

**JAUNDICE EMBALMING: THE SUPERIORITY OF
GLUTARALDEHYDE VERSUS FORMALDEHYDE**

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ABSTRACT: The enormous impact of jaundice on embalming results is examined. A review of basic jaundice chemistry is conducted while examining the old-fashioned and ineffective embalming solutions for this serious embalming problem. All failure in jaundice embalming is traced to the continued use of formaldehyde in various forms as the primary arterial injectant. The inherent superiority of glutaraldehyde over formaldehyde in jaundice embalming is discussed in detail. Lab and field tests confirm the superiority of glutaraldehyde over formaldehyde in this difficult type of embalming scenario. The reluctance of the embalming industry to abandon formaldehyde for preferred alternatives that deliver better embalming results is cataloged. A summary and conclusion acknowledges and confirms glutaraldehyde as intrinsically superior to formaldehyde in jaundice embalming. Recommendations for use and implementation of glutaraldehyde as a superior and preferred embalming chemical completes the article.

*FAILURE TO SEEK CHANGE CONDEMNS ONE TO
OBSOLESCENCE.*

– JHB

INTRODUCTION: The embalming of jaundiced bodies has and always will be a serious problem in the embalming industry. It seemingly is an intractable problem with little or no workable solution. The incidences of jaundice in embalming has not waned and, in fact, the total number of jaundiced bodies

has, no doubt, increased over the years. In addition to the undesirable color problem is the inherent difficulties of preservation due to a seriously compromised disease state that promotes a high pH body by tissue saturation with nitrogenous waste products of metabolism. Consequently, the embalming problem is almost a catch-22, in that the embalming treatment modalities of the color correction and the preservation demand are in direct conflict with each other.

A jaundiced body, almost by definition, needs a maximum aldehyde concentration for adequate preservation of the tissues and, at the same time, requires that a very weak highly-buffered low-index solution be injected to minimize unappealing and disastrous yellow-greening reaction of the body when using formaldehyde. Consequently, jaundiced bodies are some of the most difficult total embalming problem cases that embalmers meet.

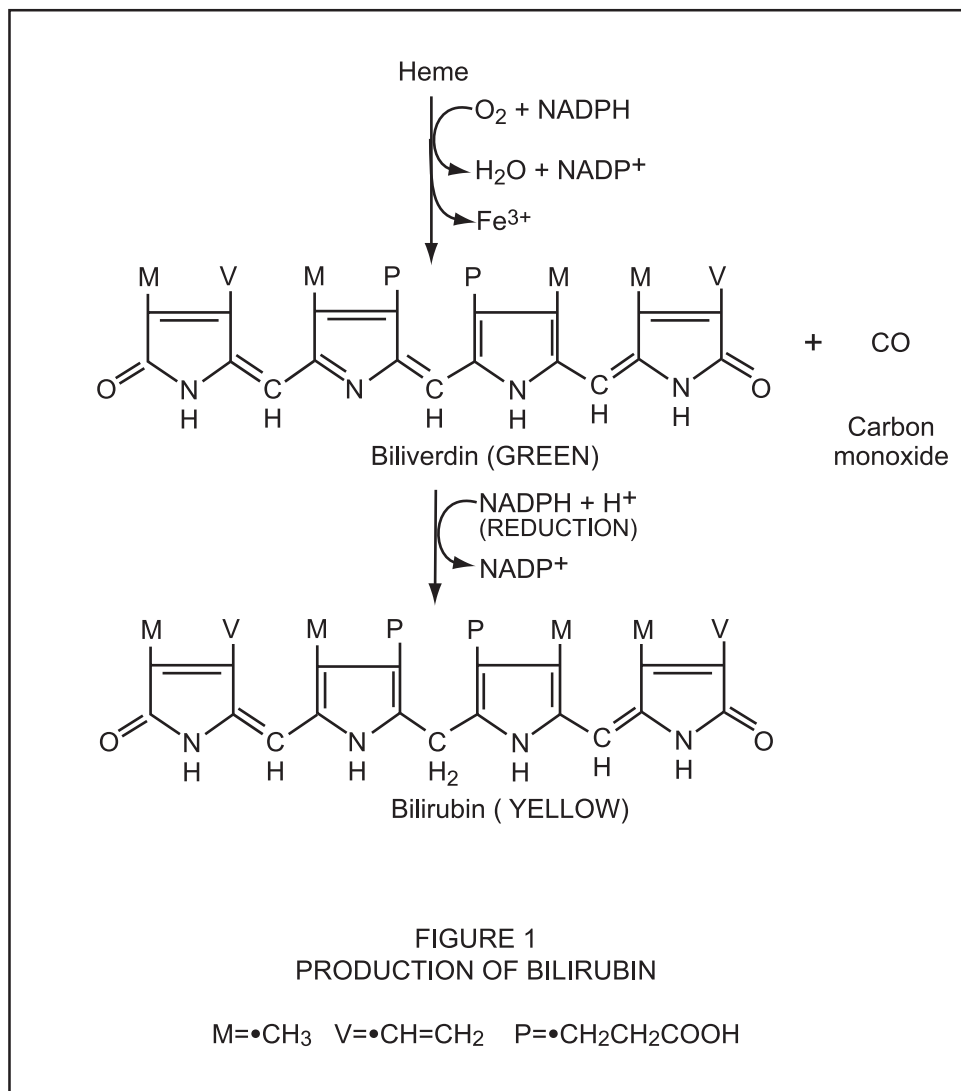
The embalming chemistry of formaldehyde in the preservation of jaundice bodies is a disaster waiting to happen. Almost all the classic tissue preservation reactions of formaldehyde enhances the yellow-greening color changes and does little to effect adequate preservation. The old-fashioned formaldehyde solutions such as low-index/high-buffer fluids, capture/binders to effect wash-out and pre-injections to clean out discoloration, basically all fail under a myriad of circumstances and cannot be counted on reliably to deliver a good embalming. Embalming jaundice bodies with formaldehyde is asking for a yellow-greening color reaction in injected tissues.

