5. Engle, R. L.: Sarcoid and sarcoid-like granulomas; a study of twenty-seven postmortem examinations, Am. J. Path., 29:53, Jan.-Feb. 1953.

6. Gleckler, W. J.: Hypercalcemia and renal insufficiency due to sarcoidosis; treatment with cortisone, Ann. Int. Med., 44:174, Jan. 1956.

7. Henneman, P. H., Carroll, E. L., and Dempsey, E. F.: The mechanism responsible for hypercalcemia in sarcoid, J. Clin. Invest., 33:941, June 1954.

8. Klatskin, G., and Gordon, M.: Renal complications of sarcoidosis and their relationship to hypercalcemia, Am. J. Med., 15:484, Oct. 1953.

9. Klinefelter, H. F., Jr., and Salley, S. M.: Sarcoidosis simulating glomerulonephritis, Bull. Johns Hopkins Hosp., 79:333, Nov. 1946.

10. Longcope, W. T., and Freiman, D. G.: Study of sarcoidosis based on combined investigation of 160 cases including 30 autopsies from Johns Hopkins Hospital and Massachusetts General Hospital, Medicine, 31:1, Feb. 1952.

11. Rosenthal, S. R.: Pathological and experimental studies of Boeck's sarcoid; report of case with panarteritis, periarteritis, terminal hypertension and uremia and the reproduction of sarcoid-like lesion in guinea pigs, Am. Rev. Tuberc., 60:236, Aug. 1949.

12. Salmon, H. W., and Meynell, G. G.: Primary hyperparathyroidism (adenoma) simulating sarcoidosis; report of case, Brit. M. J., 2:1440, Dec. 15, 1951.

13. Scholz, D. A., and Keating, F. R., Jr.: Renal insufficiency, renal calculi and nephrocalcinosis in sarcoidosis; report of eight cases, Am. J. Med., 21:75, July 1956.

14. Snapper, I.: On Boeck's sarcoids, Conn. M. J., 12:99, Feb. 1948.

15. Teilum, G.: Glomerular lesions of kidney in sarcoidosis (Boeck's sarcoid), Acta path. et microbiol. scandinav., 28:294, 1951.

Tissue Destruction and Death from Microwave Radiation (Radar)

JOHN T. McLAUGHLIN, M.D., Glendale

THE TERM *microwave* is popularly used to designate a certain range of waves in the radio frequency spectrum, including frequencies from approximately 1,000 mc. (megacycles) per second to 30,000 mc. per second or higher. Expressed in wavelength, this spectrum is from 30 cm. to 1 cm. or less. Microwaves are usually produced by means of specially constructed tubes such as the magnetron and klystron. *Radar* is a term coined for military apparatus using microwaves of this kind.

Previously reported work has emphasized the potential hazards of absorption of microwaves by body tissues. It has been reported that totally absorbed energy of 100 to 1,000 watts will cause intolerable temperature values.[7] The relationship of absorbed energy and energy flux is dependent upon the reflective coefficient of the surface of the human body.[8] There are many variables such as the radiant frequency, the amount of skin, the amount of subcutaneous fat and the temperature of such tissues. Investigation has not proceeded to the point at which it is possible to state tolerance levels in terms of radiant power, but currently 0.01 watt per square centimeter is the recommended tolerance level.

Penetration is to some extent a function of frequency, and one of the advantages of microwave diathermy is that deeper tissues can be heated more efficiently. Likewise, wavelengths of 30 to 50 cm. will give greater depth of penetration with more efficiency than 12 cm. radiation.

Cellular injury or death occurs when tissue temperature is maintained at 5°C. above the normal blood temperature. Tissue temperature even a few degrees above normal body temperature is dangerous. Irreversibility depends upon the duration of the hyperthermic episode. The higher the temperature, the shorter the time necessary to cause cell death. Tissues respond to heat denaturation with an aseptic inflammatory reaction and subsequently are prone to infection. Local temperatures of 64°C. will cause tissue necrosis and gangrene at the site. Such temperature elevations have been brought about in the hollow intestinal organs of animals and in an excised eye with 12.5 cm. microwave radiation at a power of 100 watts.

