closely. Call EMS.

Arrhythmias

If extrasystoles are detected, follow ACLS protocols. Be prepared and monitor closely for advanced arrhythmias.

Note that T-wave changes, including flattened T-wave, are normal alterations under the effects of ibogaine. Although this would normally require ACLS intervention, monitor and treat in the event of other arrhythmias.

For ventricular arrhythmias including ventricular tachycardia, ventricular fibrillation, and torsades de pointes, check pulse. If no pulse, defibrillate when indicated. Call EMS and proceed with ACLS protocols.

Note that amiodarone, included in the ACLS guidelines for cardiac arrhythmias, is known to prolong the QT interval and should not be used if ibogaine has been administered.

Asthma Attack

Emergency steroid inhaler should be administered to prevent airway from closing. Cease further administration of ibogaine. Monitor oxygen levels, pulse and blood pressure closely. Continue to check in if they are short of breath.

Steroid medications, which are commonly prescribed for asthma, treatments are known to prolong the QT interval.

Bradycardia

If patient's pulse is sustained

If bradycardia is symptomatic:

- Check level of consciousness, BP and pulse oximetry, skin temperature. Give supplemental oxygen if pulse oxygen saturation is
- Administer a total of 1 L of Hartmann's Solution or Lactated Ringers IV.
- Consider giving atropine 0.25-0.5 mg IV every 3-5 min (max dose = 3 mg).
- Considering calling EMS.
- If patient does not respond to atropine and is still in symptomatic bradycardia call EMS and continue ACLS protocols.

If bradycardia is not symptomatic and patient is fully responsive, check to see if pulse responds to stimuli like touching their feet, having them sitting up, or walking around the room.

Dehydration

If patient becomes dehydrated due to vomiting or other reasons, or the patient's tongue is white, administer a total of 1 L of Hartmann's Solution or Lactated Ringers intravenously. Monitor vital signs more regularly.

Disordered Breathing (can sound like snoring)

Check airway, vital signs and oxygen saturation (see below). This may be a sign that blood is not circulating properly. Check for cardiac arrhythmia (see above).

Feeling Faint

If patient feels faint, check vitals and inquire about other symptoms. Encourage hydration. Sugarcane juice or other source of glucose may help if low blood sugar is a factor.

Headaches

Headaches should be treated with non-steroidal anti-inflammatory such as aspirin and ketorolac. Check for dehydration and hydrate.

Hypotension

If hypotension is due to bradycardia, treat bradycardia.

If not, is it symptomatic? Is it due to hypovolemia? If yes, push oral fluids or administer a total of 1 L of Hartmann's Solution or Lactated Ringers IV. Monitor vital signs as needed for response. If 1 Liter of IV fluid treatment is ineffective, consider calling EMS.

Opioid Overdose

Call EMS. Begin assisted breathing, and continue until EMS arrives or patient completely stabilizes.

Administer 0.4-2 mg of naloxone IV (preferred), IM (via shoulder), or subcutaneously administered immediately and repeat every 2-3 min. If no response is observed after 10 mg of naloxone hydrochloride have been administered, the diagnosis of opioid induced or partial opioid induced toxicity should be questioned.

It is important to note that naloxone can cause tachycardia, and precipitates withdrawal in opioid dependent patients. Monitor closely after administration.

If necessary, wait to stabilize until possible interactions between ibogaine and stabilizing medication are minimized. Then stabilize the patient (see end of this chapter) and observe closely until able to discharge.

Oxygen Saturation below 92%

Concerns should be raised when oxygen levels drop below 95%. Below 92%, consider supplemental oxygen 2 L per nasal cannula, titrate up as needed to keep oxygen in preferred range. If oxygen saturation cannot be maintained on 1-6 L per minute refer to ACLS protocols or transfer patient to the hospital.

Prolonged QTc interval

If patient registers a dangerously prolonged QTc interval of >500 milliseconds, then patient should be transferred immediately to a hospital for cardiac monitoring and treatment.

Shortness of Breath

If patient experiences shortness of breath, check oxygen saturation (see below).

Status Epilepticus & Recurrent Convulsive Seizures

Call EMS. Administer 5-10 mg of diazepam initially I.V. (preferred) or I.M. (via shoulder). This injection may be repeated if necessary at 10 to 15 minute intervals up to a maximum dose of 30 mg. Be alert that administering IV benzodiazepines may lead to respiratory arrest.

Vomiting

If patient vomits, check the vomit to see if any part of the medicine that has been administered was regurgitated. It may be the case that the capsule has started to decompose. Factor this into considerations about dosing. Monitor vitals more closely and look for signs of dehydration.

If vomiting persists, administer 50mg diphenhydramine (IM) every 6 hours or as needed. Refer to previous notes about anti-nausea medication (Ch. 14).

Reporting Adverse Events

GITA maintains an online Adverse Event Report form that is relevant for all situations that result in hospitalization or fatality. Information submitted is used in order to help keep these guidelines up to date.

