# WHITE PHOSPHORUS- BACKGROUND


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http://www.history.navy.mil/our-collections/photography/us-navy-ships/battleships/alabama-bb-8/NH-57483.html

United States Air Force - National Museum of the U.S. Air Force

# OBJECTIVES

- Discuss the pathophysiology of white phosphorus (WP)
- Discuss the toxic effectes related to WP
- Discuss the medical management of patients exposed to WP

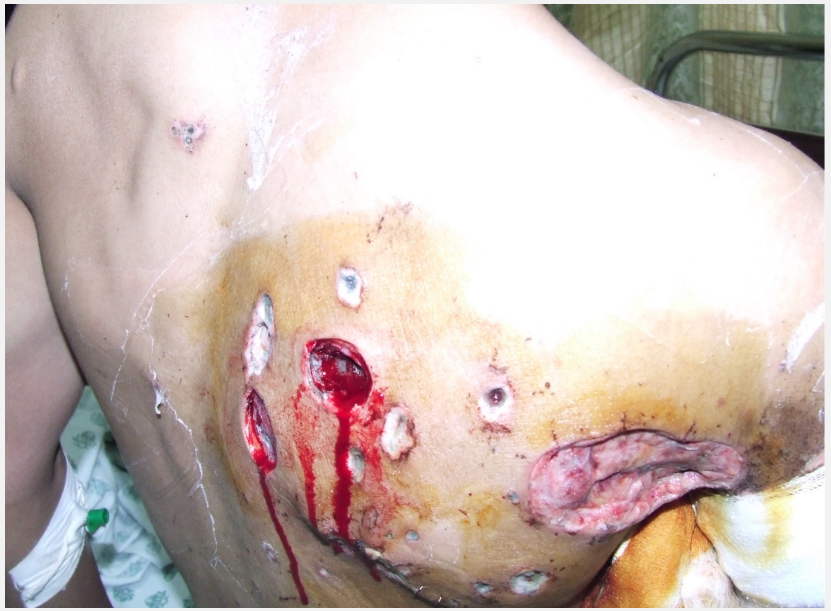
# WHITE PHOSPHORUS (WP)

- WP is a nonmetallic chemical element of the nitrogen family.
- Used in the manufacture of rodenticides, incendiaries (grenades, shells, bombs), phosphorus compounds, as an igniter in munitions and flares, as an igniter and pigment in fireworks, and as a semiconductor additive.
- WP is a smoke-producing, waxy, yellow transparent combustible solid used mainly in military and industrial settings
- It was used in the manufacture of matches in the past and was responsible for both chronic poisoning in workers and acute poisoning from ingestion of matches.

A USAF Security Police Squadron member packs an 81 mm white phosphorus smoke-screen mortar round during weapons training, 1980.


Airburst of an Israeli white phosphorus shell over Gaza City in the 2008-2009 War

https://web.archive.org/web/20130403194112/http://cc.aljazeera.net



Gazan 15 yo youth sustained severe injuries as a result of WP

International Solidarity Movement - https://www.flickr.com/photos/ismpalestine/3210017706/

# PATHOPHYSIOLOGY

# CHEMICAL +THERMAL BURN

- WP oxidizes in air ⟹ Phosphorus pentoxide + Water ⟹ Phosphoric acid (corrosive, lead to chemical injury in tissues)
- WP ignites spontaneously if temperature is greater than 34 Degrees Celsius producing thermal burns
  - Adherence of phosphorus to clothing and skin will often cause thermal injury
- When exposed to air in darkness, greenish light is emitted and white fumes with a garlic-like odor are released
- Lethal human dose 50–100 mg

**In the presence of oxygen, it spontaneously ignites with a yellow flame and produces dense smoke**

**It extinguishes only when deprived of oxygen or totally consumed.**

**On contact with exposed skin, white phosphorus produces painful chemical burns**

yellowish, necrotic, full-thickness lesions due to both chemical and thermal components.

WP is high lipid solubility, the injuries often extend deep into underlying tissues with resultant delayed wound healing.

# SYSTEMIC TOXICITY

- Systemic phosphorus poisoning may occur following oral, inhalational, and dermal exposure.
- Phosphorus poisoning may result in multiple organ dysfunction because of its effects on the erythrocytes, heart, brain,  kidney, and liver (acute fatty degeneration of the liver), and other organs
- Early sudden death due to cardiovascular collapse may be induced by hypocalcemia or a direct cardiotoxic effect.
- Induced hypoglycemia has been responsible for deaths in the past.
- Phosphorus-induced liver injury may be due to free radical injury and/or inhibition of protein synthesis.

# WP COMPLEX INJURY

The complexity of injury seen with phosphorus burns can extend to the damage caused by grenades going off in the hands of soldiers.

Both phosphorus particles and grenade fragments embed themselves in the wound. If the temperature is high enough, spectacular smoke will sometimes be seen as phosphorus particles exposed to the air, ignite.

