Action of Mercury in Dental Exposures to Mercury

JANET G BAUER

INTRODUCTION

Existing information from environmental, occupational, and clinical exposures to mercury has formed the basis for the toxicological assessment of the dangers of mercury in the general population and is pertinent to dentistry. A review of this material is used to assess the source of contamination in the cases that have been reported in the literature as instances of mercurial toxicity and hypersensitivity from dental exposures.

CHEMICAL FORMS OF MERCURY

The action of mercury on dental personnel or dental patients, when they are exposed to it, depends on its chemical form. Mercury may be introduced into the body as elemental mercury, inorganic mercury, or organic mercury. Although mercury generally has high affinity for the kidney, the toxic actions of the various forms can differ greatly in the type and degree of response.¹ Some of these toxic effects have been demonstrated in man and some only in animals.² Data from experimental animals are unsatisfactory because the experimental design may not correspond to the usual situation of human exposure or the animal selected may not behave like man. A ranking of toxicity in laboratory animals is useful, but only as a first step toward its assessment in humans.³

Summary

The cases cited in the literature indicate that the potential for toxicity or poisoning with mercury exists as an occupational hazard to the dentist and dental personnel. The cause is the elemental form of mercury resulting from vaporization due to an accidental spill of mercury. Undetected or unreported spills produced chronic and low level exposures.

When exposure to mercury affected the dental patient, it was during the placement of the amalgam restoration that an allergic reaction was precipitated. When allowed, the reaction was self-limiting, resolving by its own processes. For patients who are particularly allergic to mercury and not amenable to antihistamine therapy, removal of the newly placed amalgam restoration is recommended.

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Elemental Mercury

Elemental mercury, that is, free, or metallic, mercury exists in the un-ionized form and has a high vapor pressure. Elemental mercury is classified as an industrial and occupational contaminant rather than a hazardous environmental contaminant. The most important route of absorption of elemental mercury is through the respiratory tract. The proportion of the vapor of elemental mercury that is deposited and retained is high, about 80%, because it is monoatomic and soluble in lipid. After inhalation and subsequent diffusion, elemental mercury appears in the blood, partly unchanged and partly oxidized within the erythrocytes to the divalent, or mercuric, form. Elemental mercury is poorly absorbed from the gastrointestinal tract, less than 0.01%, because the mercury occurs as large globular particles.

Contamination by elemental mercury and its oxidized form, inorganic mercury, is indicated by an increase in excretion of mercury in the urine. The correlation between the degree of exposure and severity of symptoms, however, is not always good. In exposure to elemental mercury the critical organ for disease is the central nervous system because mercury is soluble in lipid, and the transfer from the blood to the brain is sufficiently rapid to give it a differential distribution that is toxicologically significant. Because the critical effects are neurologic, the relationship between dose and effect is not measurable. Levels of mercury in urine and blood are useful as indicators because they are roughly proportional to the level of exposure to mercury in the air. The best correlation with response is the actual concentration of mercury breathed by the subject, as contrasted with the concentrations of mercury in the urine or blood of exposed subjects.

In man, with exposures to concentrations of mercury in air below 0.1 mg m⁻³, Goldwater states elimination is complete; with greater concentrations, some mercury is retained temporarily combined with tissue until concentrations are reached that manifest damage to the tissue. This is why the Occupational Safety and Health Administration has set the toxic limit value (TLV) low, 0.05 mg m⁻³, more as a preventive measure than for the onset of disease which is set at around 0.1 mg m⁻³.

The kidneys have a great tolerance for, or a definite capacity to eliminate, mercury so that accumulation takes place only when the rate of absorption exceeds this capacity. Thus man is able to absorb and excrete substantial amounts of mercury without developing any untoward manifestations.

Inorganic Mercury

Inorganic mercury is the oxidized or ionized form of elemental mercury and readily forms salts and complexes, notably with sulfhydryl groups. There are two ionized forms, of which the divalent, or mercuric, form is highly soluble and thus more relevant to a discussion on toxicity, whereas the monovalent, or mercurous, form is highly insoluble. As with elemental mercury, inorganic mercury is considered an industrial and occupational contaminant rather than a hazardous environmental contaminant and is only minimally absorbed by plants and animals.

