Directed Energy: Medical Effects of Radio Frequency Exposure (Microwave & Millimeter Wave) – A Literature Review

Bruce A. Wright, PhD
Eric Powell, Capt, USAF, BSC
William W. Dodson III, MD, MPH, FACPM

January 2013

Air Force Research Laboratory
711th Human Performance Wing
School of Aerospace Medicine
Aeromedical Research Department
2510 Fifth St.
Wright-Patterson AFB, OH 45433-7913
NOTICE AND SIGNATURE PAGE

Using Government drawings, specifications, or other data included in this document for any purpose other than Government procurement does not in any way obligate the U.S. Government. The fact that the Government formulated or supplied the drawings, specifications, or other data does not license the holder or any other person or corporation or convey any rights or permission to manufacture, use, or sell any patented invention that may relate to them.

Qualified requestors may obtain copies of this report from the Defense Technical Information Center (DTIC) (http://www.dtic.mil).

AFRL-SA-WP-SR-2013-0004 HAS BEEN REVIEWED AND IS APPROVED FOR PUBLICATION IN ACCORDANCE WITH ASSIGNED DISTRIBUTION STATEMENT.

//SIGNATURE//       //SIGNATURE//

DR. WILLIAM W. DODSON III          DR. RODGER D. VANDERBEEK
Chief, Aircrew Select & Perform Res Chair, Aeromedical Research Dept

This report is published in the interest of scientific and technical information exchange, and its publication does not constitute the Government’s approval or disapproval of its ideas or findings.
**REPORT DOCUMENTATION PAGE**

Public reporting burden for this collection of information is estimated to average 1 hour per response, including the time for reviewing instructions, searching existing data sources, gathering and maintaining the data needed, and completing and reviewing this collection of information. Send comments regarding this burden estimate or any other aspect of this collection of information, including suggestions for reducing this burden to Department of Defense, Washington Headquarters Services, Directorate for Information Operations and Reports (0704-0188), 1215 Jefferson Davis Highway, Suite 1204, Arlington, VA 22202-4302. Respondents should be aware that notwithstanding any other provision of law, no person shall be subject to any penalty for failing to comply with a collection of information if it does not display a currently valid OMB control number. PLEASE DO NOT RETURN YOUR FORM TO THE ABOVE ADDRESS.

