Rapid Gastric Emptying of an Oral Glucose Solution in Type 2 Diabetic Patients

William T. Phillips, Joyce G. Schwartz, and C. Alex McMahan

Departments of Radiology and Pathology, The University of Texas Health Science Center at San Antonio, San Antonio, Texas

Gastric emptying of a liquid glucose meal was measured with scintigraphic techniques in nine recently diagnosed Type 2 diabetic patients and nine sex- and age-matched nondiabetic control subjects. Seven of the nine Type 2 diabetic patients were receiving oral hypoglycemic therapy which was discontinued the evening prior to the study. The other two diabetic patients were taking no medication. The average gastric half-emptying time was 33.6 min (s.e.m. = 3.2) for the diabetic patients and 64.6 min (s.e.m. = 4.2) for the nondiabetic controls ($p = 0.0005$). These measurements indicate rapid gastric emptying in Type 2 diabetic patients which may contribute to worsening of glucose control in these patients.

J Nucl Med 1992; 33:1496–1500

Gastric emptying abnormalities (diabetic gastroparesis) occurring as a *late* manifestation of diabetes have been studied repeatedly (*1–8*) since Rundles (*1*) first proposed autonomic neuropathy as the cause of the gastrointestinal dysfunction. Most studies have demonstrated *slower* gastric emptying of solid and liquid meals in diabetic patients. Previous studies (*1–8*) of gastric emptying in diabetic patients have generally involved diabetics with gastrointestinal symptoms. Many studies have grouped patients with Type 1 and Type 2 diabetes into the same study. Most of the patients in these studies have had diabetes for a prolonged period of time. Gastric emptying studies performed on Type 2 diabetic patients have generally used meat or eggs for the solid component of the meal. The authors know of no studies that have adequately assessed the rate of gastric emptying of a single component carbohydrate solid or a glucose solution in nonsymptomatic Type 2 diabetic patients. In the current study, we used a scintigraphic technique to compare the gastric emptying rates of a liquid glucose meal in recently diagnosed nonsymptomatic Type 2 diabetic patients and sex- and age-matched control subjects.

Received Nov. 25, 1991; revision accepted Mar. 10, 1992.

For reprints contact: Dr. William T. Phillips, Department of Radiology, The University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Dr., San Antonio, TX 78284-7800.

PATIENTS AND METHODS

Patients

Nine recently diagnosed Type 2 diabetic subjects (diagnosed within 2 yr) and nine sex- and age-matched nondiabetic subjects underwent gastric emptying studies. The subjects (14 males, 4 females) ranged in age from 32 to 62 yr of age. Seven of the nine diabetic subjects were Hispanic; one, a non-Hispanic white, and one an Iranian. All nine nondiabetic subjects were non-Hispanic whites with normal fasting glucose values. The diabetic patients had been previously diagnosed as being diabetic using a 75-g oral glucose tolerance test with blood sampled fasting and at 2 hr according to current WHO criteria (*9*). No evidence of diabetic neuropathy or autonomic dysfunction was present in any of our diabetic patients. Although oral hypoglycemic medication had been prescribed previously for all of our diabetic patients, only seven of the nine took their medication on a routine basis. This medication was discontinued the evening before the study. None of our patients had a history of any recent surgical procedures and none were taking any other type of prescribed or over-the-counter medication with the exception of their oral hypoglycemic medication.

Gastric Emptying Studies

Gastric emptying studies utilizing a gamma camera (Scintronix USA Inc., Woburn, MA) were performed with a 0.62 mol/liter (50 g glucose in 450 ml water) glucose solution. The use of this glucose solution in gastric emptying has been previously studied by the authors (*10*). Each study was begun at approximately 7:30 a.m. and finished by 12:00 p.m. Approximately 200 μCi of 99 metastable technetium sulfur colloid (^{99m}Tc-SC, CIS-US, Bedford, MA) were added and mixed with the glucose solution. The subjects drank the glucose solution in its entirety in a 5-min span shortly after the ^{99m}Tc-SC had been added to the solution.