Inorganic mercury, as aerosols of mercuric salts, is absorbed mainly through inhalation, the extent to which has not been well established. In the gastrointestinal tract, inorganic mercury is absorbed to the extent of about 7%. In chronic exposures, nephrotoxicity is characterized by proteinuria, specifically the loss of albumin and proteins of low molecular weight. In severe cases, the loss of plasma protein is great enough to cause hypoproteinemia with edema of dependent parts. Exposure to inorganic mercury can also cause severe inflammation of the mouth, esophagus, stomach, and small intestine. In the erythrocytes, the divalent form of inorganic mercury binds to hemoglobin, in the blood, to plasma proteins. Inorganic mercury is distributed preferentially to the kidney; secondarily, it accumulates in the liver.

Inorganic mercury is excreted mainly through the urine, the mechanism by which is complex and not well understood except for highly toxic doses. Under toxic doses, excretion occurs by exfoliation of renal cells. It may be possible to quantify the elevated excretion of protein in the urine and thus define the relationship between the dose of inorganic mercury and its effect, the toxic manifestations being renal. This effect, however, has not been studied adequately.
Based on limited data, the clearance, or biologic half-life, of inorganic mercury is 40 days. The relationship between dose and response can only be estimated.

**Organic Mercury**

Organic mercury, in contrast to elemental mercury and inorganic mercury, is an environmental contaminant and pollutant. Although its use has been curtailed in therapeutics, the alkoxyalkyl mercury diuretics are still used. The chemical structure of the organic compounds of mercury is diverse and they vary in the stability of the carbon-mercury bonds. They include all compounds in which mercury forms a bond with one atom of carbon. As a group, they include methyl, ethyl, phenyl, and the family of alkoxyalkyl compounds of mercury.

Of most concern is methyl mercury because of its potential to enter the food chain, becoming concentrated as it moves up the chain and thus becoming a considerable toxic pollutant. Evidence for this is the serious poisoning of humans in Minimata and Niigata, Japan, as a result of eating contaminated fish, in New Mexico from contaminated pork, and in Iraq from contaminated cereal grains.

Organic mercury is efficiently absorbed in the gastrointestinal tract to the extent of 95% or greater and, after diffusion, is very stable and circulates unchanged in the blood. Unlike inorganic mercury, organic mercury is excreted mainly in the feces by two separate processes. These are biliary excretion and exfoliation of intestinal epithelial cells.

Toxic manifestations specific to contamination by organic mercury have the central nervous system as their target organ. As with elemental mercury, organic mercury concentrates to a high degree in the brain; its action, however, is distinctly different. Whereas the effects of elemental mercury are neuropsychiatric, those of organic mercury are sensorimotor. Tremor occurs, but motor effects such as incoordination, paralysis, and abnormal reflexes are more consistent and probably result from defects in the sensory input. In exposure to organic mercury, the earliest signs are paraesthesia and constriction of the visual field. At somewhat higher levels, other sensory effects such as loss of hearing, of vestibular function, and of the senses of smell and taste occur, followed by stupor, coma, and death.

Most of these effects are not known to occur in exposure to elemental mercury. Some neuropsychiatric effects occur but not consistently. Shyness and irritability are not observed but spontaneous fits of laughing and crying and intellectual deterioration are specific to this type of exposure.

The mechanism of action of organic mercury has been studied but there is no one hypothesis that adequately accounts for all the neurologic phenomena that have been observed clinically and experimentally.

The biological conversion of ingested inorganic mercury to organic mercury, specifically methyl mercury, has been demonstrated in fish, bacteria in sediment and, under laboratory conditions, in strains of bacteria from animals and humans. This conversion, or methylation, is a detoxication response that occurs under strict environmental and chemical conditions within a narrow range of pH. Further, in bacteria in sediment, the best conversion rate under ideal conditions is less than 1.5% per month. If poisoning is to occur, the sources are more likely to be contaminated food and direct ingestion of methyl mercury rather than methylation of inorganic mercury in situ. Thus the implications of inorganic mercury being released from amalgam restorations, followed by conversion to methyl mercury, assuming ideal conditions, and then absorbed in the human intestinal tract to later produce a toxic exposure are conjectural and not supported by any human clinical data.

**MODES OF ENTRY TO THE BODY**

In general, mercury can enter the body as a vapor, an aerosol, or a mixture of both, and as a free metal or compounds of metal. The most toxic forms are methyl and ethyl mercury, followed by elemental mercury, inorganic mercury, and phenyl and methoxyethyl salts of mercury. In dentistry, the possible avenues of contamination may be from the dental environment and the amalgam restoration.