<table>
<thead>
<tr>
<th>1. REPORT DATE (DD-MM-YYYY)</th>
<th>14 Jan 2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. REPORT TYPE</td>
<td>Special Report</td>
</tr>
<tr>
<td>3. DATES COVERED (From – To)</td>
<td>Jan 2011 – Mar 2012</td>
</tr>
<tr>
<td>4. TITLE AND SUBTITLE</td>
<td>Directed Energy: Medical Effects of Radio Frequency Exposure (Microwave &amp; Millimeter Wave) – A Literature Review</td>
</tr>
<tr>
<td>5a. CONTRACT NUMBER</td>
<td></td>
</tr>
<tr>
<td>5b. GRANT NUMBER</td>
<td></td>
</tr>
<tr>
<td>5c. PROGRAM ELEMENT NUMBER</td>
<td></td>
</tr>
<tr>
<td>5d. PROJECT NUMBER</td>
<td></td>
</tr>
<tr>
<td>5e. TASK NUMBER</td>
<td></td>
</tr>
<tr>
<td>5f. WORK UNIT NUMBER</td>
<td></td>
</tr>
<tr>
<td>6. AUTHOR(S)</td>
<td>Bruce A. Wright, Eric Powell, William W. Dodson III</td>
</tr>
<tr>
<td>7. PERFORMING ORGANIZATION NAME(S) AND ADDRESS(ES)</td>
<td>USAF School of Aerospace Medicine Aeromedical Research Dept/FHC 2510 Fifth St. Wright-Patterson AFB, OH 45433-7913</td>
</tr>
<tr>
<td>8. PERFORMING ORGANIZATION REPORT NUMBER</td>
<td>AFRL-SA WP-SR-2013-0004</td>
</tr>
<tr>
<td>9. SPONSORING / MONITORING AGENCY NAME(S) AND ADDRESS(ES)</td>
<td></td>
</tr>
<tr>
<td>10. SPONSORING/MONITOR’S ACRONYM(S)</td>
<td></td>
</tr>
<tr>
<td>11. SPONSOR/MONITOR’S REPORT NUMBER(S)</td>
<td></td>
</tr>
<tr>
<td>12. DISTRIBUTION / AVAILABILITY STATEMENT</td>
<td>Distribution A: Approved for public release; distribution is unlimited. Case Number: 88ABW-2013-1908, 22 Apr 2013</td>
</tr>
<tr>
<td>13. SUPPLEMENTARY NOTES</td>
<td></td>
</tr>
<tr>
<td>14. ABSTRACT</td>
<td>This literature review provides a relatively compact summary of research efforts on diagnosing, managing, and treating injuries caused by radio frequency (RF) radiation exposure. We examined standards, reference documents, and peer-reviewed research that have been published from 2000 to present. The majority of these documents and articles were primarily focused on describing cellular effects and dose-response relationships; very few articles address the medical implications of RF exposure. Furthermore, most of these articles appear in specialized journals, which are not commonly found in medical libraries, or in technical reports with very limited distribution. As a result, there is a wide disparity in what is known about directed energy in the laboratory and what is known by clinicians. The intent of this paper is to help transition the body of knowledge from the laboratories to the clinicians. To that end, the adverse biological effects resulting from RF exposure are due to temperature elevation. The increase in temperature can be severe enough to cause localized burning of tissue or elevation of body temperature to dangerous levels. The major difference between RF-induced injuries and ordinary burns is the location of the damage. A unique characteristic of RF energy is its ability to penetrate deeper into the body and heat internal structures, such as muscles and organs, without elevating skin temperature. Investigating the therapeutic potential of pharmacologic agents and exploring new management and treatment options for RF-induced burn injuries are recommended.</td>
</tr>
<tr>
<td>15. SUBJECT TERMS</td>
<td>Radio frequency, directed energy, internal burns, RF radiation exposure</td>
</tr>
<tr>
<td>16. SECURITY CLASSIFICATION OF:</td>
<td></td>
</tr>
<tr>
<td>a. REPORT</td>
<td>U</td>
</tr>
<tr>
<td>b. ABSTRACT</td>
<td>U</td>
</tr>
<tr>
<td>c. THIS PAGE</td>
<td>U</td>
</tr>
<tr>
<td>17. LIMITATION OF ABSTRACT</td>
<td>SAR</td>
</tr>
<tr>
<td>18. NUMBER OF PAGES</td>
<td>19</td>
</tr>
<tr>
<td>19a. NAME OF RESPONSIBLE PERSON</td>
<td>Dr. Bruce A. Wright</td>
</tr>
<tr>
<td>19b. TELEPHONE NUMBER (include area code)</td>
<td></td>
</tr>
</tbody>
</table>

Standard Form 298 (Rev. 8-98)
Prescribed by ANSI Std. Z39.18
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>SUMMARY</td>
<td>1</td>
</tr>
<tr>
<td>RADIO FREQUENCY CLASSIFICATION</td>
<td>1</td>
</tr>
<tr>
<td>LOW-LEVEL (PERMISSIBLE) RF EXPOSURES</td>
<td>2</td>
</tr>
<tr>
<td>MILITARY APPLICATIONS OF RF</td>
<td>3</td>
</tr>
<tr>
<td>OVEREXPOSURE OF EYES, SKIN, AND INTERNAL TISSUES TO RF</td>
<td>4</td>
</tr>
<tr>
<td>OCULAR DAMAGE</td>
<td>5</td>
</tr>
<tr>
<td>CORE TEMPERATURE ELEVATION</td>
<td>5</td>
</tr>
<tr>
<td>PRESENT TREATMENT &amp; MANAGEMENT</td>
<td>6</td>
</tr>
<tr>
<td>TREATMENT ASPECTS OF HYPERTERMIA</td>
<td>6</td>
</tr>
<tr>
<td>TREATMENT ASPECTS OF BURNS (SKIN &amp; INTERNAL)</td>
<td>7</td>
</tr>
<tr>
<td>IMPLICATIONS FOR FUTURE MEDICAL RESEARCH</td>
<td>9</td>
</tr>
<tr>
<td>CONCLUSIONS</td>
<td>9</td>
</tr>
<tr>
<td>REFERENCES</td>
<td>9</td>
</tr>
<tr>
<td>LIST OF ABBREVIATIONS AND ACRONYMS</td>
<td>13</td>
</tr>
</tbody>
</table>
This page intentionally left blank.
SUMMARY

This literature review provides a relatively compact summary of research efforts on diagnosing, managing, and treating injuries caused by radio frequency (RF) radiation exposure. We examined standards, reference documents, and peer-reviewed research that have been published from 2000 to present. The majority of these documents and articles were primarily focused on describing cellular effects and dose-response relationships; very few articles address the medical implications of RF exposure. Furthermore, most of these articles appear in specialized journals, which are not commonly found in medical libraries, or in technical reports with very limited distribution. As a result, there is a wide disparity in what is known about directed energy in the laboratory and what is known by clinicians.