The embalming chemistry of glutaraldehyde, on the other hand, minimizes the dangerous pH changes and maximizes preservation and sanitation without implementing or encouraging the classic and undesirable yellow-greening color reaction that typically haunts jaundice embalming. The comparisons between glutaraldehyde and formaldehyde embalmed bodies is significant, repeatable and observable. Glutaraldehyde, by its intrinsic chemical reaction in protein fixation delivers an adequately preserved and embalmed body with little or no probability of a yellow-greening color reaction to the infused tissues of a jaundiced body. Lab and field test confirm the drastic difference in jaundice embalming between glutaraldehyde-based and formaldehyde-based preservative solutions, with glutaraldehyde consistently delivering the preferred embalming with virtually no undesirable color change.

By the end of this article, you will understand the reasons for the inherent superiority of glutaraldehyde versus formaldehyde in jaundice embalming and the rationale for glutaraldehyde as a preferred alternative to formaldehyde in jaundice embalming and difficult case embalming in general. This is a difficult set of facts and results for the embalming industry, as a whole, to accept. The basic formaldehyde apology-driven attitude of the industry blinds it to the blatant negatives and disadvantages of formaldehyde in many embalming circumstances and requires the belief that there are no alternatives to formaldehyde in embalming. By professing that formaldehyde and only formaldehyde will embalm dead bodies, the industry is sentencing itself to inferior embalming results in a great majority of embalming scenarios and doing a great disservice to the embalming profession. There is no logical reason to continue to embalm with massive amounts of formaldehyde, as the industry has done for over 100 years, when acceptable, preferred and superior alternative preservatives exist that drastically enhance

the overall embalming result, while minimizing the total exposure impact and disadvantages of formaldehyde.

JAUNDICE CHEMISTRY: The chemistry of jaundice, from an embalmers viewpoint, is essentially the chemistry of bilirubin, a yellowish pigment found in bile, an excretory product of the liver. Jaundice is the classic and most conspicuous sign of liver damage or hepatic disease and is characterized by generalized yellowing of the tissues of the body, most noticeably the skin and sclera of the eyes.



Bile is a golden-yellow thick, viscous, mucoid liquid produced by the liver and stored in the gallbladder, which usually contains 50-60ml of bile. Typically 500-1000ml of bile is produced by the average human on a daily basis. Bile has several essential metabolic functions including: bile acid activation of lipase enzymes and lipid emulsification, hydrolysis and adsorption of lipids, while the bile salts scavenge and absorb the lipid soluble vitamins (such as Vitamin A, D, K and carotene). Bile spontaneously air

oxidizes and exhibits a definite path of color change from yellow→golden→green→bluish/green→blue→brown→blackish/brown, due to production of various colored derivatives of bile pigments.