The subjects were then placed in a semi-reclining position (45° from horizontal) and the gamma camera was positioned anteriorly. Only anterior views were used in calculating the gastric emptying, since it has been shown (*11*) that the geometric means of the anterior and posterior projections, using liquid meals, were very similar to those of the anterior views alone. Data were collected continuously and summed at 60-sec intervals. Images were acquired during an interval of 120 min (e.g., if no solution remained in the stomach after a 90-min period, the gastric emptying study was terminated; plasma-glucose levels were measured for the full 120-min period).

The Scintronix gamma camera was used with a low-energy, all-purpose collimator at a 20% window setting centered at 140 keV. The camera was connected to a Medical Data Systems Computer (Ann Arbor, MI). Counts in the stomach region of

interest were calculated and drawn separately for each 60-sec image. After correcting for radioactive decay, the count rates were converted to a percentage of the maximum count rate recorded.

Plasma Glucose

Plasma-glucose samples were drawn at 15-min intervals beginning just prior to ingestion of the glucose solution and ending at 120 min. The patients' blood samples were collected in grey-top vacutainer tubes containing potassium oxalate and sodium fluoride (Becton Dickinson Vacutainer Systems, Rutherford, NJ). Glucose analysis was performed on a Paramax instrument (Baxter Healthcare Corp., Irvine, CA).

Statistical Methods

The data were analyzed using a paired t-test. The gastric half-emptying time for each patient was calculated by linear interpolation. The area under a curve was calculated using the trapezoid rule. If the gastric emptying study was not carried out for the full 120 min, the last observed value was used to compute the area under the curve from the last point to 120 min.

RESULTS

The gastric emptying pattern of the diabetic patients and the normal control subjects are shown in Figure 1. The half-emptying time (Fig. 2) was significantly shorter ($p = 0.0005$) for the diabetic patients (average = 33.6 min, s.e.m. = 3.2) than for the nondiabetic control subjects (average = 64.6 min, s.e.m. = 4.2). The area under the gastric emptying curve during the first hour, representing an overall time-weighted average, for the diabetic patients was 75% of the area under the curve for the nondiabetic control subjects ($p = 0.001$). The area under the curve during the second hour for the diabetic patients was 63% of the area under the curve for the nondiabetic control subjects ($p = 0.0001$). The half-emptying time and the area under the curve indicate substantially faster emptying for the diabetic patients. The largest separation of gastric emptying rates between diabetic patients and nondiabetic control subjects occurred at 75 min ($p = 0.003$) (Fig. 3).

The mean glucose concentrations were higher in diabetic patients than in nondiabetic control subjects each time blood was sampled (Fig. 4). The mean fasting plasma-glucose concentrations were significantly different between the diabetic patients [12.5 mmol/liter (s.e.m. = 1.6)] and nondiabetic control subjects [4.9 mmol/liter (s.e.m. = 0.2)] ($p = 0.001$). The mean glucose concentration during the first hour for the diabetic patients was 17.1 mmol/liter (s.e.m. = 1.7) and 7.1 mmol/liter (s.e.m. = 0.5) for the nondiabetic patients ($p = 0.0003$). The mean glucose

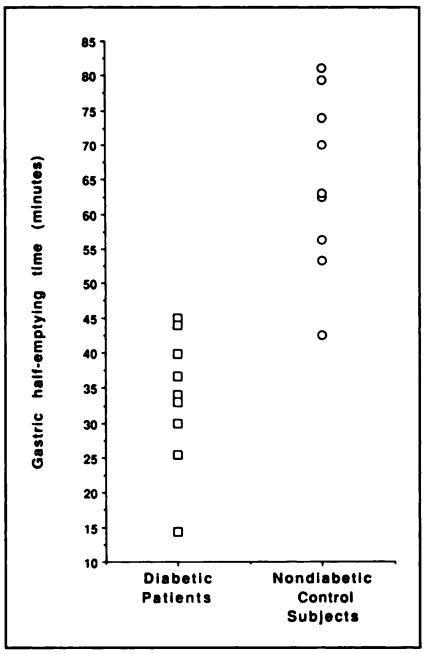

FIGURE 2. Gastric half emptying time for diabetic and nondiabetic patients.