The intent of this paper is to help transition the body of knowledge from the laboratories to the clinicians. To that end, the adverse biological effects resulting from RF exposure are due to temperature elevation. The increase in temperature can be severe enough to cause localized burning of tissue or elevation of body temperature to dangerous levels. The major difference between RF-induced injuries and ordinary burns is the location of the damage. A unique characteristic of RF energy is its ability to penetrate deeper into the body and heat internal structures, such as muscles and organs, without elevating skin temperature. Investigating the therapeutic potential of pharmacologic agents and exploring new management and treatment options for RF-induced burn injuries are recommended.

RADIO FREQUENCY CLASSIFICATION

In this paper, the term “directed energy” (DE) will be used to describe a broad range of radio frequency (RF) radiation that includes radio waves (kHz and MHz), microwaves and millimeter waves (MHz to GHz), THz radiation, and optical radiation from the infrared to the ultraviolet band of the electromagnetic spectrum (1). This energy is further divided in the literature into pulsed and continuous wave forms. These portions of the electromagnetic spectrum are non-ionizing and have very small amounts of photon energy (unlike X-rays and gamma rays), and even high-intensity non-ionizing radiation is much too weak to break molecular bonds. However, this non-ionizing energy can cause significant biologic damage if the power density is sufficiently high and duration of exposure is sufficiently long.

It should also be noted that this paper will not address other forms of DE such as electrical (e.g., TASER) or acoustics. In addition, since the actual power or exposure measurements for the DE would not be known to clinicians, these variables will not be addressed at this time. Furthermore, it is assumed that clinicians would only be presented with a patient’s reported symptoms of an exposure. Additionally, the medical implication of laser light exposure (infrared to ultraviolet) on the eye has been covered in a separate paper by Dr. Parham-Bruce, so it will not be duplicated here (2).

Microwave energy that is absorbed by biological tissues produces a measurable increase in body temperature. The sensation of a temperature change can range from a sense of warmth to unbearable skin pain. This phenomenon has been studied extensively (3,4). Several factors determine whether warmth or pain is perceived, including the rate of temperature change, ambient temperature, skin temperature prior to exposure, and location of the stimulus. Skin temperature thresholds for sensations of thermal pain have been reported between 38 °C-46 °C (5). The location of the thermal change will vary according to the wavelength of the RF
exposure. For example, 94-GHz energy (millimeter wave) is deposited in the first one-third millimeter of the skin, while 2.45-GHz energy (microwave) penetrates 2-3 cm.

LOW-LEVEL (PERMISSIBLE) RF EXPOSURES

Guidelines for occupational exposures to RF radiation have been extensively covered by several international standards and guidelines. These guidelines include the Institute of Electrical and Electronics Engineers Standard C95.1-2005 (6); the International Commission on Non-Ionizing Radiation Protection (ICNIRP) *Exposure to High Frequency Electromagnetic Fields, Biological Effects and Health Consequences (100 kHz-300 GHz)*, ICNIRP 16/2009 (7); and the Air Force Research Laboratory’s *Radio Frequency Radiation Dosimetry Handbook*, 5th edition, July 2009 (8). The intent of these guidelines is to address the need for safety requirements for those routinely exposed to RF radiation. These documents provide basic restriction and recommendations for maximum permissible exposure thresholds. Furthermore, these safety-oriented resources provide excellent summaries of the scientific evidence on dosimetry, biological effects, and health consequences following the exposure to low-level RF radiation.