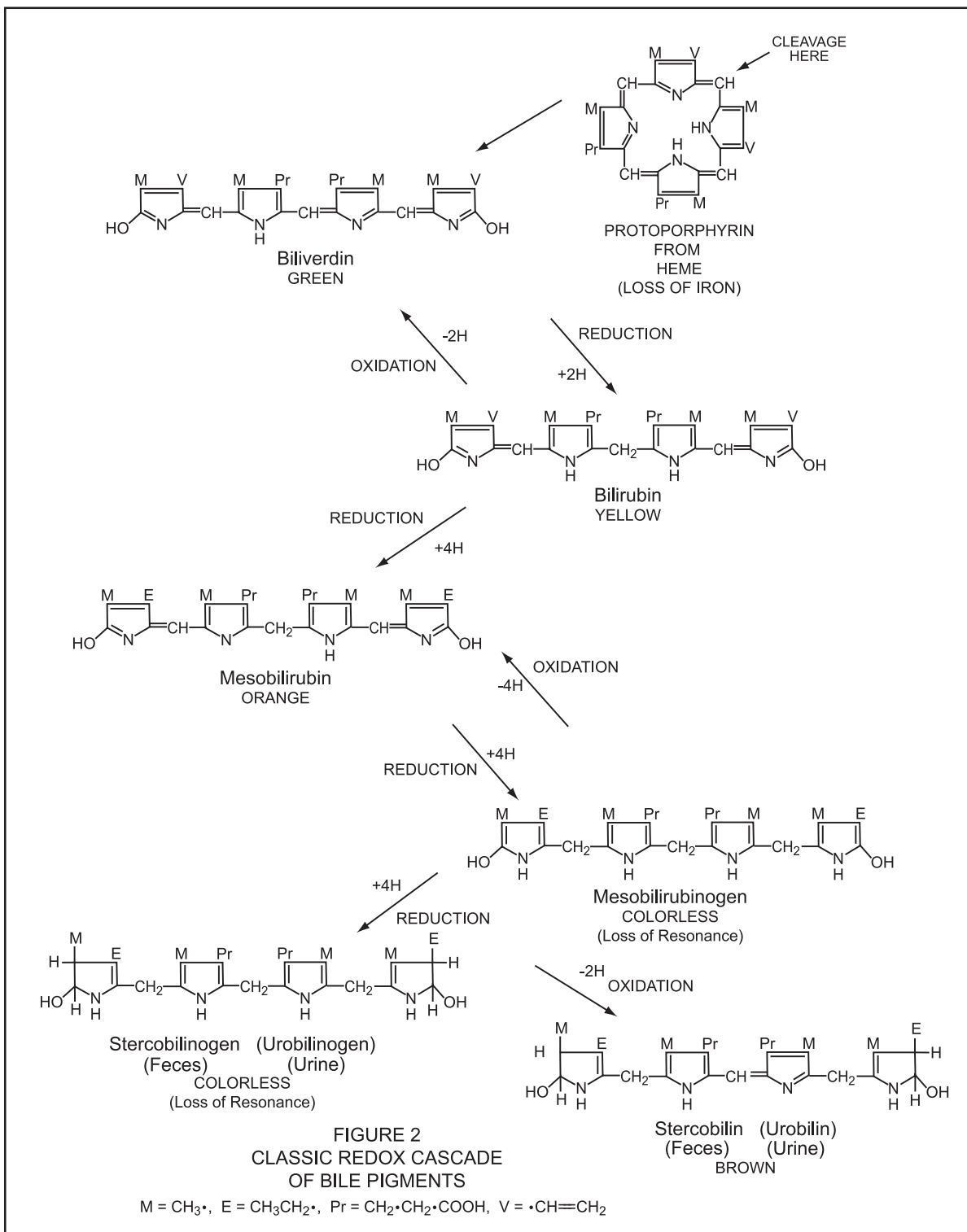
Bilirubin is the most important of the bile pigments, or bilins or bilichromes as they are sometimes referred to. Bilirubin is derived from heme pigments which are constituents of red blood cells. Hemoglobin is the oxygen/carbon dioxide transporter found in red blood cells. Surprisingly, red blood cells have an average life of 120 days in adults and even less in infants (typically 70 days). The natural breakdown and destruction of red blood cells and other hemoproteins (such as cytochrome P-450) supplies the precursors for bilirubin and the other bile pigments. Red blood cells account for 70% of these molecules and the various other hemoproteins and cytochromes make up the remaining 30%.

