During the past 20 years, an increase in survival among pediatric patients with cancer has occurred, and a need exists to be proactive prior to cancer therapy regarding issues related to fertility. Chemotherapy and radiation can cause a number of deleterious side effects to female patients, including early menopause and its associated side effects of osteoporosis and heart disease as well as the inability to carry a pregnancy to term or conceive a child. Many drugs and fields of radiotherapy are associated with an increased incidence of female reproductive complications. Options are available for the preservation of female fertility, but many are experimental. Of highest importance is the need to counsel female adolescents and young adults before beginning induction chemotherapy or radiation. Nurses play a large part in the information about future fertility that female patients receive before the initiation of cancer therapy. After reading this article, nurses will have a better understanding of the impact of cancer therapy on the female reproductive system and be more comfortable discussing the topic with their patients.
Byrne et al. (2004) concluded that cancer therapy can directly affect hormone production in females, causing early menopause and predisposing patients to complications such as increased risk of osteoporosis and heart disease. Another study by Haddy et al. (1998) found that long-term, female survivors of Hodgkin disease who received only chemotherapy were not infertile, but if therapy included radiation, the incidence of infertility increased. Also, girls who received abdominal radiation for Wilms tumor were less likely as adults to carry a pregnancy to term because of the direct effect of radiation to the growth and development of the uterus (Larsen et al., 2004).

To understand the effects of cancer therapies on the female reproductive system, one must understand the cellular biology of a healthy, functioning female reproductive system. A healthy female is born with all of the oocytes, or ovarian follicles, she will ever produce. From birth until menarche, the number of ovarian follicles decreases from more than one million to 300,000 through a process called atresia (American Society for Reproductive Medicine [ASRM], 2003). Atresia is an age-related, hormonally controlled, degenerative process that occurs throughout a female’s entire life. Research has found that some environmental factors can increase the rate of atresia, such as smoking or radiation (ASRM). Midway through the third decade of a woman’s life, the ovarian reserve has decreased to approximately 25,000, and atresia automatically accelerates, making spontaneous pregnancy more difficult compared to the rate of spontaneous pregnancy in the early 20s. By about the fifth decade of life, the number of ovarian follicles is minimal, and menopause occurs (Larsen, Muller, Schmiegelow, Rechnitzer, & Andersen, 2003). Chemotherapy, radiation, and bone marrow transplantation all play a part in accelerating follicle atresia, inhibiting hormones crucial to the normal functioning of the female reproductive system, or changing the physiologic structure of the female reproductive system, all of which lead to female infertility.

### Chemotherapy

Many young females who receive chemotherapy as part of their cancer treatment will not become infertile (Larsen, Muller, Rechnitzer, et al., 2003; Green et al., 2002). In fact, females have been found to be less susceptible than men to fertility changes related to chemotherapy (Thomson, Critchley, Kelnar, & Wallace, 2002). However, ovarian follicles can decrease in number as a result of chemotherapy. The mechanism by which chemotherapy reduces the ovarian follicle reserve occurs in a dose-related manner by accelerating atresia (Larsen, Muller, Schmiegelow, et al., 2003; Thomson et al.). Because atresia is accelerated, patients have a greater likelihood that the number of ovarian follicles after treatment with high-dose chemotherapy will be decreased, which can lead to menopause earlier in life than expected (Larsen, Muller, Schmiegelow, et al.; Thomson et al.).

Larsen, Muller, Schmiegelow, et al. (2003) found that inhibin B, a reproductive hormone, was lower in patients with spontaneous menstrual cycles postchemotherapy. The presence of menstruation suggested that patients’ reproductive hormone levels were normal; however, decreased levels of inhibin B may be indicative of advanced reproductive age because patients without a previous history of cancer therapy who are older than 35 years of age have levels of inhibin B that are similar to those of much younger patients post–cancer therapy. This suggests that inhibin B may be “the earliest endocrine marker of the decline in antral follicle number” (Larsen, Muller, Schmiegelow, et al., p. 5,312). Decreased ovarian volume also was found by transvaginal ultrasound in patients with spontaneous menstrual cycles (Larsen, Muller, Schmiegelow, et al.). The phenomenon was exhibited in all patients receiving chemotherapy despite which type they received (Larsen, Muller, Schmiegelow, et al.). Therefore, even though patients receiving only chemotherapy for pediatric cancers may be fertile, evidence shows that they may experience premature ovarian failure in comparison to healthy females who are the same age.