Systemic effects including hypoproteinemia, acute renal failure, hematuria, oliguria, hypocalcemia, generalized petechiae, hepatotoxicity, icterus, acute yellow atrophy of the liver, seizures, impaired glycogenolysis, hypocalcemia, and ischemic-like ECG changes can arise quickly

# CLINICAL TOXIC EFFECTS

# OCULAR

Ocular injury includes foreign body sensation, excessive tearing, blepharospasm, and corneal defect evident by fluorescein staining, corneal perforation, endophthalmitis, and ectropion.

Exposure to phosphorus oxides causes eye irritation, blepharospasm, photophobia, and lacrimation.

Direct eye contact can cause severe eye damage.

# DERMAL

Dermal exposure may result in severely painful, necrotic, partial to full thickness yellowish color burns from chemical and thermal effects with a garlic-like odor.

Second and third degree burns can occur within a few minutes to hours. Phosphorus absorbed from damaged skin may result in acute systemic phosphorus poisoning

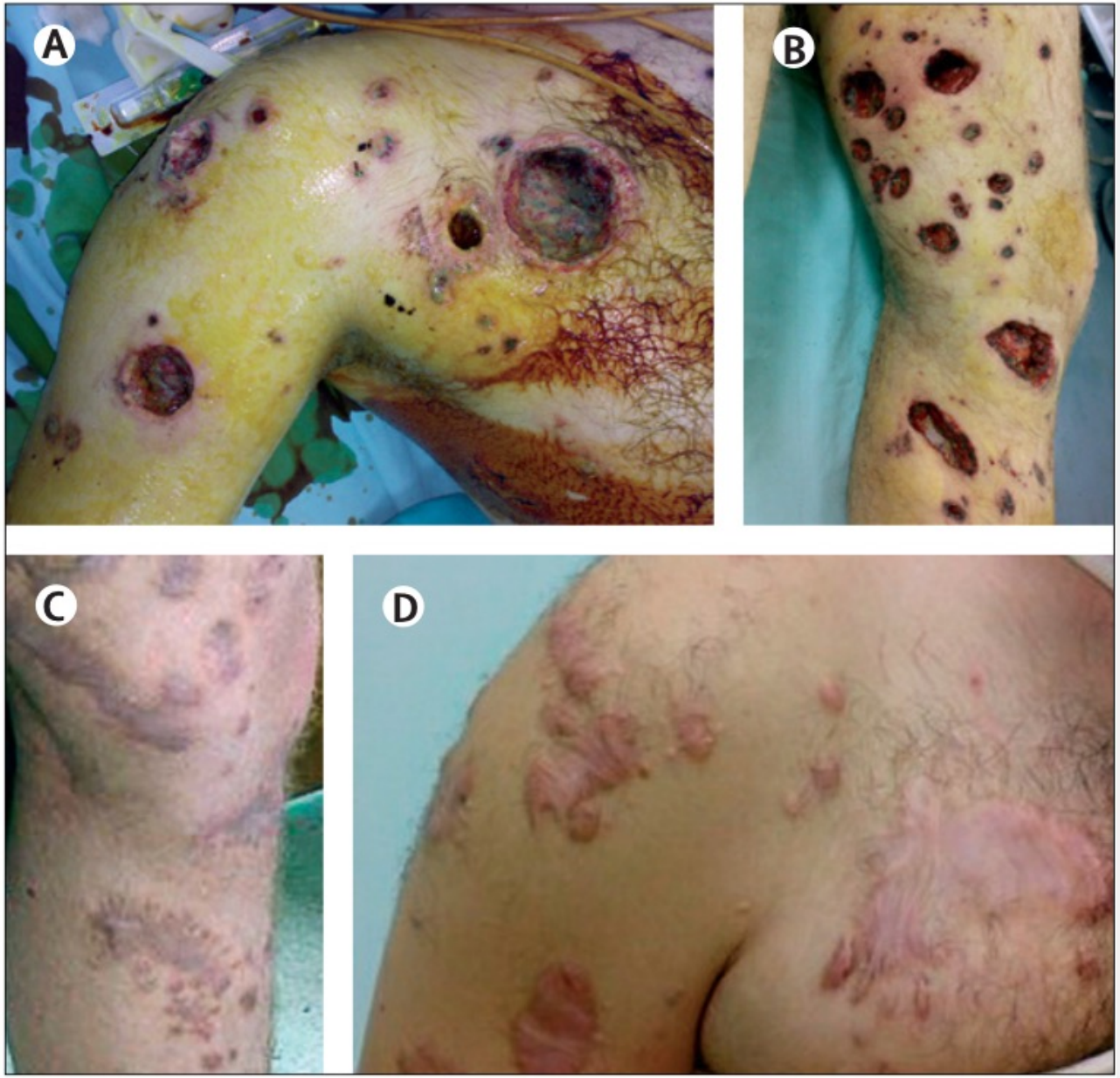



***Figure:*** **White phosphorus burn**
Many lesions, with severe underlying destruction and necrosis in the right shoulder (A) and left leg (B). After 16 months of follow-up (C, D).