Bilirubin is derived from heme in basically 2 steps: loss of iron with oxygenation and methylene bridge cleavage by heme oxygenase with NADPH to form an open ring tetrapyrrole derivative called biliverdin, followed by reduction with biliverdin reductase to bilirubin, the bile pigment of immense interest to embalmers (Figure 1). An intermediate in the first step is identifiable, that being verdohemoglobin, a greenish iron containing pigment that rapidly becomes biliverdin, a green bile pigment, before reduction to the yellowish bilirubin. As a note, the deironized hemes are chemically classified as protoporphyrins. Daily bilirubin production in a typical adult human is 4mg/kg body weight.

Bilirubin is present in normal blood plasma at .3-1mg/dl and is tightly bound to albumin for transport. Plasma concentrations, therefore, are typically 5-17uM. Bilirubin is highly lipophilic, has high membrane permeability and is virtually insoluble at physiological pH, due to strong intramolecular hydrogen bonding causing high hydrophobicity. In the liver, bilirubin is conjugated as mono and diglucuronides to enhance solubility and change bilirubins' bioproperties to permit detoxification and elimination through excretion. 75% of total bilirubin in a healthy adult is in some conjugated form for nontoxicity and solubility.

Typical jaundice is hyperbilirubinemia, an excess of bilirubin in plasma and tissues due to liver disease, deficiencies in conjugation, defective hepatic uptake or dumping by a disease state through the enterohepatic circulation. The results are yellowing of the tissues and potentially serious toxicities when bilirubin levels reach higher values. Mild jaundice manifests as observable yellowing of the sclera when serum bilirubin levels reach 2-2.5mg/dl. Serious or extreme jaundice can have astronomically high values for serum bilirubin, in addition to bilirubin that is trapped in the tissues. Bilirubin in blood is tightly bound to albumin and is easily sequestered in intracellular sites. 95% of bilirubin is unconjugated in blood plasma in a healthy adult, but in a serious disease state, this can be reversed and almost all circulating bilirubin is conjugated. It is easy to see that a body dead from a serious or extreme jaundice disease state can contain a massive amount of retained bilirubin in blood and tissue.

When plasma levels exceed 300uM, a serious CNS toxicity can result called kernicterus. Kernicterus is a potentially lethal hyperbilirubinemia encephalopathy affecting the basal ganglia and chiefly found in



neonatal cases of hyperbilirubinemia. Ultraviolet phototherapy is an effective therapy for infant hyperbilirubinemia by the use of 490-510nm blue wavelength irradiation. Photo-oxidation by singlet oxygen, cis-trans isomerization and photocyclization to form a derivative pigment called lumirubin, which is

readily soluble and excretable, significantly drops blood levels of circulating bilirubin to safe values. Another treatment option is to inhibit heme oxygenase by introducing tinprotoporphyrin to stop production of bilirubin, if kernicterus is a concern.

Bilirubin that ends up in the intestinal tract is converted into various other bile pigment derivatives by normal bacterial flora residing in the colon. These derivatized bile pigments are typically very colorful and are responsible for the characteristic appearance of feces in mammals. A chart depicting this classic redox cascade of bile pigments is contained in Figure 2. Small amounts of these various pigments enters the circulation and are excreted in the urine. Typical values for urine are 1-2mg/day and 50-250mg/day for feces.