Not all areas of the body are equally well equipped with mechanisms for regulating their temperature by means of a change in flow of blood. The chambers of the eye and the contents of the hollow viscera, such as the gallbladder, urinary bladder and lumen of the gastrointestinal tract are relatively avascular and largely devoid of effective mechanisms for regulation of temperature. Temperature increase in living tissues and organisms during exposure to high frequency electromagnetic waves depends upon at least four factors: (1) Specific areas exposed and the efficiency of the heat eliminating mechanism; (2) intensity of radiation; (3) duration of the exposure; (4) specific frequency of the radiation. Hines and Randall[4] made a study of changes in temperature in various areas of rabbits during irradiation with electromagnetic waves (Table 1). It was noted, that whereas the visceral temperatures were elevated, the oral and rectal temperatures remained within normal limits. The elevation was particularly pronounced in the relatively avascular hollow viscera. In these experiments, when only the abdomen was irradiated, it appeared that death was preceded by a syndrome resembling that noted in burns and traumatic shock.

TABLE 1.—*Changes in Temperature in Various Areas of Rabbits During Irradiation with Electromagnetic Waves*[4]

Region	Times in Minutes: 1	2	3	10	20	30
	Temperature change in degrees C.					
Ileum	—4.2	....	14.4	29.5	38.5	42.9
Stomach	1.8	3.4	5.4	....	19.2	23.1
Gallbladder	....	0.1	0.3	1.8	4.0	6.3
Urinary bladder	1.3	2.1	3.0	5.6	....	9.7
Rectal	0.1	....	0.1	0.2	....	0.8
Oral	—0.2	....	0.2	—0.5	—0.9	—1.2

Submitted September 13, 1956.

Boyle and co-workers[1] expressed belief that the heat from microwaves is due mainly to the dipolarity of water—that in blood 80 per cent of the heat is the result of dipolar effect and 20 per cent of ionic conductance. In the penetration of water the energy delivered at a depth of 2.5 cm. was 10 per cent of the emitted energy. The penetration was better in living tissue because of its homogenicity. The temperature rise was greater in hollow viscera ordinarily having a liquid content than it was in other hollow viscera.

Imig and Hines[5] noted that in testicular degeneration due to heat, the greatest damage was to the mucosa and extended outward from the mucosal surface.

Oldendorf[6] used 12.5 cm. microwave radiation to destroy brain tissue selectively in rabbits without apparent evidence of a burn on the skin.

The following is a report of a case in which the patient died from tissue destruction caused by absorption of microwave energy.

CASE REPORT

A 42-year-old white man, while working, stood directly in the beam of a radar transmitter, within ten feet of the antenna. In a few seconds he had a sensation of heat in the abdomen. The heat became intolerable in less than a minute and he moved away from the antenna. Within 30 minutes he had acute abdominal pain and vomited. When medically examined, an hour after the exposure, he was in a state of mild shock. The blood pressure was 90/30 mm. of mercury and the radial pulse rate 72 with auricular fibrillation.

Upon abdominal examination generalized acute tenderness was noted, with decided muscle spasm and rebound tenderness. There were no peristaltic sounds. Leukocytes numbered 10,300 per cu. mm. —82 per cent neutrophils, 5 per cent eosinophils, 10 per cent lymphocytes and 3 per cent monocytes.

In an x-ray film of the abdomen, there was no evidence of free air under the diaphragm. A specimen of urine was unobtainable.

The patient denied any history of symptoms suggestive of gastric disease. He had had rheumatic endocarditis in childhood and had had a mitral commissurotomy one year previously. Since the cardiac operation the patient had been taking digitalis. Whether he had had fibrillating before the present episode was not known.

The patient was immediately admitted to hospital. Additional x-ray films did not show free air in the peritoneal cavity. The stomach was greatly distended and there was a small amount of gas in the colon, pooled in the cecum and ascending colon. The general appearance of the abdomen was considered consistent with acute peritonitis. X-ray films of the chest showed considerable cardiac enlargement with elevation of the left diaphragm.

Leukocytes numbered 15,700 per cu. mm.—90 per cent segmented forms, 5 per cent stab cells and 5 per cent lymphocytes. The urine showed traces of bile. Serum amylase was 170 units on one occasion (normal, less than 180) and 161 units on another. The direct serum bilirubin was 0.02 mg. per 100 cc. (normal 0.02 or less) and the total was 1.2 mg. per 100 cc. (normal less than 1 mg.).

Diagnostic possibilities considered were mesenteric embolus or thrombosis, or a perforated peptic ulcer. The patient was prepared with fluids and other supportive measures, and operation was done six hours after the onset of pain.