In the dental environment, the exposure to mercury occurs mainly from the storage, preparation, and handling of dental amalgam and its component - mercury, which exists in the elemental form. Exposure to organic mercury
occurs from use of ointments, germicides, and sterilizing solutions. Safe substitutes for these toxic substances, however, have been recommended and are currently used, thus removing exposure to organic compounds of mercury as a potential hazard in dentistry.\textsuperscript{14,32-34}

If exposure from amalgam restorations were to occur, the likely routes would be from the placement, removal, and wear of the restoration, freeing mercury and resulting in the ingestion of the inorganic form of mercury.

**CASES REPORTED AS MERCURIAL TOXICITY**

Of the 50 cases out of 21 incidents cited as mercurial toxicity during the period from 1920 to 1980 (Table 1), the validity of the 13 cases reported between the years 1926 to 1934 has been challenged and rejected.\textsuperscript{32,38-43} The remaining documented cases reported during the period 1963 to 1978 occurred as a result of spills of mercury that went undetected or were not reported to the dentist. These irresponsible acts as well as the improper handling of mercury that led to the spills allowed the vaporization of elemental mercury to produce a chronic and toxic exposure.

The outcomes of the cited exposures varied with the onset of symptoms, averaging 62.5 days (Table 2). Two cases\textsuperscript{36-37,44} resulted in death — both involved dental assistants who succumbed from toxic effects similar to those of ingestion of a mercury salt rather than to those resulting cumulatively from high levels of elemental mercury found in occupational exposures. The death reported by Cook and Yates\textsuperscript{44} was speculated but could not be verified as an occupational exposure.\textsuperscript{37,44} Additionally, a case resulting from contamination of an office in which the affected personnel were monitored by urine analysis but were without symptoms or diagnosis of disease was reported by Pagnotto and Comproni.\textsuperscript{45} The duration of the disease in the other cases reported averaged about eight days.

The increase in excretion of mercury in the

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**Table 1. Occurrences of Mercurial Toxicity, 1920-1980**

| No of cases: | 50 |
| No of incidents: | 21 |
| Age: range | 20-61 mean - 43.3 median - 44 |
| Gender: M - | 8 cases | F - 6 cases |

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**Table 2. Onset and Duration of Disease in Mercurial Toxicity as a Result of Dental Exposures**

<table>
<thead>
<tr>
<th>1926-1934</th>
<th>1963-1968</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset of symptoms</strong></td>
<td><strong>Duration of disease</strong></td>
</tr>
<tr>
<td>None reported</td>
<td>Few weeks to 20-25 years</td>
</tr>
<tr>
<td>4 days to 6 months</td>
<td>20 months to 14 days</td>
</tr>
<tr>
<td>7.9 days</td>
<td>8.5 days</td>
</tr>
</tbody>
</table>
urine was the main systemic feature in the cases reported (Table 3). The most prevalent

biologic effect or clinical feature reported was erethism (Table 4), with this effect being reported in 34% of the cases. The features included weakness, fatigue, malaise, depression, loss of memory, feeling of hopelessness, and irritability. Erethism is a consistent, pronounced effect and an early sign of chronic exposure at low levels. Additionally, it is characterized by excessive shyness, insomnia, and emotional instability, and is accompanied by stomatitis, gingivitis, and sometimes excessive salivation and a metallic taste.\textsuperscript{2-5,7,13-16,33,37,46-52} features also reported in the cited cases.

Symptoms involving the nervous system constituted the second highest effect reported in 26% of the cases. The features included headaches, tremor, decreased reflexes, and loss of fine motor control. Tremor, a result of exposure to elemental mercury, begins in the hands and then spreads to other parts of the body with increasing duration of exposure. The reported loss in fine motor activity and the increase in salivation may indicate micromercurialism, described as one of the earliest signs of toxicity, preceding even erethism and occurring at chronic exposure to low levels of mercury. Micromercurialism is also characterized by increased excitability of the central and autonomic nervous systems, fine tremor, and salivation, but not lesions of the central ner-