- In January, 2009, an 18-year-old man presented to the ED after an attack with an incendiary shell
- painful patches of full- thickness burns, which were surrounded by sloughed tissue. His wounds covered 30% of his body surface area, and were distributed on both upper and lower limbs, and his right shoulder.
- debridement and excision of necrotic tissue, and removal of white phosphorus particles.
- After 8 days in hospital, the patient was relatively well, and was discharged without any systemic complications.
- At 16-month follow-up, our patient was well; however, hypertrophic, mildly tender scars remained on his chest, arm, and thigh (figure C and D).

*Al Barqouni Loai, et al.* ***Lancet*** **2010; 376: 68**

# INHALATION

upper respiratory irritation and possibly delayed onset of acute lung injury.

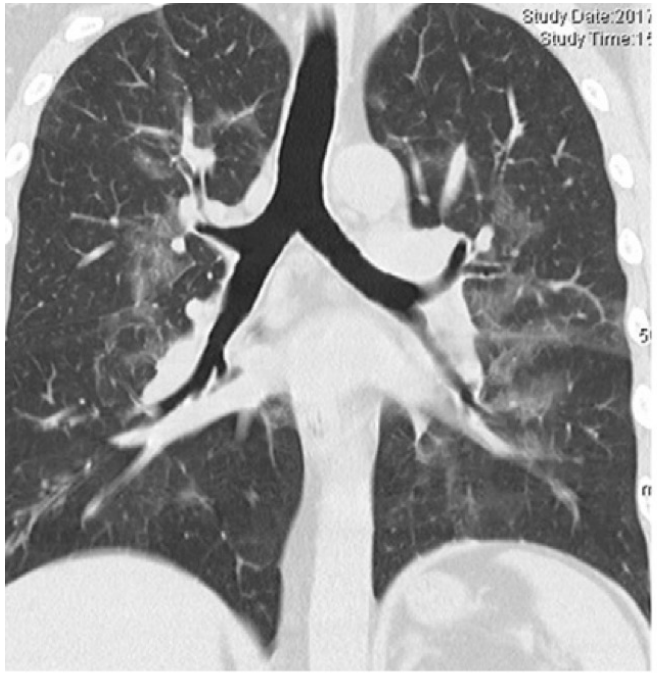
conjunctivitis, ocular irritation, and mucosal irritation of the nose and throat

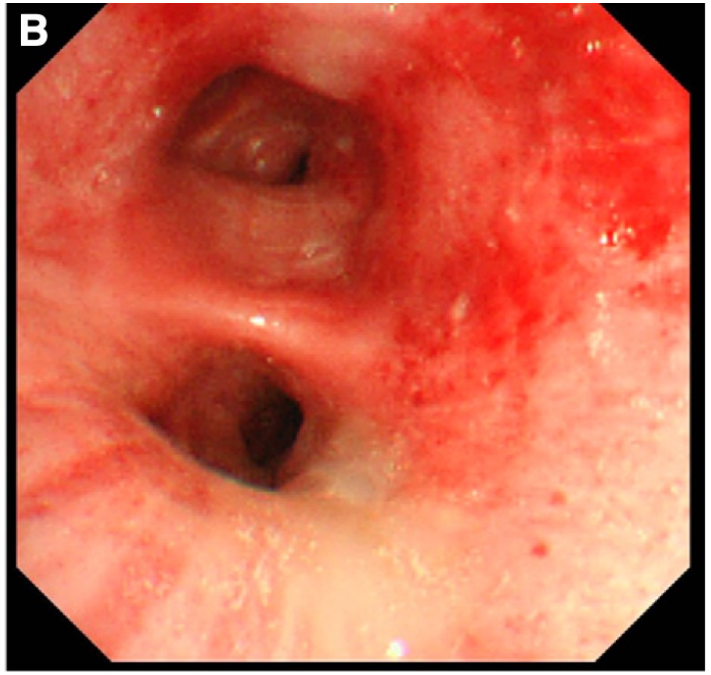
Tachypnea, shallow respirations, and hyperventilation may occur. Laryngospasm causing dyspnea and or apnea

Acute hepatic damage has also been reported.

Chronic industrial inhalational exposure to phosphorus fumes has resulted in symptoms that include bronchitis, anemia, cachexia, and mandibular necrosis ("phossy" or "Lucifer's jaw").