Scientific evidence supporting the general safety of low-level RF exposures cited by these standards and guides is overwhelming. Notable findings cited by the standards include:

- Low-level exposure to millimeter wave radiation does not alter cell viability, gene expression, or protein conformation (9).
- Consistent, strong associations were not found for RF energy exposure and adverse health events (10).
- No significant association between occupational exposure to radio frequency or microwave electromagnetic fields and brain tumors was found (11).
- There is no reproducible scientifically valid experimental basis for the claims about a linkage between (RF) exposures and the initiation, promotion, or co-promotion of cancer (12).
- Millimeter wave exposure did not promote or co-promote tumorigenesis in a well-established animal model of skin carcinogenesis (13).
- Consistent, strong associations were not found for RF exposures and malignancies, including brain cancer, leukemia, angiosarcoma, and ocular, skin, testicular, breast, salivary gland, and other cancers (14).
- Exposure to a pathogen typically produces pathogen-encoded markers in the host. Sufficient contact with a toxic agent and uptake by the host generally permit the detection of trace levels in the body as well. Similarly, ionizing radiation at sufficient dosage will induce permanent changes in host tissues that are detectable at the molecular level. In contrast, however, an individual exposed to DE does not have a toxin or pathogen-specific marker in the body or does not exhibit chemical changes in tissue (1).
- At present, no radio wave-induced molecular markers have been identified. However, DE-induced cellular changes at the molecular level are expected to be consistent with the hyperthermic responses observed in earlier experiments using physiological endpoints (1).
- There are no foreseeable long-term or delayed direct effects that could arise from use of the millimeter wave beam. Also, thermally induced bioeffects are not cumulative. That is, if a single exposure at a given level doesn’t cause an adverse effect, multiple exposures at
that level will not cause an adverse effect. Therefore, if an individual is subjected to a repeated exposure to RF radiation on another day, there is no greater risk of injury than on the first day. In fact, experience has shown that individuals once exposed become more knowledgeable and tend to remove themselves from the beam more rapidly. This results in a lesser risk of injury from the second exposure (15).

A complicating factor in evaluating the body of literature related to DE biological effects is summed up in a statement found in a 2003 literature review that stated: “Although extensive, much of the peer reviewed literature reporting bioeffects of EM [electromagnetic] energy is of poor quality. Often experiments are plagued by artifacts, many of which are the result of deficiencies in microwave engineering. In many cases reported findings cannot be replicated. Even if valid, papers sometimes do not present sufficient quantitative information for use in setting standards” (16). This conclusion is one of the main reasons why RF bioeffects research is so misunderstood by both the public at large and the medical community. One must have an understanding of both the physics of radio frequency generation and propagation and biology to properly understand and interpret this literature.

Finally, an even less encouraging observation came from a December 2007 report by the Defense Science Board Task Force on Directed Energy Weapons. They noted: “Numerous organizations have attempted to create HPM [high power microwave] effects databases that can be used as a ‘universal HPM effects predictor.’ All of those efforts have failed to produce a true predictive capability for reasons varying from seemingly slight differences in experimental conditions to the inability to share data between organizations” (17). A review of the literature and applicable RF radiation safety standards makes it clear that safety limits are very conservative. Furthermore, the adverse effects of RF energy are due to tissue heating during exposure.

MILITARY APPLICATIONS OF RF

While high-power electronic communication devices, sensors (e.g., radar), and other electronic warfare devices have existed since the World War II era, the primary target for electronic warfare attacks has been communication equipment and other electronic technologies as opposed to human targets. DE weapons have been postulated since the 1960s. While high energy lasers and HPM have the theoretical potential to deliver enough power to destroy targets of interest (counter-material) at significant ranges, few of these weapons have made it past the early phases of development. The few counter-personnel DE weapons that have left the laboratory include “low power” lasers, which are intended to temporarily disrupt vision. Another early system is the active denial system, which is a millimeter wave device designed to heat the skin to intolerable but nondamaging thermal levels. Furthermore, “lower-power lasers have increasingly been deployed on the battlefield, particularly for sensing, ranging, and targeting. Directed Infrared Countermeasure (DIRCM) lasers for blinding guidance systems of surface-to-air and air-to-air have been developed, and airborne counter-MANPAD systems have been fielded” (18).

Although the technology for these low-power DE weapons is mature, “the military is still reluctant to employ weapons that may be perceived as inhumane” (18). Despite our reluctance to deploy such weapons, other nations have explored the potential for advanced, high-power DE weapons systems. This includes credible reports of an active Chinese DE research program,
Russian supremacy in global fiber laser technology, and a German company marketing a counter-electronics suitcase bomb (19).

To achieve a true counter-material weapons effect, DE sources will likely need to utilize much greater power levels than are permissible under any U.S. or international standard. Additionally, there are many RF devices, including radar, that have already been fielded by conventional military forces around the world. The biological consequences of those overexposures are the primary topic of the next section in this paper.