<table>
<thead>
<tr>
<th>Cases Reporting</th>
<th>1926-1934</th>
<th>1963-1978</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fever</td>
<td>10</td>
<td>34</td>
</tr>
<tr>
<td>Urinary system:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatal nephritic</td>
<td>4</td>
<td>14</td>
</tr>
<tr>
<td>syndrome — uremia</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Level of Hg in urine:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2X maximum</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>2X normal</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>4X normal</td>
<td>15</td>
<td>2</td>
</tr>
<tr>
<td>15X normal</td>
<td>10</td>
<td>8</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>% of Cases Reporting</th>
<th>1926-1934</th>
<th>1963-1978</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erethism</td>
<td>10</td>
<td>34</td>
</tr>
<tr>
<td>Weakness,</td>
<td>4</td>
<td>14</td>
</tr>
<tr>
<td>fatigue, malaise</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Depression</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Loss of memory</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Feeling of</td>
<td>8</td>
<td>2</td>
</tr>
<tr>
<td>hopelessness</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Irritability</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Symptoms in</td>
<td>2</td>
<td>26</td>
</tr>
<tr>
<td>nervous system</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Headaches</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Tremor</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Decreased reflexes,</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>loss of fine motor</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>control</td>
<td>2</td>
<td>8</td>
</tr>
<tr>
<td>Visual disturbances</td>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>Pigmentation of</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>lens and retina</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Metal droplets on</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>eyes</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Digestive disturbances</td>
<td>4</td>
<td>16</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Poor appetite</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Nausea</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Stomatitis</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Metallic taste</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Burning tongue</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Sore mouth</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Increased secretions</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>Nasal</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Saliva</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Red palms</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Symptoms in</td>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>extremities</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Eczema</td>
<td></td>
<td>4</td>
</tr>
</tbody>
</table>
In 22% of the cases, visual disturbances that included pigmentation of the lens and retina and metal droplets on the eyes were recorded in chronic exposure to low levels of mercury. Digestive disturbances accompanied by diarrhea, poor appetite, and nausea were reported in 16% of the cases. Thus the evidence indicates that the toxic exposures that occurred in the dental environment resulted from the accidental contamination with elemental mercury, which was at chronic low levels not producing changes severe enough to cause permanent injury or death.

Toxic exposures to mercury in the dental environment that affected the dental patient as distinct from the personnel of the dental office were not reported. When effects of exposures were manifested in the dental patient, these were reported as mercurial hypersensitivity.

### Table 6. History of Allergy to Mercury

<table>
<thead>
<tr>
<th>Previous Exposure</th>
<th>% of Cases Reporting</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>2</td>
</tr>
<tr>
<td>Amalgam restorations</td>
<td>9</td>
</tr>
<tr>
<td>Amalgam restorations with previous allergy to mercury</td>
<td>20</td>
</tr>
<tr>
<td>Previous allergy to other mercurial-based compounds</td>
<td>8</td>
</tr>
<tr>
<td>Other allergies</td>
<td>2 or 46*</td>
</tr>
</tbody>
</table>

*Cases reported by Djerassi & Berova* cited chronic eczema but were nonspecific about relationship to current exposure.

### Table 5. Occurrences of Mercurial Hypersensitivity 1928-1980

- No of cases: 65
- No of incidents: 65
- Age: range 4–73 years mean - 22.48 median - 26 years
- Gender: M - 5 cases F - 24 cases

Almost all the cases reported a previous sensitization to mercury from either an occupational or medicinal exposure (Table 6). Thus most of the cases gave a history of allergy. The amount of mercury present during the placement of an amalgam restoration can expose the dental patient to either a sensitizing dose or the first occurrence of an allergic response. The dose can be sensitizing in particularly sensitive individuals because the allergic reaction is of the delayed type and can remain latent but active with occurrences at intervals of from 6 months to 10 years. Of the 29 cases reported by Djerassi and Berova, the origin of previous sensitization was not identified except for the presence of amalgam restorations. However, no significant relationship between contact allergy to amalgam and contact allergy to mercury could be found. When documented, the onset of symptoms occurred in a range of from 3 to 17 hours after the placement of the amalgam restoration (Table 7). The duration of the illness was about 12 days. In 20 cases, the patch test was performed as the confirmatory test for the allergic response to mercury. In four cases, pyrexia in the range 101° to 104 °F (38.3-40 °C) was the main systemic feature.

The most prevalent clinical feature reported was dermatitis in the upper extremities and around the eyes and lips, edema being the second most prevalent (Table 8). Other features reported included stomatitis, redness of the upper extremities, rash, polyps, and malaise. Oral lesions involved edema and burning of the lips, cheeks, tongue, and mucosa.