Chemical pneumonitis






# CHEMICAL PNEUMONITIS CAUSED BY INHALATION OF WHITE PHOSPHORUS FUMES

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# ORAL

- Severe poisoning may manifest as severe electrolyte abnormalities (eg, hypokalemia, hyperchloremia, hypocalcemia and either hyperphosphatemia or hypophosphatemia), hypoglycemia, encephalopathy, cardiac dysrhythmias, liver necrosis, and hepatic and/or renal failure.
- Other effects following oral ingestion may include clotting abnormalities, hypoprothrombinemia, thrombocytopenia, leukopenia, anemia, pancytopenia, tachypnea, shallow respirations, hyperventilation, and laryngospasm causing dyspnea and/or apnea.
- Death usually occurs 4 to 8 days after ingestion, but may be delayed.
- Death in the first 12 hours is usually the result of peripheral vascular collapse.
- Death within 24 to 48 hours may ensue from peripheral vascular collapse and is frequently accompanied by acute renal failure.
- Deaths within 48 to 72 hours may result from peripheral vascular collapse or cardiac arrest with hepatic and/or renal failure.

# CARDIOVASCULAR EFFECTS

CARDIOTOXICITY: Early cardiovascular collapse may occur after ingestion and may represent a direct phosphorus cardiotoxicity

Cardiovascular collapse may lead to death within 24 to 48 hours, in the first stage of poisoning

Various dysrhythmias, including nodal rhythms and ventricular fibrillation, have been noted after ingestion

ECG changes include tachycardia, ST-T wave changes, prolonged QTc, low voltage QRS after ingestion (

ECG changes may be due to a direct toxic action of phosphorus on the myocardium or may be due to metabolic disturbances.

Hypocalcemia and hypomagnesemia with associated QTc prolongation and ventricular dysrhythmias including bigeminy, ventricular tachycardia, refractory ventricular fibrillation, and cardiac arrest may occur following ingestion and dermal exposure

Bradycardia has also been reported

# GU

Abnormal renal function tests, hematuria, albuminuria, and oliguric or anuric renal failure may develop following ingestion

A common pathological finding at autopsy is fatty degeneration of the liver and kidneys

CASE SERIES: A study evaluated 11 cases of poisoning in children (median age, 36 months; range: 24 to 48 months) after ingestion of an unknown amount of fireworks (typically containing 10% yellow phosphorus and 50% potassium chlorate) and matchsticks, from February 2008 to June 2014, and identified renal impairment in 3 patients; dialysis was not required in any patients (Yuksekkaya et al, 2019).


# CNS

- Toxic encephalopathy
- Neurological symptoms may occur soon after ingestion or late in the clinical course with fulminant hepatic encephalopathy (
- Lethargy, irritability, delirium, generalized weakness, seizures, and coma may be seen
- Headache, seizure, confusion, Loss of consciousness

# MANAGEMENT

# PPE

- Dust mask; gloves of rubber vinyl; chemical safety glasses; rubber shoes.

- Use flame-retardant clothing, gloves, face shields (eight-inch minimum), and any other appropriate protective clothing to prevent any possibility of skin contact. Use dust and splash-proof safety goggles. Any clothing which becomes contaminated should be removed immediately.

- Respiratory protection (supplied-air respirator with full facepiece or self-contained breathing apparatus) should be available where these compounds are manufactured or used and should be worn in case of emergency and overexposure

## DECONTAMINATION
PREHOSPITAL

- First aid management of white phosphorus burns includes removal of the patient's clothes and application of saline or a water-soaked dressing
- Emesis is not recommended because of the corrosive potential of phosphorus.
- Phosphorus absorption is enhanced when dissolved in solvents (eg, alcohol, digestible fats, oils). These agents are contraindicated in the management of oral or dermal phosphorus exposure.
- Activated charcoal is never indicated.
- Following dermal exposure, prompt removal of all clothing, including jewelry, and copious irrigation with cool water should occur as soon as possible.
- Cool water- or saline-soaked dressings applied to the affected area will allow the patient to be transported without re-ignition of the remaining particles.
- Keep dressing moist until debridement is accomplished