OVEREXPOSURE OF EYES, SKIN, AND INTERNAL TISSUES TO RF

Mankind has experienced substantial exposure to RF energies over the last century without significant signs of biological hazard (20). For example, millions of people have been exposed to diathermy treatments with power up to 125 W. The previously mentioned safety guidelines from the Institute of Electrical and Electronics Engineers, ICNIRP, and the Air Force Research Laboratory cite numerous articles documenting the bioeffects of both RF and laser energy on cells, tissues, and a variety of animal models. According to all of those sources, the specific bioeffects produced by DE vary by the wavelength, intensity of the energy, duration of exposure, and type of tissue impacted and were summarized in Ziskin (2009) (15). As noted previously, electromagnetic waves at frequencies lower than that of millimeter waves penetrate deeply into the body and can cause heating of the entire body. However, the penetration of millimeter waves is limited to just the outer layer of the skin, and heating, unless prolonged, remains very superficial.

Most of our experience with overexposure to RF radiation is from personnel who inadvertently find themselves within the path of a radar beam or workers who ignore warning signs and enter restricted areas. As a result of those experiences and laboratory research, we understand the main effect of RF radiation on the body, whether from short pulses or continuous wave irradiation, is thermal heating. Exposures to short (15- to 25-ms) pulses of high-power microwave energy were observed to cause brain temperature to increase by up to 4 °C in mice. Subjects became “hypokinetic,” although they began to recover within 5 minutes. Rats exposed to longer pulses (50-360 ms) experienced an increase in brain temperature of up to 8 °C, and they exhibited seizures for approximately 1 minute followed by unconsciousness lasting up to 5 minutes (21).

Higher frequency RF exposures from GHz and THz sources are associated with skin-related thermogenic effects. Thermogenic effects remain superficial unless exposure is prolonged. The electroencephalogram results from an animal model demonstrated that the stress reaction induced by overexposure to millimeter waves was principally caused by thermal pain (22). If skin temperatures are elevated to 45 °C-55 °C, the individual may experience significant pain. However, subjects should not experience any damage or injury unless the exposure is prolonged. If the magnitude and duration of the exposure are higher, burns will develop. The threshold for a first-degree burn is 55 °C-60 °C and for a second-degree burn is 60 °C-65 °C; temperatures above 70 °C lead to third-degree burns. There are no differences in the burn threshold due to gender or race.
OCULAR DAMAGE

According to Ziskin (2009) (15), the areas of the body that are most vulnerable to RF radiation overexposure are the nervous system and the eyes. Damage to the eyes is caused by RF energy elevating the temperature of ocular tissue. There is no blood flow surrounding the lens, and heat dissipation for this structure is greatly diminished. Thermal buildup in the lens can lead to cell damage and cataract formation. The cornea is also vulnerable, but damage to the cornea usually heals. The only credible ophthalmologic effects of RF overexposure are those due to thermal mechanisms. Therefore, the pathology to look for would be burns of the eyelids, lenticular cataracts, retinal burns, and other evidence of burns in the surrounding tissues. No unique pathologic changes occur only from RF overexposure and not from other causes.

Cataracts were produced in animals when RF induced a temperature elevation to 41 °C for a period of 30 minutes. The thresholds for cataract formation in humans are believed to be higher and would be expected to produce unacceptable pain and damage to other parts of the eye and face prior to cataract formation (15). An important distinction with millimeter wave RF energy is the lack of penetration into the eye, so the lens, retina, and other structures are not affected.

CORE TEMPERATURE ELEVATION

Overexposure to RF radiation typically produces a thermal or pain sensation in the exposed area of the body. The resulting sensations will progress from a sense of warmth to overt pain. If the exposure is great enough, most subjects will attempt to move away from the beam. Additionally, metal objects on, near, or even inside the body are known to reflect and even concentrate RF fields. This concentrated radiation can possibly lead to localized sensations of extreme heat and significant burn trauma. Since the source of the radiation may not be visible, difficulty in evading the beam may make matters worse for the target subject.

Central nervous system function deteriorates at temperatures above 42 °C to 43 °C and convulsions may occur. At this temperature level, proteins begin to denature and cells may be damaged. Thermoregulatory responses of sweating and vasodilation cease at approximately 43 °C, after which body temperatures may rise very rapidly if external cooling is not imposed (23).