The peritoneum contained approximately 500 cc. of serosanguinous fluid, which proved to be sterile. The entire parietal and visceral peritoneum were dusky red and the portion of the small bowel that could be seen was beefy in color. All visible surfaces were covered with petechiae approximately 1 mm. in diameter. The appendix appeared gangrenous. It was removed. The patient did reasonably well after operation. The abdomen was continually decompressed and on the fourth day after operation, peristaltic action apparently having begun, fluids were given by mouth. On the fifth postoperative day, diarrhea and abdominal distention occurred. Chloramphenicol was substituted for the previously prescribed tetracycline but diarrhea persisted. Tests of stool specimens taken the fifth day and sixth postoperative day were negative for occult blood and E. coli grew on culture.

On the sixth postoperative day leukocytes numbered 17,000 per cu. mm. of blood—85 per cent segmented forms, 7 per cent stab cells and 5 per cent lymphocytes. The platelets numbered 156,000 per cu. mm. and it was impossible to get clot formation for clot retraction measurement. The serum potassium and chloride values were within normal limits.

Distention continued and a film of the abdomen on the eighth postoperative day showed evidence of bowel obstruction, probably in the right lower quadrant at the level of the terminal ileum. Despite continuous decompression with a Miller-Abbott tube, the condition was unchanged in another x-ray film 24 hours later.

On the tenth postoperative day evisceration of the abdominal wound occurred. The patient was in profound shock. Operation was done immediately. At a point in the jejunum 1.25 meters proximal to the ileocecal valve there was an oval perforation 4 cm. long with smooth edges in the long axis of the bowel. The portion of the bowel at the point of perforation lay in the left side of the abdomen at the level of the umbilicus. A six inch segment of bowel was resected and the abdomen was closed. The patient continued in shock and died in 24 hours.

Pathologist's Report on Surgical Specimens

The appendix was intact, 8 x 1.3 cm., with proximal stenosis. Distally the lumen was filled with pus and the surface was covered with pus. A culture of material taken from the surface at operation was sterile. Dense polymorphonuclear infiltration was

observed throughout the wall, which had been thinned by expansile pressure.

Multiple sections of the small intestine showed plastic peritonitis. Fibroblastic and capillary proliferation formed a moderately thick tissue on the surface, with a moderate mixture of polymorphonuclear leukocytes and a large component of small round cells. Mild inflammatory cell infiltrate was present throughout the muscularis, and in the submucosa there was severe edema with a scattering of inflammatory cells of a type similar to those in the peritoneal exudate. However, there was a greater prominence of plasma cells in this location. Pronounced enteritis was present, with an increase of plasma cells and small round cells between the orderly straight glands of the mucosa. The vessels contained no thrombi, and there was no evidence of vasculitis. In sections taken from the area of perforation a plastic reaction was noted around the edges with a fairly large component of polymorphonuclear leukocytes. It was not essentially different from the reaction elsewhere in the small intestine. No amebae and no areas of focal necrosis or tubercles or other specific inflammatory lesion were seen.

Pathologist's Report on Autopsy

At postmortem examination the abdominal organs were covered with a purulent exudate and the abdomen contained a moderate amount of turbid, yellow fluid.

A culture of a specimen of stool grew E. coli.

There was an extensive old, partly organized mural thrombus of the left atrium extending into the auricle.

The liver, which weighed 1,500 grams, showed fine nodular darkening but no passive congestion.

The spleen was twice normal size, weighing 300 gm. It contained two large pyramidal hemorrhagic infarcts of recent origin. The capsule was not thickened and there were no thrombi in the hilar vessels.

The stomach and duodenum were normal. The small intestine was dilated and the mucosa was edematous. 1.25 meters proximal to the cecum there was an intact side-to-side anastomosis with a 4 cm. lumen. Edema was noted throughout the small bowel. In the distal jejunum and ileum there was pronounced follicular hyperplasia of submucosal aggregates with prominent redness and central white spots. These lesions, which were 2 to 3 mm. in diameter, were present in every square cm. of the submucosa. There was no associated ulceration. The site of appendectomy was intact. Scattered patches of induration and exudate were noted on the mesentery and mesocolon. Upon dissection of the mesentery no thrombosis was observed. The adrenal glands were remarkably small, the combined weight being 3 gm. The cortices were thin and regular, the medulla autolysed.

Sections of the small intestine obtained at autopsy were examined microscopically and an inflammatory reaction identical to that previously noted in the surgical specimen—plastic peritonitic exudate and diffuse inflammation and edema throughout the wall, with prominent small round cell infiltrate of the mucosa. On the periphery was a thin crust of fibrin and cellular debris. The reddish spots observed in the gross specimen corresponded to areas of inflammatory cell infiltrate, mostly lymphocytes, and of telangiectasia. Again there was no evidence of amebiasis or tuberculosis or other specific inflammatory patterns.