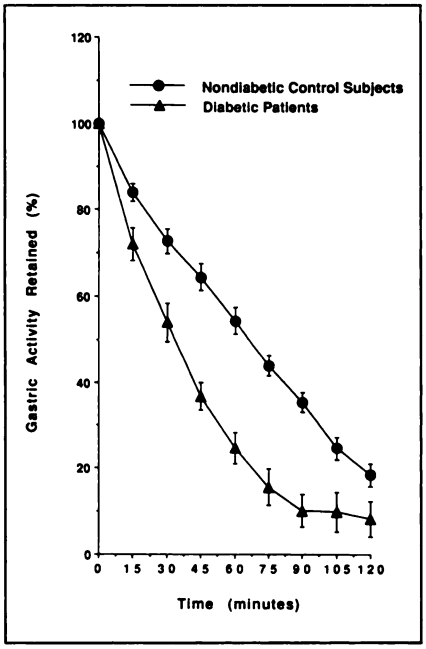

FIGURE 1. Gastric activity retained over a 2-hr period for diabetic patients and nondiabetic patients.

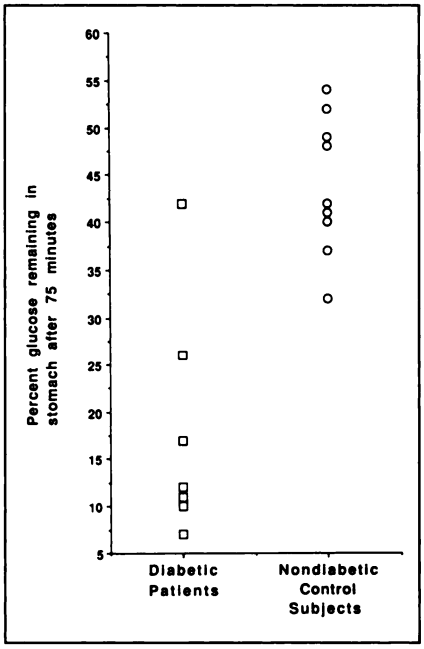

FIGURE 3. Percent solution remaining in stomach 75 min after ingestion of oral glucose solution.

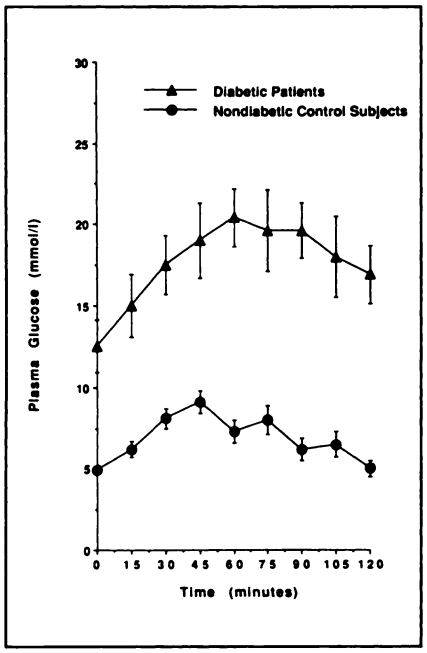

FIGURE 4. Plasma glucose concentration of diabetic and nondiabetic patients after ingestion of oral glucose solution.

concentration during the 2 hr for the diabetic patients was 18.1 mmol/liter (s.e.m. = 1.7) and 6.7 mmol/liter (s.e.m. = 0.5) for the nondiabetic control subjects ($p = 0.0001$). The glucose curve in Figure 4 was much steeper from 0 to 45 min in the diabetic patients when compared to the nondiabetic control subjects ($p = 0.015$). Even though the diabetic subjects had more rapid gastric emptying, their plasma-glucose peak was delayed (60–75 min) when compared to the nondiabetic control group (45 min).