# ED MANAGEMENT

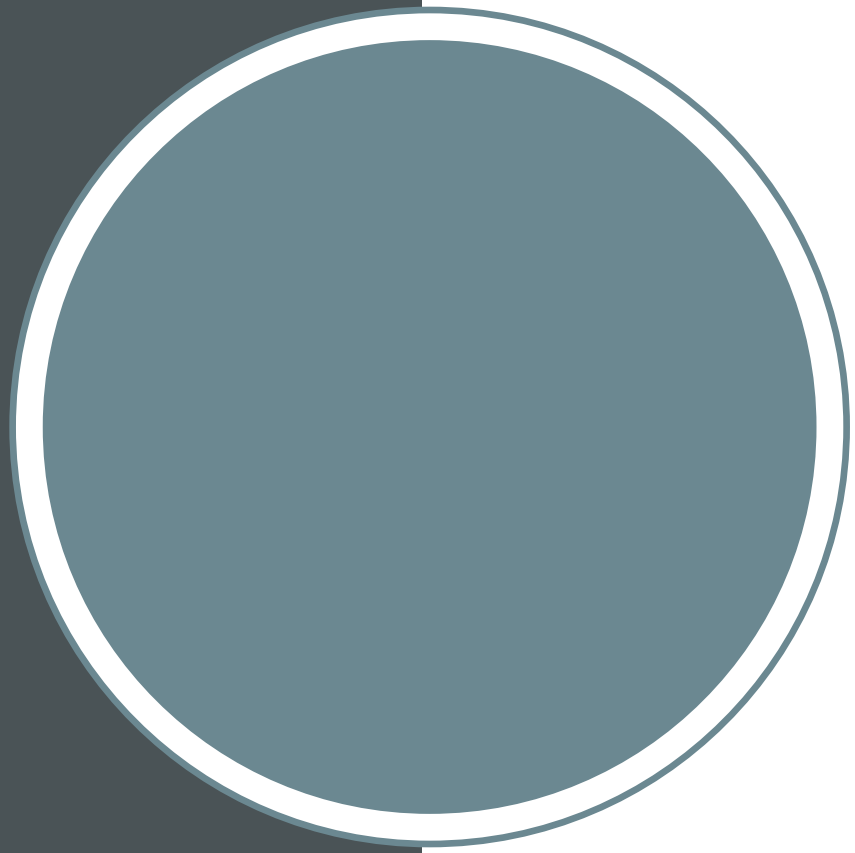
- Wear PPE
- Continuous irrigation with water is recommended to minimize the complications of the burn
- large easily identifiable particles of WP should be debrided

# INHALATION EXPOSURE

- Move patient to fresh air.
- Monitor for respiratory distress.
- If cough or difficulty breathing develops, evaluate for respiratory tract irritation, bronchitis, or pneumonitis.
- Administer oxygen and assist ventilation as required.
- Treat bronchospasm with an inhaled beta2-adrenergic agonist.
- Consider systemic corticosteroids in patients with significant bronchospasm.

# DERMAL EXPOSURE

- Brush all nonadherent phosphorus from the skin.
- Avoid application of any lipid based ointments as these may increase the skin penetration of phosphorus.
- Remove clothing and promptly begin continuous water irrigation of the affected site
- Continuous irrigation can prevent further oxidation and allow removal of white phosphorus particles from the skin surface without re-ignition
- Water- or saline-soaked dressings applied to the affected area will allow the patient to be transported without re-ignition of the remaining particles. Keep dressing moist until debridement is accomplished



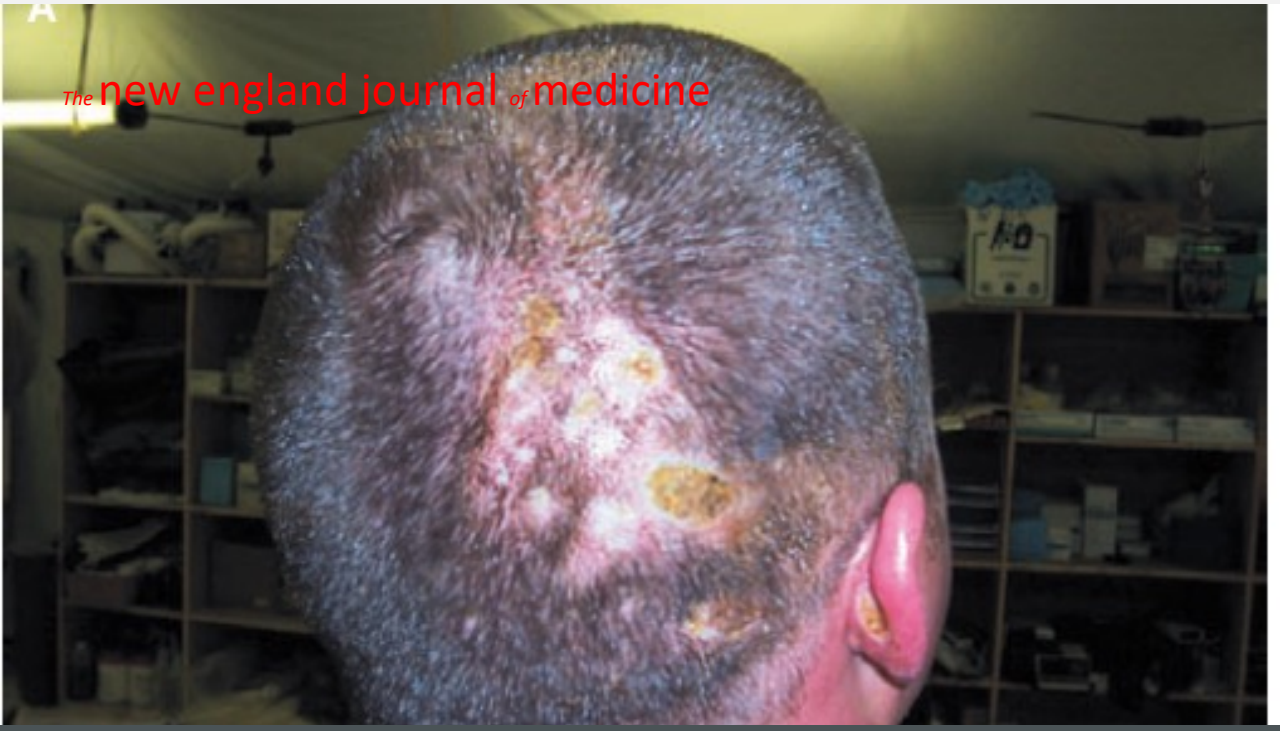
- submerge affected area in cool water or covering any areas with clean materials soaked in water or 0.9% sodium chloride solution to limit the white phosphorus contact with atmospheric oxygen
- Fragments from the wound should be placed under water to prevent a fire hazard.
- Wound debridement (Because of the increased solubility of Ointments until the wound is completely decontaminated)
- Copper (II) sulfate solution: not recommended anymore due to systemic toxicity of copper / silver nitrate can be used instead