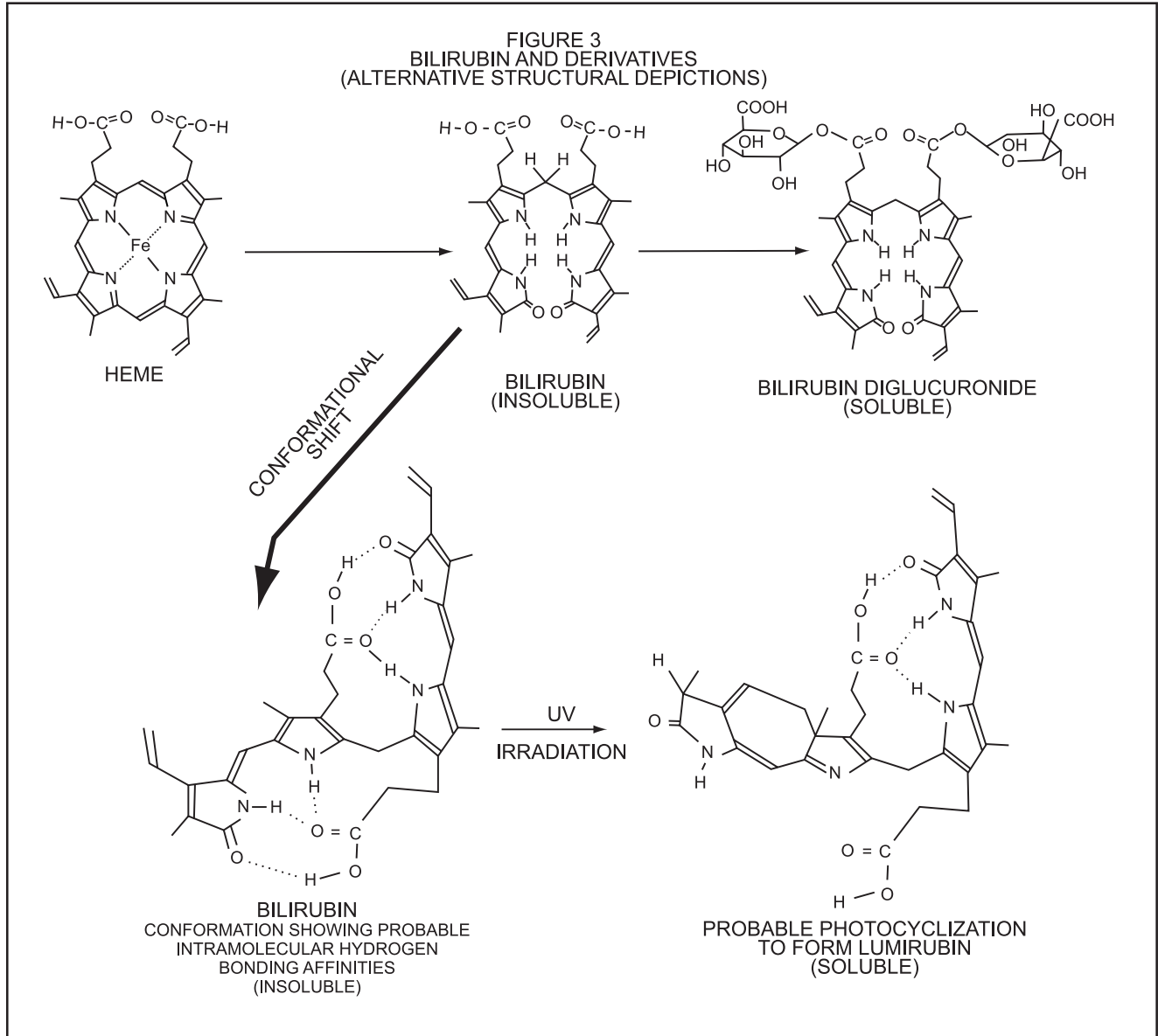
Interestingly, heme degradation stops at biliverdin (the greenish bile pigment) in birds, amphibians and reptiles and only mammals reduce biliverdin to bilirubin. Colorful porphyrin derivatives are found in various other life-forms. Red and green algae, the blue and green colors of some egg shells, bodies of colorful insects, colored fish scales and the ink secretions of gastropods (e.g. octopus) are all porphyrin derivatives.

Lab tests for bilirubin are all based on oxidation of the bile pigments by acids with a colorful layering of derivatives being seen on reaction. Gmelin's test uses nitric acid in a test tube and Rosenbach's modification uses nitric acid on dry filter paper. Van Den Bergh's analysis is the typical assay and depends on an acid-catalyzed production of an azo dye by a sulfanilic acid reagent. The color progression is that typically seen in feces from yellows→goldens→greens→blues→reddish browns.

Bilirubin, as usually depicted in a linear structure drawing, is very misleading as to the actual conformational structure of the strongly intramolecularly hydrogen bonded chemical that exhibits little solubility unless conjugated or bound for transport. In fact, looking at Figures 1, 2 and 3, you might conclude that some of the structures are incorrectly depicted. All the representations are correct when viewed 3-dimensionally. The apparent differences are due to the depiction of the possibility of keto-enol tautomers and a reverse aspect view of the tetrapyrrole chain. The keto tautomers show the hydrogen bonding capability more obviously and the enol tautomers (which is the usual and classic depiction) demonstrates the resonance effects. Figure 3 shows a probable conformation that more accurately depicts the intrinsic nature of bilirubin and its derivitization from heme and conjugation by glucuronic acid. Figure 3 also demonstrates how cis-trans isomerization affects the structure and the high likelihood of a photocyclization to lumirubin could happen.

Jaundice causes one other serious consequence for embalmers — elevated blood ammonia levels. NH₃, ammonia, is formed in the gastrointestinal tract by bacterial action and digestive processes. Most ammonia enters the portal circulation and is effectively eliminated by the liver. In liver disease states, minimal removal of ammonia occurs and bypass collateral circulation, in addition to reduced ability for enzymic conversion of ammonia to urea, extremely elevated blood ammonia levels are possible. General alkalemia is the result as blood pH values rise and ammonia readily permeates cells and tissues and

causes serious disease consequences with potentially lethal toxicities. The embalming consequence is sky high aldehyde demand and extreme formaldehyde neutralization resulting in poor or failed preservation.