The average rate of calories emptied into the intestine (calculated using the gastric half-emptying time) by the diabetic patients was 3.3 kcal/min (s.e.m. = 0.5), while nondiabetic control subjects emptied at a rate of 1.6 kcal/min (s.e.m. = 0.1). The extremes in caloric emptying varied between a diabetic subject emptying 6.9 kcal/min and a nondiabetic control emptying 1.2 kcal/min.

DISCUSSION

The important role of the stomach in the regulation of glucose homeostasis has only recently become recognized (*12*). In 1982, Thompson (*12*) described the rate of gastric emptying as an important determinant of blood glucose concentration after an oral glucose load and suggested that the glucose tolerance test could be used to assess the rate of gastric emptying. A more rapid gastric emptying of a glucose load from the stomach would result in more rapid intestinal absorption of glucose and increased postprandial glucose levels. In diabetic patients, rapid gastric emptying of high carbohydrate meals would obviously make blood glucose control more difficult.

The gastric emptying rate in normal subjects for the glucose solution used in this study is consistent with our previous findings (*13*) and those reported by other authors in the literature (*14,15*). In a previous study, we determined that a group of nondiabetic men and women emptied glucose from their stomachs at a rate of 1.6 kcal/min (*13*). In a study by Liddle (*14*), using scintigraphy, normal subjects who ingested a glucose solution (60 g of glucose in 400 ml of H_2O) had a calorie emptying rate of approximately 1.7 kcal/min. Brener (*15*), using an aspiration technique to measure gastric emptying, stated that normal subjects emptied a variety of glucose solutions at 2.13 kcal/min. Although the gastric emptying rate determined by Brener's study is minimally faster than the gastric emptying rate determined by Liddle's study and the author's previous study, the *aspiration technique* of measuring gastric emptying is well known to overestimate the gastric emptying rate in comparison to the *scintigraphic technique* (*16,17*).

In this study, we noted a significantly more rapid gastric emptying rate in the recently diagnosed Type 2 diabetic patients when compared to the sex- and age-matched nondiabetic control subjects. The Type 2 diabetic patients emptied their stomach at an average rate of 3.3 kcal/min, while the nondiabetic control subjects emptied at a slower average rate of 1.6 kcal/min. One of our recently diagnosed diabetic subjects emptied glucose from his stomach at a surprisingly rapid rate of almost 7 kcal/min (in an exponential fashion similar to water), while the most rapid nondiabetic control subject emptied at a rate of less than 2.0 kcal/min. The steeper rise of the glucose curve from 0 to 45 min in the Type 2 diabetic patients is probably due to the rapid gastric emptying in these patients. It is interesting to note that even though the Type 2 diabetic patients had twice the rate of gastric emptying compared to the nondiabetic subjects, their plasma-glucose levels did not peak until 60 min or later, whereas all nondiabetic control subjects had glucose peaks between 30–45 min. The delayed plasma-glucose peak seen in our patients with Type 2 diabetes is probably due to insulin resistance or a delayed insulin response. This delay of the plasma-glucose peak in diabetic patients has been observed by other authors (*18*).

Although seven of our nine diabetic subjects were taking their oral hypoglycemic medication, their fasting plasma-glucose values as a group remained high (mean 12.5 mmol/liter, s.e.m. 1.6; range 6.3–18.4 mmol/liter). In addition, there was no correlation in our group of diabetic patients between the rate of gastric emptying and the dose (or lack of dose) of oral hypoglycemic agents taken by the patient.