Despite a decreased number of ovarian follicles, patients still can become pregnant. Huong et al. (2002) isolated the affects of cyclophosphamide, an alkylating agent, on female fertility in their analysis of 84 women aged 13–53 years who received cyclophosphamide for nononcologic disorders such as systemic lupus erythematosus. All patients received approximately 1 g of cyclophosphamide every month for an average of 13 cycles. The researchers found a correlation between an increased risk of ovarian failure and patient age at the initiation of cyclophosphamide therapy. Older patients were more likely to experience ovarian failure sooner than younger patients (Huong et al.). Women aged 13–25 years who received cyclophosphamide therapy were able to become pregnant after completing cyclophosphamide therapy.

The drugs administered to cure many different types of cancer are likely, in combination, to decrease the number of ovarian follicles. Green et al. (2002) studied patients who had survived childhood cancer and, when compared to their healthy siblings, found a twofold risk of being unable to conceive a child or carry a child to term if they had received certain chemotherapy agents. Blatt (1999) also reviewed a number of drugs that could hinder female fertility and found that some chemotherapy agents may not have an effect on fertility. A third study by Muller (2003) found a number of drugs that could cause a high risk of gonadal damage. The results of the studies are detailed in Table 1. Some variation occurred among the results of the studies regarding which drugs cause female infertility. More research is needed in this area but may be complicated by the use of combination chemotherapy regimens and concurrent radiotherapy, which also causes a high rate of female infertility.

### Radiotherapy

Radiation therapy has been shown in many studies to be the primary cause of female infertility post–cancer therapy despite a patient’s age at treatment. Cranial-spinal and abdominal radiation have been shown to lead to ovarian failure. Even more damaging is radiation in combination with chemotherapy. However, the modality of radiation that, by far, has caused the most bodily insult is total body irradiation used as a conditioning regimen prior to bone marrow transplantation. Combination treatments may offer the best chances of survival for patients with cancer but may cause extensive fertility and reproductive difficulties later in life.
Cranial-Spinal Irradiation

Cranial-spinal irradiation prohibits the functioning of the hypothalamic-pituitary axis and inhibits the release of gonadotropin-releasing hormone. This modality of radiotherapy is used commonly in children who have malignant neurologic tumors and has been used for prophylactic treatment of the central nervous system in patients with leukemia and non-Hodgkin lymphoma (Muller, 2003). Hypogonadotropic hypogonadism, or ovarian failure secondary to the inhibition of gonadotropin-releasing hormone from the hypothalamus, is a very common complication associated with cranial-spinal irradiation and occurs in 25% of patients (Byrne et al., 2004; Muller). The risk of hypogonadotropic hypogonadism is highly age- and dose-dependent. Byrne et al. found that patients who received cranial-spinal radiation within two years before or after menarche may have an increased risk of hypogonadotropic hypogonadism. Although the researchers noted that 30–40 Gy doses of radiation to the hypothalamic-pituitary axis caused gonadotropin deficiency, the study suggested that “irradiation of the hypothalamic-pituitary area with doses in the range of 18–24 Gy, when administered to females around the time of menarche, may result in subtle abnormalities that may affect fertility” (Byrne et al., p. 596).

A high rate of ovarian failure in some children with leukemia and non-Hodgkin lymphoma who received cranial-spinal radiation from the mid-1970s to the early 1990s may have been caused by radiation therapy to the spinal field that may have scattered to the ovaries and not by hypogonadotropic hypogonadism (Green et al., 2002). Adults who received this type of radiation as children have experienced similar effects to those who received abdominal radiation for primary intra-abdominal tumors, such as decreased uterine size, leading to an increased rate of spontaneous abortion (Green et al.).

Abdominal Irradiation

Radiation to the entire abdomen affects the female reproductive organs, primarily by impairing uterine growth and blood flow (Crichtley, Bath, & Wallace, 2002). A risk of ovarian follicle damage is also possible with whole abdominal radiation (Crichtley et al.). Whole abdominal radiation doses of 12–50 Gy have been responsible for ovarian failure in all women who have received the therapy during childhood for intra-abdominal malignancies (Nussbaum Blask et al., 1999). A study by Nussbaum Blask et al. showed, using sonography, that the ovaries in this subset of patients were small and abnormal or atretic.