Sooner or later, in embalming lore, a disease state called black jaundice is mentioned for which it is professed that there is no embalming treatment. To my knowledge, there is no syndrome called black jaundice in the modern or archaic medical literature. My best guess to explaining this bit of embalming folklore is possibly Weil's disease. Weil's disease is an old term for Leptospirosis, a bacterial infection with potentially serious and fatal consequences, if untreated. The disease is spread by the urine of rats and cavewater and its runoff is a common reservoir. The disease, in serious form, manifests as fever, nausea,

muscle ache, nosebleed, bruising and discoloring of large areas of the skin and profound jaundice. In earlier times, a body dead of this disease would look like an extreme bluish/black form of jaundice to an embalmer. The embalming result with this type of body, would no doubt have been described as awful and hopeless.

OLD SOLUTIONS: There have been several attempts at minimizing the damage of formaldehyde on jaundice cases throughout the years. The pre-injection concept seems the most obvious on the surface. However, pre-injection washout techniques with or without formaldehyde or buffers or otherwise have not had any significant success. Usually the body just ends up waterlogged and no color clearance or even lightening is rarely noticed. After understanding bilirubin chemistry, pre-injection as an anti-jaundice technique is seen to be doomed to failure. Too much pigment is tightly intermolecularly bound up in various localized tissue compartments that there is little hope for washout. Some pigment might be removed from the available blood prior to embalming, but that is all and insignificant compared to the tissues.

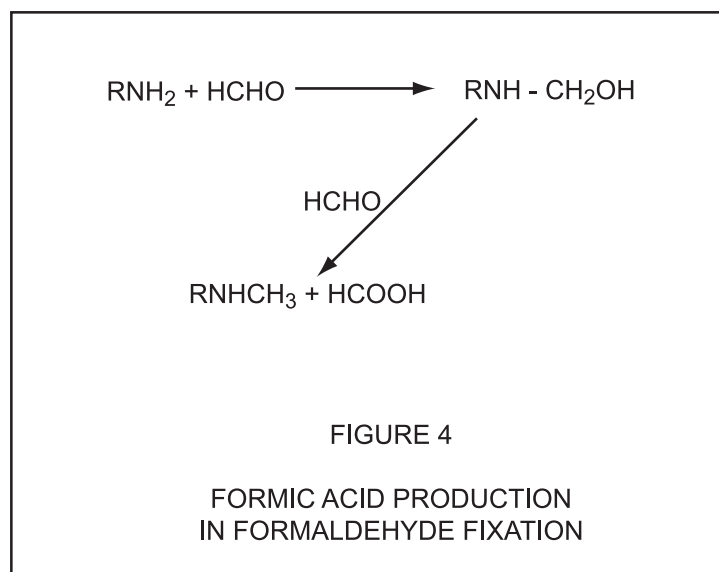
Clathrate-type or capture/bind chemicals have been attempted in the past with lackluster results, despite, on the surface, that it appears to have some theoretical validity. Obviously, the significant tissue binding of bilirubin precludes this concept having any beneficial impact on overall formaldehyde jaundice embalming results. Milk has even been professed as a vehicle for bilirubin capture and washout. Milk is no more successful than any of the other attempted methods of jaundice embalming in the past. I won't even mention the potential liabilities and disadvantages of this dubious methodology.

Formaldehyde jaundice fluids that are predicated on low index/high buffer capability are legion in the embalming industry. Everybody has got at least one. Some are reasonably good, others are fair and many are just a joke. The basic theory is embalm the body with gallons and gallons of weak formaldehyde solution that has a lot of buffer in it to hopefully control the acid explosion and minimize the greening color reaction. This is basically a hope and pray strategy — and sometimes it works. At other times, it is a dismal failure and the embalming results are truly horrendous. It is also difficult to achieve good preservation using this technique as the aldehyde demand of the body is through the roof and the more formaldehyde you use the more likely a greening reaction will occur. Despite all this, acceptable embalming results are achieved at least half the time, maybe more — which speaks well for the capabilities of some modern formulated embalming fluids. Champions' straight up formaldehyde formulation for jaundice achieves success ratios in the high 70 percentile, verified by years of field reports. Still, formaldehyde, when viewed critically in jaundice embalming, is a losing proposition and a potential disaster waiting to happen.