High peripheral glucose values have been reported by other investigators (*19–21*) to delay gastric emptying in normal subjects. In the diabetic subjects of this study, the elevated plasma-glucose values appear to result in little, if any, delay in gastric emptying. We have hypothesized that there may be a loss (or absence) of feedback control of gastric emptying in diabetic patients. A recent publication supports this view by demonstrating a *lack* of delayed gastric emptying in Type 2 diabetic patients due to induced acute hyperglycemia (*22*). Two other recently published

abstracts describe rapid gastric emptying in Type 1 diabetics (*23,24*). Nowak (*23*) reported accelerated gastric emptying in asymptomatic patients with insulin-dependent diabetes. In this study, diabetic subjects had a gastric half-emptying time of 93 min while nondiabetic controls had a half-emptying time of 147 min. Mecklenbeck (*24*) has also recently described two different gastric emptying kinetics in insulin-dependent diabetics. Of 12 diabetics studied, 7 had accelerated gastric emptying and 5 had delayed gastric emptying. The diabetics with accelerated emptying had only 26% of the meal remaining in their stomachs at 1 hr in comparison to 53% remaining in the stomachs of nondiabetic control subjects. Granneman and Stricker (*25*), conducting research on streptozotocin-induced diabetic rats, have also described a significantly increased gastric emptying rate of a high carbohydrate meal in diabetic rats compared to control rats. This rapid gastric emptying rate was attributed to an abnormal insensitivity in these diabetic rats to postabsorptive events that occur when a high carbohydrate diet is consumed.

Other indirect evidence supports the hypothesis that rapid gastric emptying of carbohydrates may commonly occur in Type 2 diabetic patients. Various authors (*26–28*) have shown that a majority of Type 2 diabetic patients have an increased level of gastric inhibitory peptide hormone (GIP) after consumption of either an oral glucose meal (*27*) or a solid/liquid meal (*28*). GIP is considered to be a good marker of glucose absorption by the small intestine (*29,30*). These elevated GIP levels are consistent with rapid gastric emptying, although they have been previously attributed to a defective insulin response which leads to a diminished feedback inhibition of GIP secretion by insulin (*31*).

A genetic predisposition to rapid gastric emptying may play an important role in the early development of Type 2 diabetes. Rapid gastric emptying, as an early homeostatic derangement, would result in an initially higher than normal peak plasma-glucose level, which may eventually lead to down regulation of the glucose transport system and resultant Type 2 diabetes. This proposed mechanism is consistent with the hypothesis of other investigators for the development of Type 2 diabetes. In 1988, DeFronzo (*32*) hypothesized that chronic hyperglycemia could lead to a generalized desensitization of all cells in the body and cause a down regulation of the glucose transport system. In 1989, Robertson (*33*) also suggested that the etiology of Type 2 diabetes may be due to the "homologous desensitization of the β-cell secretory apparatus to glucose." A pharmacologically induced decrease in the rate of gastric emptying in the diabetic or prediabetic patient with rapid gastric emptying might be a new approach to slowing postprandial carbohydrate absorption in addition to previously described methods such as soluble fiber supplements, low glycemic index diets, alpha-glucosidase inhibitors or nibbling (*34*).

In summary, many Type 2 diabetic patients exhibit abnormally rapid gastric emptying which may contribute to a worsening of their glucose control. By performing gastric emptying studies on more patients who have been recently diagnosed with diabetes or patients with a family history of diabetes, we hope to determine if rapid gastric emptying is a possible etiology or risk factor for this disease.