Of women who receive whole abdominal radiation as children and maintain fertility, pregnancy may be difficult. If a woman who received whole abdominal radiation as a child became pregnant, she may have a high risk of premature labor and low birth weight because the uterus sustains significant damage during radiation therapy (Crichtley et al., 2002). Schwartz (1999) argued that flank radiation for treatment of Wilms tumor may not affect the ovaries but could cause decreased blood flow to the uterus, leading to the inability of the uterine muscle to grow during pregnancy. Estrogen replacement has been used following radiation therapy to counteract the effects of radiation on the uterus; however, the uterus still tends to grow to only 40% of the adult size (Green et al., 2002; Larsen et al., 2004). Women in this situation are likely to have children that are premature and weigh less than 2,500 g at birth (Green et al.).

Table 1. Risk of Female Gonadal Damage by Chemotherapeutic Agents: A Review of the Literature

<table>
<thead>
<tr>
<th>DRUG CLASS</th>
<th>DRUG</th>
<th>BLATT, 1999</th>
<th>GREEN ET AL., 2002</th>
<th>MULLER, 2003</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alkylating agents</td>
<td>Busulfan</td>
<td>0</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Chlorambucil</td>
<td>0</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Cisplatin</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Cyclophosphamide</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Dacarbazine</td>
<td>+</td>
<td>–</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Ifosfamide</td>
<td>+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Melphalan</td>
<td>0</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Nitrogen mustard</td>
<td>0</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Procarbazine</td>
<td>0</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Nitrosureas</td>
<td>Carmustine</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Lomustine</td>
<td>+</td>
<td>–</td>
<td>+</td>
</tr>
<tr>
<td>Antitumor antibiotics</td>
<td>Bleomycin</td>
<td>–</td>
<td>–</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Dactinomycin</td>
<td>–</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Daunorubicin</td>
<td>+</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Doxorubicin</td>
<td>+</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>Antimetabolites</td>
<td>Asparaginase</td>
<td>–</td>
<td>0</td>
<td>0</td>
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<tr>
<td></td>
<td>Cytarabine</td>
<td>–</td>
<td>0</td>
<td>–</td>
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<tr>
<td></td>
<td>Mercaptopurine</td>
<td>+</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Methotrexate</td>
<td>–</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td></td>
<td>Thioguine</td>
<td>–</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Plant alkaloids</td>
<td>Etoposide</td>
<td>+</td>
<td>–</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Tenoposide</td>
<td>0</td>
<td>–</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Vincristine</td>
<td>–</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Vinblastine</td>
<td>–</td>
<td>–</td>
<td>+</td>
</tr>
</tbody>
</table>

Note. A “+” indicates that a drug harms ovaries, a “–” indicates little or no effect on ovaries, and a “0” indicates that a drug was not reviewed.
Bone Marrow Transplantation

For female patients receiving bone marrow transplantation as part of their treatment regimens, the combination of alkylating chemotherapy and total body irradiation causes a very high incidence of hypogonadism and infertility, requiring hormone-replacement therapy (Leung et al., 2000). Two researchers found that high-dose alkylating agents, such as cyclophosphamide in doses greater than 200 mg/kg, are responsible for ovarian failure (Larsen, Muller, Schmiegelow, et al., 2003; Thomson et al., 2002). When Larsen, Muller, Schmiegelow, et al. examined female patients who had received a cyclophosphamide conditioning regimen prior to bone marrow transplantation, 7 of 10 were receiving hormone-replacement therapy for ovarian failure and three reported spontaneous menstrual cycles but exhibited lower total follicle numbers and small ovarian volume on physical examination. The effects were not seen in patients with spontaneous menstrual cycles who received chemotherapy and/or radiotherapy without bone marrow transplantation (Larsen, Muller, Schmiegelow, et al.). Inhibin B levels were decreased significantly and estradiol levels were increased in the patients who experienced ovarian failure post-bone marrow transplantation, indicating little or no ovarian function. Ovarian volume was minimal, and the ovaries of the patients appeared small and shriveled on sonography (Larsen, Muller, Schmiegelow, et al.).