Humans have a greater thermoregulatory capacity in comparison to certain animal models. Researchers have observed behavioral signs of thermal stress in animal models after microwave exposure raised core body temperatures by as little as 1 °C (24). Although very efficient, human physiology’s heat stress countermeasures have their limits. When this limit is reached “heat stroke” may occur. Heat stroke is a set of symptoms that are produced by excessive body heating. The impact of such heat is determined not only by the absolute level of increased body temperature but also by the duration of the exposure to the heat source, ambient temperature, and humidity. Disturbances of the central nervous system (CNS) are always present in heat stroke, and the level of consciousness is often depressed (e.g., coma, sleep, or delirium) (23).

Substances such as pharmaceutical drugs can also impair heat tolerance potential (7). For example, drugs such as barbiturates or phenothiazines depress reflex regulation of body temperature, while anticholinergic drugs suppress sweating and vasodilation. Therefore, these
drugs could impact an individual’s ability to cope with a DE overexposure by diminishing CNS activity, cardiovascular reserve, or body hydration.

PRESENT TREATMENT & MANAGEMENT

The role of the clinician in assessing and treating an RF overexposure begins with an appropriate history and physical examination. Symptoms of overexposure range from anxiety; to warmth; to fatigue, malaise, headache, nausea, and vomiting; to localized burns and pain; to heat stroke and even death. Additionally, organ damage may not be detectable for several hours or several days after an overexposure.

Physical examination should include an ophthalmologic exam. The skin should be examined for areas of erythema and surface burns in the vicinity of metallic objects. An X-ray study may be helpful if the patient has any internally implanted metal objects. A neurologic exam should be performed to evaluate any paresthesias, paralysis, or possible injury to the CNS. Laboratory tests should be performed in accordance with the likelihood and the severity of the overexposure. If there are any cardiac symptoms present, an electrocardiogram should be performed. If there is suspicion that internal organ damage has occurred, appropriate serum enzyme levels should also be obtained. If there are significant neurologic symptoms, it may be helpful to obtain an electroencephalogram and/or a magnetic resonance imaging (MRI) examination of the involved region of the nervous system (15).

TREATMENT ASPECTS OF HYPERThERMIA

Certain weapons that cause heating of a targeted body may cause the body core temperature to rise. Hyperthermia of a body becomes a significant issue when it causes heat stroke. Progressively elevated core temperatures will cause mental status changes, which will progress to coma then death unless intervention occurs. One should not wait for the possible cessation of sweating to intervene in the face of progressive mental status changes. Sweating often stops in hyperthermia because the brain loses thermoregulatory function by the time its own temperature reaches 41 °C. In these cases, the cessation of sweating is not due to dehydration; in fact, hyperthermia often occurs in patients who happen to be normovolemic or even hypervolemic.

Medical responders must be careful to not misdiagnose hyperthermia as dehydration. The aggressive infusion of fluids into patients who are not hypovolemic has resulted in deaths due to cerebral edema. The initial and primary life saving intervention in hyperthermia is to cool the person’s body down to no more than 38.8 °C. Skin and ear canal thermometers are not as accurate as oral and rectal versions for following core temperatures (25).The following are interventions:

- Shade from sunlight
- Circulate air over them either manually or via fans while periodically wetting them for evaporative cooling
- Apply ice packs to armpits, groin, neck, and back (do not place ice directly against the skin due to danger of frostbite)
- Partially immerse in cold water (with ice if available), ensuring that mouth and nose are clear of the water
Rhabdomyolysis often occurs in muscle tissue due to hyperthermia. Creatine kinase level can be done to track this. Renal failure is a risk due to rising myoglobin levels. Intravenous (IV) fluids should maintain a urine output of 3 mL/kg/h. Alkalinization of the urine to a pH of 7.5 to 8.0 is recommended. Mannitol can be used to increase urinary output toward the goal above, if needed (26).

To prevent or treat renal failure, multiple variations of hemodialysis approaches can be used including cold hemodialysis, cold continuous hemodiafiltration (27), and venous-venous hemofiltration (28).

As a patient’s brain temperature increases, not only does it cease to be able to control thermoregulatory functions throughout the body, but it ceases to regulate multiple organ functions, which can lead to death. An example would be failure of the brain’s respiratory center, resulting in respiratory arrest, then death. That said, there have been research initiatives focusing on cooling the brain itself as rapidly as possible including hypothermic retrograde jugular vein flush with saline at 4 °C (29).

Disseminated intravascular coagulation and inflammatory responses also can occur during heat stroke. These two responses can lead to organ failure then death; activated protein C is thought to counter both of these responses and is another area of heat stroke research (30).