Some embalmers sidestep the problem altogether and just embalm the body with formaldehyde to rock hardness, accept whatever color change and other untoward reactions occur and attempt to minimize the damage with heavy internal dye during injection and a thick pancake makeup job afterward.

Occasionally, ultraviolet irradiation or even just leaving on the fluorescent lights in the embalming room has been suggested or tried as a way to reduce jaundice coloration. This is a highly improbable and uncertain methodology that is fraught with potential unwanted consequences. Color reduction is theoretically possible but unlikely and photo-oxidation to more intense darker colorations is definitely a possibility. The excretion/elimination mechanism in medical phototherapy is, obviously, not possible with a dead body and there is no guarantee that darker, more intense color derivatives will not result from UV exposure.

There have been reports that ozone generators in embalming rooms have actually darkened embalmed jaundiced bodies overnight when left running in the embalming room. Ozone is a powerful oxygen free-radical oxidizer and this would not be an unexpected result. Ozone is useless for formaldehyde control in embalming rooms and does an excellent job of oxidizing and destroying rubber, latex, vinyl and plastic materials and equipment in embalming rooms. Ozone generators are effective for odor control and little else. If you are interested in the concept of ozone and ozone generators in



embalming rooms, I have written an earlier Champion Encyclopedia that investigates this topic in-depth, and I refer you to it for reference.

THE PROBLEM WITH FORMALDEHYDE: By its very nature, formaldehyde, when used as the primary fixative, almost guarantees an unsatisfactory embalming result when jaundice is involved. This result is due to the inherent chemistry of formaldehyde in its protein fixation reaction with jaundiced tissues during an embalming operation. Formaldehyde is a reducing agent and rapid reactant with proteinaceous tissues, peptides, amino acids, amines and free or sequestered ammonia and ammoniacals. It has always been professed that since formaldehyde is a reducing agent and the conversion of yellow bilirubin

bin to green biliverdin in jaundice cases is an oxidation reaction, therefore, it is impossible for formaldehyde to be the cause, some other unknown process or chemical must be involved. An examination of formaldehyde chemistry in fixation and electroless plating demonstrates the cause of the yellow-greening reaction in jaundice embalming to be the very use of formaldehyde itself.

Formaldehyde, in various fixation reactions, typically exhibits pKa drops of 4-5 units, which drives the pKa values far into the acid titration range. Acidity promotion is a natural consequence of formaldehyde fixation whether fixing isolated tissues in the lab or embalming a cadaver. In addition, formaldehyde can react as a reducing agent with formaldehyde fixation intermediates with formic acid as a reaction product (Figure 4). This type of reducing reaction of formaldehyde with formic acid formation as a product is well documented in electroless plating reactions, where formaldehyde is used to plate out various metals. Near flawless silver mirrors can be manufactured by using a silver complex in a formaldehyde bath to effect the plating out of silver (Figure 5). The formic acid produced is immediately scavenged as ammonium formate by ammonia in the plating bath. Copper can also be plated out by formaldehyde in an alkaline solution with a thin pre-layer of palladium deposited as a catalyst. Nickel and bismuth can also be reduced with formaldehyde in a similar manner. Formaldehyde functions well in these plating reactions as a mild reducing agent that is easily controlled and neutralized.

In jaundice embalming, the use of formaldehyde as the primary preservative initiates a chain of events that causes the unwanted yellow-green color reaction to occur in most cases. The potential for the formation of formic acid as a natural reaction product in addition to the general promotion of acidification during formaldehyde fixation reactions almost insures the undesirable color changes in jaundice embalming. Bilirubin is encouraged to oxidize to biliverdin in tissues by this acidification, similar in mechanism to the standard lab test for bilirubin where oxidation by acids causes greenish biliverdin and other darker colored derivatives to appear. Formaldehyde, therefore, is the ultimate cause of yellow-green coloration changes in embalmed jaundiced bodies.

The problem of overwhelming aldehyde demand also plagues formaldehyde when embalming jaundiced bodies. Nitrogenous waste products and high titers of blood ammonia effectively neutralize a large amount of injected formaldehyde and create a high pH environment which is not conducive to good formaldehyde fixation. The formaldehyde demand in a jaundice body is very high and the obvious solution is to drastically increase the formaldehyde concentrations of arterial injection. If increased concentrations of formaldehyde is used in jaundice embalming, more fixation will occur, but so will acidification from formaldehyde reaction. The increased acidification resulting from increased formaldehyde usage will make the yellow-greening reaction almost certain to happen. Consequently, there is no acceptable or adequate solution for the embalming of jaundice bodies with formaldehyde as the primary preservative. You will either dangerously underembalm the body and count on formaldehyde neutralization to minimize the yellow-green color reaction, or you will adequately embalm the body and guarantee that a greening reaction will occur. If success ever occurs in formaldehyde embalming of a jaundice case, it is due to a combination of mildness of the disease state and just plain luck.