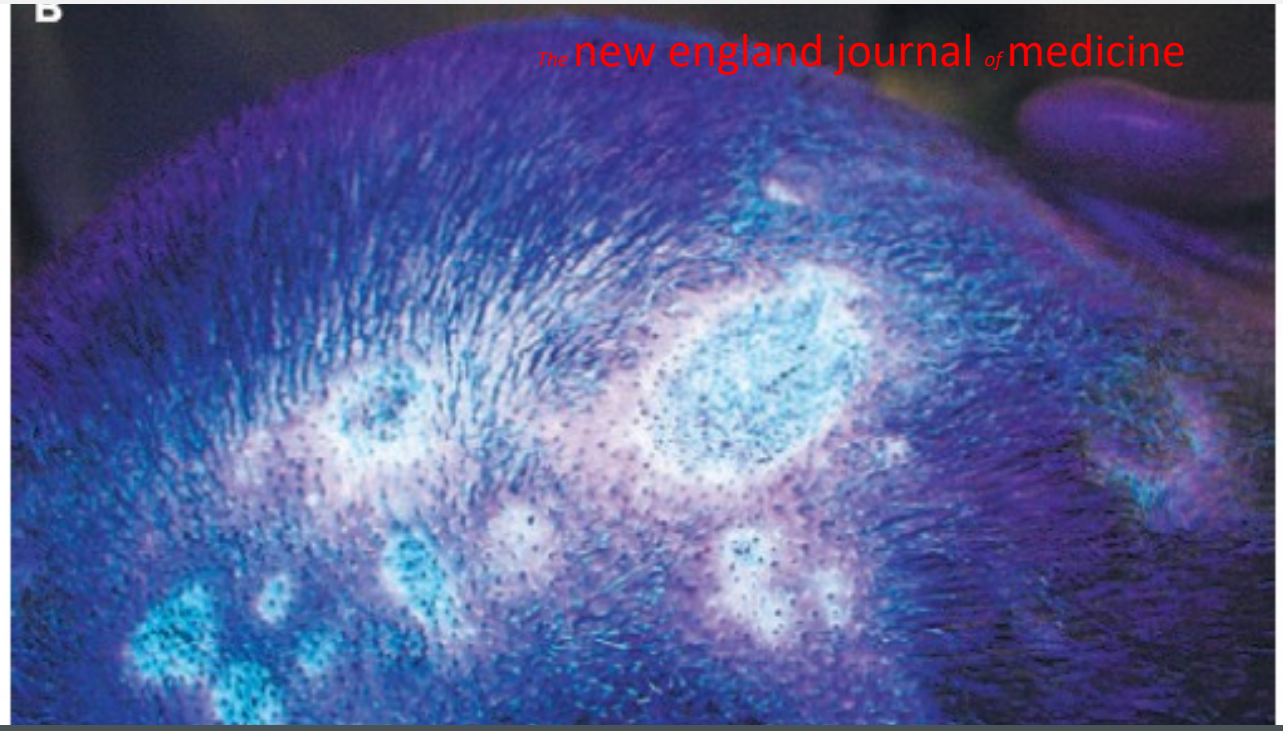
# COPPER SULFATE

- Traditionally copper sulfate solution has been topically applied to skin burns caused by yellow phosphorus
- The rationale for the use of copper sulfate is based on a chemical reaction that binds up the phosphorus, thereby preventing further burning due to phosphorus oxidation. The granules of Cu3P2 are black and decompose easily (Zong-Yue et al, 1985). 3Cu(+2) + 2P + 6e --> Cu3P2
- Cu3P2 + 402 --> 3Cu(+2) + 2PO4(-3)
- CAUTION - Acute renal failure and massive hemolysis may occur if significant copper sulfate is absorbed from the burn site