REFERENCES

1. Rundles, RW. Diabetic neuropathy. *Medicine* 1945;24:111–160.
2. Loo FD, Palmer DW, Soergel KH, Kalbfleisch JH, Wood CM. Gastric emptying in patients with diabetes mellitus. *Gastroenterology* 1984;86: 485–494.
3. Horowitz M, Harding PE, Maddox AF, et al. Gastric and oesophageal emptying in patients with Type 2 (noninsulin-dependent) diabetes mellitus. *Diabetologia* 1989;32:151–159.
4. Mulhall BP, O'Fearghail M. Diabetic gastroparesis. *Anaesthesia* 1984;39: 468–469.
5. Saltzman MB, McCallum RW. Diabetes and the stomach. *Yale J Biol Med* 1983;56:179–187.
6. Kassander P. Asymptomatic gastric retention in diabetic patients (gastroparesis diabeticorum). *Ann Intern Med* 1958;48:797–812.
7. Katz LA, Spiro HM. Gastrointestinal manifestations of diabetes. *N Engl J Med* 1966;275:1350–1360.
8. Campbell IW, Heading RC, Tothill P, Buist TAS, Ewing DJ, Clarke BF. Gastric emptying in diabetic autonomic neuropathy. *Gut* 1977;18: 462–467.
9. WHO expert committee on diabetes mellitus: second report. *WHO Tech Rep Ser* 646, 1980.
10. Schwartz JG, Phillips WT, Aghebat-Khairy B. Revision of the oral glucose tolerance test: a pilot study. *Clin Chem* 1990;36:125–128.
11. Christian PE, Datz FL, Sorenson JA, Taylor A. Technical factors in gastric emptying studies [Teaching Editorial]. *J Nucl Med* 1983;24:264–268.
12. Thompson DG, Wingate DL, Thomas M, Harrison D. Gastric emptying as a determinant of the oral glucose tolerance test. *Gastroenterology* 1982; 82:51–55.
13. Phillips WT, Schwartz JG, Blumhardt R, McMahan CA. Linear gastric emptying of hyperosmolar glucose solutions. *J Nucl Med* 1991;32: 377–381.
14. Liddle RA, Rushokoff RJ, Morita ET, Beccaria L, Carter JD, Goldfine JD. Physiologic role for cholecystokinin in reducing postprandial hyperglycemia in humans. *J Clin Invest* 1988;81:1675–1681.
15. Brener W, Hendrix TR, McHugh PR. Regulation of the gastric emptying of glucose. *Gastroenterology* 1983;85:76–82.
16. VanDeventer G, Thomson J, Graham LS, Thomasson D, Myer JH. Validation of corrections for errors in collimation during measurement of gastric emptying of nuclide-labeled meals. *J Nucl Med* 1983;24:187–196.
17. Beckers EJ, Leiper JB, Davidson J. Comparison of aspiration and scintigraphic techniques for the measurement of gastric emptying rates of liquids in humans. *Gut* 1992;33:115–117.
18. Reaven GM. Banting lecture 1988. Role of insulin resistance in human disease. *Diabetes* 1988;37:1595–1607.
19. Groop LC, DeFronzo RA, Luzi L, Melander A. Hyperglycaemia and absorption of sulfonylurea drugs. *Lancet* 1989;ii:129–130.
20. MacGregor IL, Gueller R, Watts HD, Meyer JH. The effect of acute hyperglycemia on gastric emptying in man. *Gastroenterology* 1976;70: 190–196.
21. Fraser RJ, Horowitz M, Maddox AF, Harding PE, Chatterton BE, Dent J. Hyperglycaemia slows gastric emptying in Type I (insulin-dependent) diabetes mellitus. *Diabetologia* 1990;33:675–680.
22. Dao T, Chee B, Bouvard G, Justum AM, Verwaerde JC, Valla A. Lack of modulation of gastric emptying by acute hyperglycemia in Type 2 diabetes mellitus [Abstract]. *Gastroenterology* 1990;A342.
23. Nowak TV, Johnson CP, Wood CM, et al. Evidence for accelerated gastric emptying in asymptomatic patients with insulin-dependent diabetes. *Gastroenterology* 1990;96.
24. Mecklenbeck W, Rathmann W, Vosberg M. Gastric emptying in diabetic neuropathy: two different forms of disturbed kinetics [Abstract]. *Eur J Nucl Med* 1991;18:FP-3H3-1.
25. Granneman JG, Stricker EM. Food intake and gastric emptying in rats with streptozotocin-induced diabetes. *Am J Physiol* 1984;247:

R1054–1061.
26. Creutzfeldt R, Ebert M, Nauck M, Stöckmann F. Disturbances of the entero-insular axis. *Scand J Gastroenterol* 1983;18:111–119.
27. Elahi D, Anderson DK, Müller DG, Tobin JD, Brown JC, Andres R. The enteric enhancement of glucose-stimulated insulin release. The role of GIP in aging, obesity and noninsulin dependent diabetes mellitus. *Diabetes* 1984;33:950–957.
28. Serrice FJ, Rizza RA, Westland RE, Hall LD, Gerich JE, Go VLW. Gastric inhibitory polypeptide in obesity and diabetes mellitus. *J Clin Endocrinol Metab* 1984;58:1133–1140.
29. Jenkins DJA, Wolever TMS, Ocana AM, et al. Reducing the rate of glucose ingestion by single bolus versus continuous sipping. *Diabetes* 1990;39: 775–781.
30. Thomas FB, Shook DF, O'Dorisio TM, et al. Localization of gastric emptying inhibitory polypeptide release by intestinal glucose perfusion in man. *Gastroenterology* 1977;72:49–54.
31. Ross SA, Brown JC, Dupré J. Hypersecretion of gastric inhibitory polypeptide following oral glucose in diabetes mellitus. *Diabetes* 1977;26: 525–529.
32. DeFronzo RA. The triumvirate: β-cell, muscle, liver. A collusion responsible for NIDDM. *Diabetes* 1987;37:667–687.
33. Robertson P. Type II diabetes, glucose "non-sense," and islet desensitization. *Diabetes* 1989;38:1501–1505.
34. Jenkins DJA, Jenkins AL, Wolever TMS, Vukson V. Fiber and physiological and potentially therapeutic effects of slowing carbohydrate absorption. *Adv Exp Med Biol* 1990;270:129–134.

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SELF-STUDY TEST
Radiobiology and Radiation Protection
ANSWERS

2. Finch SC. Acute radiation syndrome. *JAMA* 1987;258:664–667.
3. Mole RH. The LD_{50} for uniform low LET irradiation in man. *Br J Radiol* 1984;57:355–369.

ITEM 10-13: Federal Radiation Advisory and Regulatory Agencies

ANSWERS: 10, B; 11, A; 12, C; 13, B

The Nuclear Regulatory Commission (NRC) is charged by Congress with the responsibility for the regulation of the uses of source material (natural uranium and thorium), special nuclear material (enriched uranium and plutonium), and by-product material (fission products and materials that are created by fission–neutron transmutations). The NRC does not regulate naturally occurring (such as radium, radon, and potassium-40) and accelerator-produced radioactive materials (NARM). To assure the safe use of by-product materials, the NRC publishes regulations that establish minimum training and experience requirements for licensure to use by-product materials and radiation protection standards that must be observed by licensees. Many of these regulations apply directly to the clinical practice of nuclear medicine. The regulations published by the NRC carry the full weight of law, and violation of the regulations or license conditions may result in civil penalties and suspension or revocation of the license. Most states have established radiation protection regulations and licensing procedures similar to those of the NRC for the safe use of NARM. Many states have signed formal agreements with the NRC to assume the responsibility for regulation of by-product material uses within their own borders and, thus, license all radioactive materials, whether by-product or NARM; such states are called Agreement States.