GLUTARALDEHYDE - THE SOLUTION: Glutaraldehyde in chemical reaction during preservation exhibits none of the disadvantages of formaldehyde in jaundice embalming. During protein fixation with glutaraldehyde only a slight acid shift to pKa's of 8-8.5 occurs, which leaves these values well in the alkaline titration range. Consequently, acidification is mild to non-existent with glutaraldehyde embalming and the potential for yellow-green acid-catalyzed conversion is highly unlikely. The reaction product is more stable and no acid moieties are produced from secondary reactions with fixation intermediates, further minimizing the possibility of color reaction due to acidification in localized tissue compartments.

Glutaraldehyde has a much wider range of pH effectiveness and can survive a much higher alkaline environment and still react effectively, while formaldehyde would essentially be neutralized. Glutaraldehyde can more effectively meet a high aldehyde demand during a jaundice embalming and deliver an adequately preserved body without the likelihood of unwanted color changes. Glutaraldehyde is always more effective than formaldehyde in compromised embalming cases where preservation is difficult to assure due to disadvantageous blood chemistry, high aldehyde demand or inhibition of fixation due to decomposition processes.

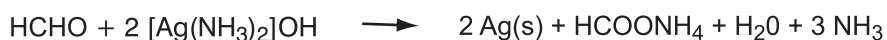


FIGURE 5

FORMALDEHYDE IN METAL PLATING
AS REDUCING AGENT
(FORMIC ACID CAPTURED AS FORMATE)

Years of field reports confirms the effectiveness of glutaraldehyde based embalming in jaundice cases and the almost 100% certainty that yellow-green conversion will not occur. Arterial injection with glutaraldehyde stand-alone fluids (such as Arterial 24) are natural jaundice fluids and allow a jaundiced body to be embalmed essentially as a normal case, without excessive dilution or the necessity for multiple gallons of injection. High glutaraldehyde/low formaldehyde blended fluids also have an excellent track record in jaundice embalming. Glutaraldehyde-based jaundice fluids (such as Jaun-Dial) which contain a very small amount of formaldehyde exhibit excellent jaundice embalming results, with color conversion being rare. These glutaraldehyde/formaldehyde blended fluids also exhibit excellent results during normal embalmings, as the basic chemistry of reaction/preservation/fixation is the same for all embalmings. Glutaraldehyde embalming followed by small dose formaldehyde addition (spiking) in the final part of the arterial injection also achieves consistently excellent results, as the formaldehyde is delivered late in the fixation reaction equation and in very small quantity.

SUMMARY/CONCLUSION: Glutaraldehyde is far superior to formaldehyde in delivering effective embalming of jaundice cases without the undesirable yellow-green color conversion. The reaction profile

and kinetics of formaldehyde are all wrong for jaundice embalming. With formaldehyde, acidification is unavoidable, yellow/green color conversion is highly likely and ineffective preservation is the probable result due to pH constraints and neutralization/complexation by ammonia and related amine chemicals.

Glutaraldehyde minimizes or eliminates the disadvantages of formaldehyde-based embalming of jaundice bodies and delivers effective preservation, even in cases of high aldehyde demand and ammonia-saturated bodies with high pH, and virtually assures that an undesirable yellow-green color reaction will not occur. Glutaraldehyde is the much preferred alternative to formaldehyde in jaundice embalming.

BIBLIOGRAPHY: The literature on bilirubin and related bile pigments is enormous. Bilirubin is well studied, tabled, charted and categorized in numerous textbooks, articles and reviews. Following is a sampling of the well-established literature. An exhaustive listing of formaldehyde references has been published by me in an earlier Champion Encyclopedia, which I refer you to, to avoid needlessly reprinting that voluminous listing.

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A Note About Figures: The depictions of bilirubin and related chemicals came from no particular source. Numerous graphs, charts, drawings and representations abound in the literature. A surprising amount are incorrect with missing double bonds and misplaced or missing functional groups and improper oxidation state. The figures presented in this article are my preferred representations of bilirubin and derivatives with all notations and notes my responsibility.