Prematurely Terminating Ibogaine Administration

If during the course of interventions ibogaine administration has to be terminated, but the patient is experiencing ongoing withdrawal symptoms (verify using SOWS and OOWS measures), then administer small doses of morphine until withdrawal symptoms have abated, monitor closely, and reassess treatment plan.

15. Discharge

When to Discharge

Except in the case of a medical emergency in which the client is admitted to a hospital, a client should never be discharged until at least 72 hours after administration, and until any serious risks to health have subsided.

The client has the right to leave the premises when in right mind. By being offered a discharge waiver that acknowledges the client understands the risks associated with leaving the premises prior to the conclusion of the window of administration, and by being presented with any available options, the client may be permitted to leave. Patient may not be physically detained unless they are a physical threat to themselves or others.

Follow-up

A member of the staff who was reasonably well connected to the patient's detoxification process will be available for follow-up phone calls and other support for at least 6 weeks after discharge. The time spent in follow-up is not required to exceed more than 3 hours per week except in extenuating circumstances that arise directly from the treatment (for example: lasting medical conditions or psychological states).

Clients will be encouraged to seek out additional therapy, peer support programs, residential services, or any other services that may assist in their recovery. It will be impressed that accessing these services has a significant correlation with increased long-term success.

Generally healthy life-style choices are easier to ingrain after an ibogaine experience, and counseling and support around simple choices such as diet, meditation and exercise is crucial.

Availability of Records

All records and treatment notes should be kept in a confidential and secure location. The general requirements in the United States for this kind of document storage is 5 to 7 years, however, well kept data may be useful for future retrospective analysis. As stated in the Ibogaine Patient's Bill of Rights, patients have the right to review these records at any time, or to receive a copy upon request (Appendix A).

Ibogaine Patient's Bill of Rights

The *Ibogaine Patient's Bill of Rights* was originally created by Howard Lotsof for the Dora Weiner Foundation, in order to represent both the rights and responsibilities for those undergoing treatment. GITA upholds this document and provides Patient Advocacy support for individuals who feel like their rights have not been respect, or who have another grievance.

Ibogaine Patients' Rights

The following rights are granted to ibogaine patients upon successful intake into an ibogaine therapy program.

1. You have the right to understand and use these rights. If for any reason do not understand your rights or you need help in understanding your rights, the ibogaine provider must make assistance available, including an interpreter.
2. You have the right to be informed of the dose and form of ibogaine you will receive.
3. You have the right to receive complete information about your diagnosis, treatment and prognosis.
4. You have the right to participate in all decisions about your treatment.
5. You have the right to receive treatment without discrimination as to race, color, religion, sex, national origin, disability, or sexual orientation.
6. You have the right to receive considerate and respectful care in a clean and safe environment free of unnecessary restraint.
7. You have the right to be informed of the name, position, experience and credentials of the primary caregiver who will be in charge of your ibogaine therapy.
8. You have the right to receive all the information that you need to give informed consent for any proposed procedure or treatment you will receive and the possible risks and benefits of the proposed procedure or treatment.
9. You have the right to refuse treatment and be told what effect this may have on your health.
10. You have the right to be informed of alternate therapies.
11. You have the right to privacy while undergoing Ibogaine treatment and confidentiality of all information and records regarding your care.
12. You have the right to review your treatment record without charge and obtain a copy of your treatment record for which your provider can charge a reasonable fee, with the understanding that you can not be denied a copy solely because you cannot afford to pay.
13. You have the right to complain without fear of reprisals about the care and services you are receiving, and to have the provider respond to you.
14. You have the right to file a Grievance Report and have a patient advocate intervene on their behalf.

Ibogaine Patients' Responsibilities

By accepting the above granted rights, you are also accepting the following responsibilities:

1. You are responsible for providing accurate and complete information about past illnesses, hospitalizations, medications, and other health-related matters.
2. You are responsible for informing their treatment providers and other caregivers if they anticipate problems in following prescribed treatment.
3. You must take responsibility for requesting additional information or clarification about their health status or treatment when they do not fully understand the current information or instructions.

Appendix B: List of Recommended Equipment & Medications

Equipment

- 3-lead Heart monitor for each client under supervision, preferably with manual defibrillation
- Oxygen tanks, pressure regulators, tubing, nasal cannulas, masks and ambu bag/masks
- IV ports and tubing
- Automatic External Defibrillator (AED) preferred, especially if no manual defibrillators on heart monitors, or there are periods where ACLS staff would not be present to operate them
- Sterile syringes and secure disposal unit
- Scale accurate to .01g or better
- Locked box for all medications
- Blood pressure cuff
- Pulse oximeter
- Mindfold Relaxation Masks or other comfortable eye shades

Medications

- Crash cart with all ACLS meds
- Non-steroidal anti-inflammatories
- Oral & IV diphenhydramine or appropriate substitute (Ch. 12)
- Oral alprazolam
- Oral morphine sulfate
- 1L bags of Hartmann's solution or Lactated Ringers
- IV atropine
- Epinephrine
- Dopamine
- Oral & IV diazepam
- Oral & IV lorazepam
- IV naloxone
- Magnesium sulfate
- Potassium chloride

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