The Food and Drug Administration is charged by Congress with assuring the safety and efficacy of drugs used in the United States. During those years that the Atomic Energy Commission (AEC) was responsible for the licensing of by-product material, FDA deferred to the judgement of AEC in determining which radiopharmaceuticals should be licensed to physicians for general use. The FDA assumed responsibility for the premarketing evaluation and approval of radiopharmaceuticals some years ago and now subjects radiopharmaceuticals to the same regulatory approval process as applies to all other types of drugs.

Occasions may arise wherein a researcher would like to use an unapproved radioactive drug to investigate a very well-defined question in a limited population of research subjects, e.g., to establish pharmacokinetics of a particular drug in a rare disease. The Radioactive Drug Research Committee (RDRC) regulations were established by the FDA to deal with these special circumstances. Where applicable, these regulations facilitate research with particular radioactive drugs because they obviate submission of a "Notice of Claimed Investigational Exemption for a New Drug" (IND). After an institution has established an FDA-approved RDRC, the committee is authorized to review and approve research studies of the type defined by the RDRC regulations; further approval of the research protocol by the FDA is not required. The RDRC regulations are quite specific as to what types of investigation are permitted, and prescribe organ and whole-body dose limits, as well.

The Environmental Protection Agency (EPA) is charged by Congress with the responsibility for ensuring that the environment is not harmed by the actions of industry, government, or private citizens. The dividing line between EPA and NRC responsibilities with regard to radiation hazards has always been fuzzy, and in many cases NRC licensees must deal with both agencies in trying to resolve differences between regulations established by each agency. The EPA has been assigned the lead agency responsibility for establishment of broad radiation protection policies for federal agencies, a role formerly filled by the now-defunct Federal Radiation Council. For example, the International Commission on Radiation Protection (ICRP) recently recommended a new approach to radiation protection standards. The EPA was responsible for determining whether the United States should adopt the new ICRP approach. The EPA is also the lead agency in attempting to solve the radioactive waste disposal problem.

The National Council on Radiation Protection and Measurements (NCRP) was chartered by Congress as a nonprofit organization in 1964. The NCRP is an advisory group of eminent radiation scientists who develop recommendations on how to deal with specific radiation protection questions. These recommendations are published as NCRP reports. NCRP reports are strictly advisory, but the imprimatur of the NCRP makes them de facto national standards of good radiation protection practice. In the absence of specific regulations from NRC or other federal agencies, most health physicists implement NCRP recommendations as an integral part of their radiation protection programs.

The Center for Devices and Radiological Health (CDRH) is a branch of the FDA that develops performance standards for medical devices and for applications of radiation in humans. The CDRH also develops educational programs designed to enhance user and community awareness of proper ways to use radioactive materials and radiation-emitting machines. Performance standards established by CDRH are published as FDA regulations in Title 21 of the Code of Federal Regulations.

ITEMS 14-17: ICRP Publication 26

ANSWERS: 14, F; 15, T; 16, T; 17, T

ICRP Publication 26 reflects the results of a complete restudy of the radiobiologic literature and a fresh look at radiation protection guidelines. ICRP 26 recommends that occupational exposure limits be based on control of annual exposure without considering separately the pattern of exposure over the working lifetime of the individual. Thus, ICRP recommends that the 5(N – 18) formula for calculating acceptable lifetime dose be eliminated. ICRP 26 further recommends that the critical organ concept be abandoned in favor of assessment of the total radiation insult to the body. The absorbed dose to each organ within the body is to be calculated, and then appropriate weighting factors for each organ are applied to arrive at the total effective dose-equivalent. There probably will be further refinement in the numerical values of the weighting factors, but the general concept seems to be firmly established. Finally, ICRP 26 closed a long-standing loophole regarding the dose contribution from internally deposited radionuclides. Previous guidance from ICRP and still-current regulations of the U.S. Nuclear Regulatory Commission provide dose limits for irradiation by sources external to the body and require that the activity of any internally deposited radionuclides be assessed and recorded, but included no requirement that the absorbed dose due to the internally deposited radionuclides be included in the 5 rems/year

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