# SILVER NITRATE

- Silver nitrate application to a burn area caused by phosphorus can aid in visualization of the imbedded phosphorus, although it too can be absorbed and produce systemic silver poisoning
- The silver coats the phosphorus and prevents further combustion.
- $5AgNO_3 + P + 4H_2O \rightarrow 5Ag + H_3PO_4 + 5HNO_3$
- $6AgNO_3 + P \rightarrow Ag_3P + 3AgNO_3 + 3NO_3(-)$
- Additional studies are needed to determine the efficacy of silver nitrate in the treatment of phosphorus-induced skin burns.




# PHOSPHORUS VISUALIZATION

- Visualization of phosphorus particles may fluoresce under an ultraviolet light source (black light, Wood's lamp).
- With the exposed areas immersed in water, loose or imbedded phosphorus particles that are visualized under UV light can be mechanically but delicately removed safely under water.
- This technique is a safer alternative than either the use of copper sulfate or silver nitrate, and should be the method of choice

# IMBEDDED PHOSPHORUS REMOVAL -

- Remove visualized particles delicately with metal forceps
- Remaining particles may be detected by discontinuing the moist dressing long enough for oxidation to begin again, which can be visualized as smoke being released from the area where an imbedded particle remains
- For deep and extensive injury, consider prompt excision to the fascia and skin grafting.

# OCULAR EXPOSURE

- DECONTAMINATION: Remove contact lenses and irrigate exposed eyes with copious amounts of room temperature 0.9% saline or water for at least 15 minutes.
- If irritation, pain, swelling, lacrimation, or photophobia persist after 15 minutes of irrigation, the patient should be evaluated by an ophthalmologist
- Keep exposed eyes covered with wet dressings until definitive surgical removal of phosphorus can be accomplished.

# BURNS

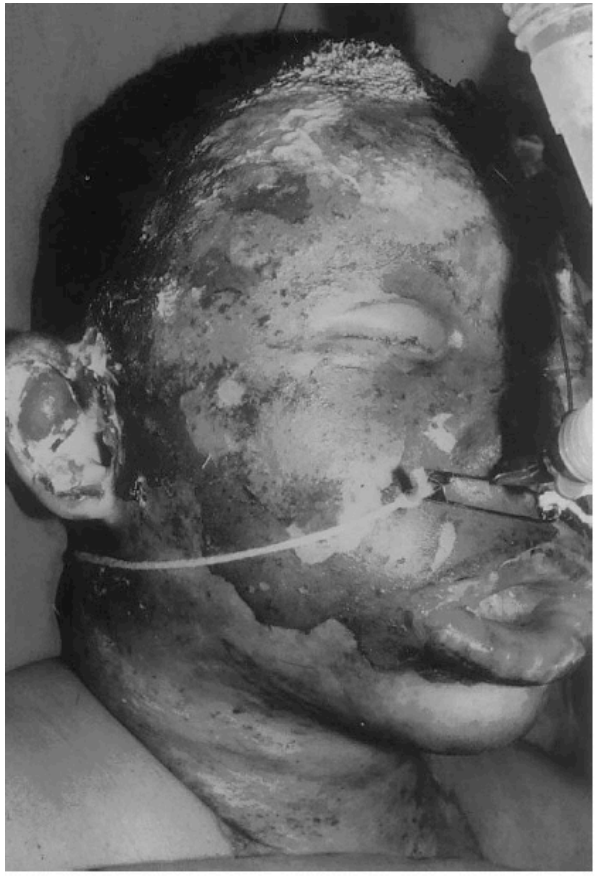
MINOR chemical burns (FIRST DEGREE; SECOND DEGREE: less than 15% body surface area in adults; less than 10% body surface area in children; THIRD DEGREE: less than 2% body surface area).

DEBRIDEMENT

**a)** After initial flushing with large volumes of water to remove any residual chemical material, clean wounds with a mild disinfectant soap and water.

**b)** DEVITALIZED SKIN: Loose, nonviable tissue should be removed by gentle cleansing with surgical soap or formal skin debridement

**c)** BLISTERS: Removal and debridement of closed blisters is controversial. Current consensus is that intact blisters prevent pain and dehydration, promote healing, and allow motion; therefore, blisters should be left intact until they rupture spontaneously or healing is well underway, unless they are extremely large or inhibit motion



4. The whole face revealed generalized swelling with fish-mouth earance after phosphorus flame burn, and nasotracheal intubation used for keeping airway patent.