TREATMENT ASPECTS OF BURNS (SKIN & INTERNAL)

Heat from any source including RF that is sufficiently above a human tissue’s normal temperature can cause injury to organ tissue. The longer the duration of heating at the given supranormal temperature, the greater the damage. The injury effects are similar to inflammatory and traumatic injury to that organ up to the point where the heating causes a burn significant enough to cause immediate necrosis of cells. An effect not seen with blunt trauma or inflammation, unique to a burn of third or more degree intensity and internal burns, is potential cauterity of the tissue and local blood vessels, with a beneficial effect of reducing hemorrhage and/or extravasations of other problematic materials such as bile, etc. Tetanus shots (usually a booster) are given to patients with significant skin burns; however, this might not be indicated in a patient with a strictly internal burn only (31).

An organ will continue to allow viability of a person until damage reduces its function below a critical level. Medical interventions can sometimes mitigate the loss of function, allowing the individual to survive. The medical intervention may be able to be used indefinitely, or until the organ has regained function sufficiently for the intervention to be halted. For example, when lung function is compromised due to damage (trauma, infection, or heat), a ventilator is used. Once the lungs recover, ventilator use is halted.

The approach to detecting organ injury is the same whether the cellular injury and/or cellular death is from inflammation due to heating, infection, ischemia, etc. So, in general, the physician would order the same tests as he/she would for detecting pathology from the other etiologies (e.g., heart – cardiac enzymes, electrocardiogram; lungs – computed tomography, arterial blood gases; liver/pancreas – enzymes, MRI; brain – MRI, computed tomography, etc.).

Hyperbaric oxygen treatment (HBOT) is an option for certain burn situations. Indeed, “thermal burn” is one of the 15 accepted indications for HBOT as noted in the Undersea and Hyperbaric Medical Society indications manual (32), which is accepted by the Department of Defense, Veterans Administration, Centers for Medicare & Medicaid Services, and a majority of leading nonfederal health insurers. HBOT has been shown to improve predicted patient courses
particularly in facial burns (improved cosmesis potential) and hand burns (improved function potential). For maximal beneficial effect, up to three HBOT sessions should occur in the first 24 hours post-burn. Although there is no mention of HBOT being used for internal RF burns, one would postulate that the same beneficial effects would occur including enhanced angiogenesis and fibroblast activity (allowing faster healing, increased survivability, and perhaps a more complete recovery).

Skin burn patients who have more than 15%-20% total body surface area (TBSA) burns should have fluid resuscitation within 2 hours to avoid shock. It is recommended that the Consensus Formula, formerly known as the Parkland Formula, be used: 4 mL/kg/TBSA% of lactated Ringer’s solution, half given by 8 hours post-burn, half given during the next 16 hours. Urine output goal is 0.5 mL/kg/h, 0.5-1.0 mL/kg/h in children < 30 kg. If the pulse pressure becomes narrow, or the pulse increases above 120 in an adult, shock may be approaching. Because of limb edema, arterial lines are preferred over cuffs for blood pressure monitoring. Nasogastric tubes should be used for nutrition augmentation. If not possible, then parenteral nutrition augmentation should be done. Glucose should be kept at 80-110 mg/dL (insulin can be used for this). Patient environments should be kept at 85 °F or higher. For >30% TBSA burn patients, bladder pressure monitoring should occur. Observation should be ongoing for abdominal compartment syndrome and for extremity compartment syndrome. Deep vein thrombosis prevention efforts are indicated (including anti-deep-vein-thrombosis medication). Gastrointestinal stress ulcer prevention medication is indicated as well. For patients with large TBSA burns, androgenic steroids such as oxandrolone can be given as well as propranolol, with a goal of 20% pulse rate reduction. Central venous catheter sites should be changed every 3 days. Patients should be washed via irrigation daily with chlorhexidine and warm tap water. Silver sulfadiazine can be used as a topical agent (alternate topical must be used if sulfa allergy or patient is newborn, pregnant, or nursing). Full thickness burns can be grafted; partial thickness burns can be covered with silver-containing dressings (blisters >6 mm should be debrided). IV opioids can be used for pain control, and IV ketamine can be used for dressing changes. A burn center should be contacted in every situation for both current treatment guidance as well as guidance regarding potential transfer (33).

Patients less than 16 years old are at increased risk of encephalopathy and death from hyponatremia due to their larger brain vs. skull ratio. Methodical body weights and serum electrolytes should be followed. These patients can develop initial hyponatremia symptoms and signs at higher sodium levels than adults. Hyperpyrexia and emesis are danger signs that may or may not precede seizures, coma, and death (34).