The allergic episodes were treated by removing the amalgam restorations or by letting the...
Table 7. Onset and Duration of Disease in Mercurial Hypersensitivity as a Result of Dental Exposures

<table>
<thead>
<tr>
<th>Onset of symptoms:</th>
<th>Range</th>
<th>Mean</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 cases reporting with onset groupings</td>
<td>1 hour to 2 days</td>
<td>13.6 hours</td>
<td>3 hours</td>
</tr>
<tr>
<td>earlier</td>
<td>1 hour to 7 months</td>
<td>23.6 days</td>
<td>16.8 hours</td>
</tr>
<tr>
<td>later</td>
<td>7 hours to 5 months</td>
<td>25 days</td>
<td>12 days</td>
</tr>
</tbody>
</table>

Table 8. Clinical Features as a Result of Mercurial Hypersensitivity from Dental Exposures

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>% of Cases Reporting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergic reaction, rash</td>
<td>5</td>
</tr>
<tr>
<td>Dermatitis</td>
<td>42</td>
</tr>
<tr>
<td>upper extremities</td>
<td>6</td>
</tr>
<tr>
<td>eyes</td>
<td>2</td>
</tr>
<tr>
<td>gingiva</td>
<td>2</td>
</tr>
<tr>
<td>lips</td>
<td>9</td>
</tr>
<tr>
<td>Redness of upper extremities</td>
<td>6</td>
</tr>
<tr>
<td>Edema</td>
<td>18</td>
</tr>
<tr>
<td>Stomatitis</td>
<td>8</td>
</tr>
<tr>
<td>Polyps</td>
<td>5</td>
</tr>
<tr>
<td>Malaise</td>
<td>2</td>
</tr>
</tbody>
</table>

Table 9. Treatment Regimens in Mercurial Hypersensitivity

<table>
<thead>
<tr>
<th>Treatment</th>
<th>% of Cases Reporting</th>
</tr>
</thead>
<tbody>
<tr>
<td>Removal of restorations</td>
<td>17</td>
</tr>
<tr>
<td>Antihistamine therapy both orally and topically</td>
<td>6</td>
</tr>
<tr>
<td>Corticosteroid therapy with removal of restorations</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>2</td>
</tr>
</tbody>
</table>

Disease run its course (Table 9). While removing the amalgam restorations was the earliest treatment reported, later reportings suggested antihistamine therapy as a prophylactic and for relief of symptoms while leaving the amalgam restorations intact.

In most cases cited, the allergic reaction was related more to the placement of the amalgam restoration than to the existing restoration present in the mouth, and the skin was the most common site affected. When left to resolve itself, the allergic reaction was self-limiting and tended to cease after a definite period of time as a result of its own processes. At no time did symptoms produce severe complications such as damage to the central nervous system or death. In one case, however, sensitization from occupational exposures effectively shortened the career of the affected dentist.

The same precautions used in occupational exposure — prevention and adherence to proper procedures — can significantly reduce discomfort in the sensitive individual. Sensitive individuals can be identified through an adequate medical history that would reveal previous allergic episodes. Proper procedures, such as use of the rubber dam, water spray with high-speed evacuation, cavity liners and bases, good hygiene, and properly condensed, carved, and polished amalgam restorations, as well as cleansing of any contaminated areas with soap and water, can reduce and minimize the duration of the exposure. Antihistamine therapy used topically and orally may be useful as a palliative treatment and amalgam restorations should be removed only when resolution is not otherwise obtainable.
PREVENTIVE MEASURES

The occupational hazard from exposure to elemental mercury in the dental environment has been well discussed by the dental profession, pointing out the potential causes of contamination by mercury in the dental environment. These include improper hygiene, breakable and leaking containers of elemental mercury, poorly ventilated working areas, heating the amalgam or mercury, spills of mercury that occur over cabinets with cracked and seamed tops, over floors with cracks, and over carpets. Thus prevention and adherence to proper procedures can reduce significantly the occupational hazard of mercury and have done so, with studies showing that most offices practice good hygiene and prescribe to accepted practices. Preproportioned, sealed capsules of amalgam, water spray with high-speed evacuation, adequately ventilated working areas, and proper collection of globular particles of mercury and amalgam scrap in a tightly sealed, polythene container with water, slurry of sulfur and calcium oxide, or commercial suppressant have been suggested as good preventive measures along with devices to monitor, contain, and remove contamination and spills of mercury. It has also been suggested to avoid storage of mercury around sources of heat, the use of the ultrasonic condensers for amalgam, and the use of counter tops that are cracked and seamed, floors with cracks, and carpets in the dental operator and supportive areas.

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