# ANTIBIOTICS

- TREATMENT
  **a)** TOPICAL ANTIBIOTICS: Prophylactic topical antibiotic therapy with silver sulfadiazine is recommended for all burns except superficial partial thickness (first-degree) burns
- For first-degree burns bacitracin may be used, but effectiveness is not documented
  **b)** SYSTEMIC ANTIBIOTICS: Systemic antibiotics are generally not indicated unless infection is present or the burn involves the hands, feet, or perineum.

# DRESSING

- WOUND DRESSING:
  Depending on the site and area, the burn may be treated open (face, ears, or perineum) or covered with sterile nonstick porous gauze. The gauze dressing should be fluffy and thick enough to absorb all drainage.

- DRESSING CHANGES:
  **1)** Daily dressing changes are indicated if a burn cream is used; changes every 3 to 4 days are adequate with a dry dressing.
  **2)** If dressing changes are to be done at home, the patient or caregiver should be instructed in proper techniques and given sufficient dressings and other necessary supplies.
- Analgesics, tetanus prophylaxis

2765 ADMISSIONS BETWEEN 1984 AND 1998, 326 PATIENTS HAD CHEMICAL BURNS (7 DUE TO WP)

- 1% copper sulfate solution for neutralization and identification of phosphorus particles,
- copious normal saline irrigation
- keeping wounds moist with saline-soaked thick pads even during transportation
- prompt debridement of affected areas
- porcine skin coverage or skin grafts for acute wound management
- intensive monitoring of electrolytes and cardiac function in our burns center.
- Intravenous calcium gluconate is mandatory for correction of hypocalcemia.
- Of the seven, one patient died from inhalation injury and the others were scheduled for sequential surgical procedures for functional and cosmetic recovery.
- Cooling affected areas with tap water or normal saline, prompt removal of phosphorus particles with mechanical debridement, intensive monitoring, and maintenance of electrolyte balance are critical steps in initial management.
- Fluid resuscitation can be adjusted according to urine output. Early excision and skin autografts summarize our phosphorus burn treatment protocol.

T.-D. Chou et al. The management of white phosphorus burns . Burns 27 (2001) 492–497

# MONITORING

- CBC and platelet count
- serum electrolytes, liver enzymes, renal function, urinalysis, urine output
- ECG, urine output, serum calcium and phosphorus concentrations, and prothrombin time.
- Following inhalational exposure to phosphorus fumes, evaluation should include pulse oximetry, chest radiograph, arterial blood gases, and pulmonary function tests.

- In critically ill patients, excision of the necrotic tissue and skin grafting, plus appropriate fluid replacement, and close monitoring of electrolytes and ECG are required to avoid predictable complications like hypocalcaemia, hyperphosphataemia, and cardiac arrhythmia.

# ORAL EXPOSURE

- Emesis is not recommended because of the corrosive potential of phosphorus.
- Phosphorus absorption is enhanced when dissolved in solvents (eg, alcohol, digestible fats, oils).
  - These agents are contraindicated in the management of oral or dermal phosphorus exposure.
- Activated charcoal is never indicated.
- NG: Consider insertion of a small, flexible nasogastric tube to aspirate gastric contents after large, recent ingestion of caustics.
  - The risk of worsening mucosal injury (including perforation) must be weighed against the potential benefit.
- Supportive Rx: Treatment of arrythmias, seizure control, airway protection, electrolyte disturbances

# DISPOSITION

- White phosphorus burns are associated with significant morbidity often necessitating lengthy hospital stays. Extreme cases can be fatal.
- ADMISSION CRITERIA/ORAL
  Patients with severe symptoms should be admitted to the hospital. Patients with persistent cardiac dysrhythmias, mental status changes, seizures, and respiratory failure should be admitted to an ICU setting. Patients with significant burns should be admitted to a burn center.
- HOME CRITERIA/ORAL
  Patients with white phosphorus exposure should be evaluated in a healthcare facility.
- CONSULT CRITERIA/ORAL
  Due to the unusual nature of this exposure, consult a medical toxicologist or a regional poison center/ ministry of health for any patient with systemic symptoms, severe exposure, or in whom the diagnosis is unclear. Patients with severe burns will also need burn specialist consultation.

- OBSERVATION CRITERIA/ORAL
  In some cases of oral ingestion, early gastrointestinal symptoms may resolve after a few hours.
  - A relatively asymptomatic period may follow before more severe toxicity becomes apparent.
  - Early improvement should not be interpreted as meaning that serious exposure has not occurred.
  - Patients with phosphorus exposure should be monitored during the first 48 hours after exposure with frequent laboratory checks.
  - Patients that remain asymptomatic after this monitoring period can be discharged.

- Dermal/ inhalational
- Depending on the extent of burns/ debridement requirment/

# CAUTION

- Phosphorus absorption is enhanced when dissolved in alcohol, digestible fats, olive oil, or mineral oil.
- These agents are contraindicated in the management of oral or dermal phosphorus exposure.

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