Some burn centers believe that patients do better if they are only kept transfused to a hematocrit of 21%. If a patient’s hematocrit is 34% or below, error from a single channel glucometer will be >5%. Maintaining the serum glucose in the 80-110 mg/dL range has been shown to have a significant beneficial effect upon burn patient outcomes. The results from newer 4-channel glucometers are not affected by a low hematocrit and, therefore, are preferred in these situations. If a single-channel glucometer is used on a patient with a low hematocrit, one should use a hematocrit correction factor specific to that glucometer to determine the actual serum glucose (35).

The hypermetabolic response that begins several days after a severe burn of approximately 40% or more TBSA may last up to 2 years. A major negative effect of this is muscle protein degradation. In addition to what is used in adult patients, such as Oxandrolone, recombinant human growth hormone is now often used with pediatric burn patients (36).
Additional hypermetabolic mitigation agents that are being evaluated at this time include fenofibrate and ketoconazole (36) as well as metformin, glucagon-like peptide-1, and peroxisome proliferator-activated receptor agonists (37).

Infection contributes to much morbidity and mortality in burn patients. Patients with significant burns release cytokines that impair T-cell and neutrophil function. In addition, the systemic inflammatory response in these patients reduces end organ perfusion. Also, their inflammatory response renders unreliable the usual signs of infection: fever and white blood cell count elevations. Systemic antibiotics should not be routinely used as infection prophylaxis. Indications for commencing antibiotics include change in wound odor and/or exudate, a swab culture positive for a pathogen, and a deterioration in patient condition without an obvious cause other than probable infection (38).

Recent studies with stem cells show a potential reduction in inflammatory response. This should result in improved T-cell and neutrophil function, improved end organ perfusion, and less morbidity/mortality in burn patients. This is an active area of research at this time (39).

**IMPLICATIONS FOR FUTURE MEDICAL RESEARCH**

Research into the discovery of specific biomarkers indicative of RF overexposure should continue to be explored. As DE technology advances and power levels are increased, unique bioeffects may yet be encountered. These bioeffects will need to be further studied while focusing on their implications for clinical management. While clinical management of thermal injury is well known, new treatments and diagnostic technologies specific to the treatment of RF-induced injuries should be further investigated.

One specific area for investigations should be the development of personal dosimeters sensitive to the RF spectrum presented in this manuscript. These RF dosimeters would provide clinicians objective data to help them determine whether an RF exposure incident was medically relevant to the exposed subject. Since both duration and location of exposure are important in determining injury potential, the dosimeter should incorporate both time and spatial averaging. A second area for investigation is the use of HBOT in the treatment of internal RF burns.

**CONCLUSIONS**

The medical consequences resulting from RF radiation overexposure depend on the severity of the temperature increase, what tissues were involved, and whether those tissues can heal or regenerate. In general, adverse biological effects resulting from RF exposure are due to temperature elevation. The increase in temperature can be severe enough to cause localized burning of tissue or elevation of body temperature to dangerous levels. The major difference between RF injuries and ordinary burns is the location of the damage. Indeed, a unique characteristic of RF energy is its frequency-dependent ability to penetrate deeper into the body and heat internal structures, such as muscles and organs, without elevating skin temperature.

**REFERENCES**

6. Institute of Electrical and Electronics Engineers. IEEE standard for safety levels with respect to human exposure to radio frequency electromagnetic fields, 3 kHz to 300 GHz. C95.1-2005. New York: Institute of Electrical and Electronics Engineers; 2005.


### LIST OF ABBREVIATIONS AND ACRONYMS

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
<td>central nervous system</td>
</tr>
<tr>
<td>DE</td>
<td>directed energy</td>
</tr>
<tr>
<td>HBOT</td>
<td>hypobaric oxygen treatment</td>
</tr>
<tr>
<td>HPM</td>
<td>high power microwave</td>
</tr>
<tr>
<td>ICNIRP</td>
<td>International Commission on Non-Ionizing Radiation Protection</td>
</tr>
<tr>
<td>IV</td>
<td>intravenous</td>
</tr>
<tr>
<td>MRI</td>
<td>magnetic resonance imaging</td>
</tr>
<tr>
<td>RF</td>
<td>radio frequency</td>
</tr>
<tr>
<td>TBSA</td>
<td>total body surface area</td>
</tr>
</tbody>
</table>