In the ileum, the follicular hyperplasia was nonspecific and necrosis was not present. Sections of the colon showed a peritonitic exudate, but no noteworthy intrinsic inflammation.

In the spleen, hemorrhage in the pulp was noted in the areas of infarction. Otherwise it was normal. The lungs, liver, pancreas, adrenals, kidneys and brain were normal microscopically.

Diagnosis

I. Enteritis, type undetermined
   A. Recent ulceration and perforation of the jejunum
      1. Recent resection of perforated segment (and) appendectomy
      2. Subacute suppurative peritonitis.

II. Adrenal gland atrophy (3 gm.)

III. Rheumatic heart disease
   A. Mitral stenosis
      1. Mural thrombus in left atrium
      2. Old healed commissurotomy.

In this very perplexing case multiple sections were made of the small intestine particularly, in order to evaluate the sequence of events and the direction of spread of the inflammatory process. The most critical problem was whether or not the enteritis, which involved the entire wall of the intestine, was simply a neighborhood reaction from peritonitis. If that were the case, the perforation of the small intestine was unexplained. However, the very definite and diffuse mucosal inflammation of a small round cell type and the lack of dense continuity with the plastic peritoneal exudate strongly suggested that peritonitis was secondary to enteritis. There was definite hypoplasia or atrophy of the adrenal glands, but whether that was related to the cause of death was not apparent.

Pseudomembranous enterocolitis was precluded by the absence of blood and tissue in the stool, by the presence of E. coli and by the postmortem appearance of the colon.

The frequency and power factors of the microwave radiation to which the patient was exposed were unavailable because of security regulations. It was enough to cause a painful sensation of heat in the abdomen, however, and when heat can be felt, the tolerable level has been exceeded.[8] Although the power factor is not known it is known that the armed forces are using equipment emitting 2.5 megawatts peak power.[7]

It seems probable that the fluid contained in the small bowel received at least 10 per cent of the energy flux and that the temperature immediately was elevated high enough to denaturize the protein of the mucosal lining and initiate a severe enteritis. The whole reaction was one of inflammation, rather than infection.

The pathological findings are comparable to those brought about by Boysen[2] in experimental animals. Using radiation equipment emitting 5 to 500 watt power, he achieved pathological changes with whole body radiation. The jejunum and ileum were especially susceptible, showing hyperemia, hemorrhage and necrosis. Hyperemia of the spleen and hemorrhage into the myocardium were observed. Bloodless diarrhea ensued in each instance.

In the case here reported, the sudden onset, with sensation of unbearable heat, the cooked, hemorrhagic appearance of the small bowel and the pathological reports all point to the local absorption of heat in the umbilical and hypogastric regions with some generalized whole body radiation effect appearing in the myocardium, liver and spleen.

The hemorrhagic infarcts of the spleen were similar to those seen by the author in two other patients who were exposed to sufficient microwave radiation to cause pathological changes in the tissues.

1252 South Central Avenue, Glendale 4.

REFERENCES

1. Boyle, C., Cook, H. F., and Buchanan, T. J.: Effects of microwaves: Preliminary investigation, Brit. J. Phys. Med., 13:2-9, Jan. 1950.

2. Boysen, J. E.: Hyperthermic and pathologic effects of electromagnetic radiation (350 Mc), A.M.A. Arch. Indust. Hyg. and Occup. Med., 7:516, 1953.

3. Brody, S. J.: Military aspects of the biological effects of microwave radiation. I.R.E. Transactions on Medical Electronics, PGME, 4:8-9, Feb. 1956.

4. Hines, H. H., and Randall, J. E.: Possible industrial hazards in the use of microwave radiation, Elec. Eng., Oct. 1952.

5. Imig, C. J., Thomson, J. D., and Hines, H. H.: Testicular degeneration as a result of microwave irradiation, Proc. Soc. Exp. Biol. and Med., 69:382-386, 1948.

6. Oldendorf, W. H.: Focal neurological lesions produced by microwave irradiation, Proc. Soc. Exp. Biol. and Med., 72:432-434, Nov. 1949.

7. Salisbury, W. W., Clark, J. W., and Hines, H. H.: Exposure to microwaves, Electronics, 22:66-67, May 1949.

8. Schwan, H. P., and Piersol, G. M.: The absorption of electromagnetic energy in body tissues, Am. J. Phys. Med., 34:3, June 1955.