Total body irradiation poses the biggest risk of infertility in female patients following bone marrow transplantation. Total body irradiation impairs the secretion of gonadotropin-releasing hormone from the hypothalamus, leading to hypogonadotropic hypogonadism (Mayer et al., 1999). Simon, Lee, Partridge, and Runowicz (2005) suggested that total body irradiation is responsible for approximately 90% of ovarian failure in patients who have received the regimen and that less than 3% of patients receiving total body irradiation will be able to become pregnant. A study by Mayer et al. showed that gonadal function after a total dose of 12 Gy total body irradiation prior to bone marrow transplantation caused eventual early impaired gonadal function. Even in patients who did undergo spontaneous puberty, their follicle-stimulating hormone levels were declining at the time of the study, indicating ovarian failure (Mayer et al.). Thomson et al. (2002) found that, of 144 patients treated with cyclophosphamide (120 mg/kg) and total body irradiation (9.2–15.75 Gy), all developed short-term amenorrhea and only 9 of the 144 recovered their menses, indicating infertility in 93.75% (135 of 144) of patients. Of 176 patients treated with cyclophosphamide and busulfan or cyclophosphamide and total body irradiation as a conditioning regimen prior to bone marrow transplantation, ovarian failure was present in only 68% of patients who received cyclophosphamide and busulfan versus 90% in patients who received cyclophosphamide and total body irradiation (Thomson et al.).

A patient’s age at the time of treatment may be a factor in the onset of puberty. Bakker et al. (2000) suggested an increased incidence of spontaneous pubertal onset in patients who received cyclophosphamide and total body irradiation as a conditioning regimen for bone marrow transplantation before puberty. The patients, however, did exhibit eventual gonadal failure during the teen years, requiring synthetic hormones to counteract the effects of menopause (Bakker et al.). In patients who underwent bone marrow transplantation and were fertile for a period of time, studies have shown that spontaneous abortion rates were almost 40% and that the incidence of preterm labor was higher than 80% (Bakker et al.). The increased incidence of spontaneous abortion and preterm labor could be secondary to the effects of total body irradiation on the vasculature and tissue of the uterus, as in patients who receive abdominal radiation only (Critchley et al., 2002).

These findings conclude that, after bone marrow transplantation, all female patients should be followed by an oncologist in consultation with an endocrinologist to monitor for endocrine dysfunction. Patients and parents should be counseled regarding the high risk of infertility following cyclophosphamide and total body irradiation before bone marrow transplantation.

Counseling Female Patients: The Evidence Base

First, female patients with cancer must understand that becoming pregnant while receiving chemotherapy is possible. Knowing the risk, sexually active adolescent and young adult women must use contraception or be abstinent to prevent pregnancy during cancer therapy (Bergstrom & Altman, 1998). In a study by Huong et al. (2002), four patients became pregnant during IV cyclophosphamide therapy. Two infants were born with deformities, such as microcephaly, ear and eye malformations, and hypoplastic thumbs. The infants had been exposed to IV cyclophosphamide in the first trimester of the pregnancies. One pregnant patient chose to therapeutically abort the child early in pregnancy secondary to the risk of teratogenicity. The fourth child was conceived 10 days after IV cyclophosphamide therapy was completed, and the pregnancy ended in a healthy, live birth. Pregnancy also has occurred in female adolescents during treatment for childhood cancer. Bergstrom and Altman presented two case reports of female, adolescent patients who became pregnant while undergoing maintenance treatment for acute lymphoblastic leukemia. One patient chose to therapeutically abort the child. The second patient stopped cancer therapy when physicians discovered the pregnancy at five months gestation, and the patient carried the child for 34 weeks. At that time, the patient developed hemolysis, elevated liver enzymes, and low platelet count syndrome, and labor was induced. At the time of publication, the mother and child were well. The child had no abnormalities that could be attributed to the mother’s cancer therapy. The mother did not resume cancer therapy and was in remission at six months after the child’s birth.

Even with planned birth, fertility may not be the only problem experienced by patients who are interested in becoming pregnant after cancer therapy. Chemotherapy and radiation cause organ damage, which can cause complications such as cardiopulmonary stress, which may put patients’ lives in jeopardy (Hobie, Ruccione, Harvey, & Moore, 2002). The recommendation is that any patient who has received cancer therapy and is interested in becoming pregnant should seek the advice of an obstetrician specializing in high-risk pregnancies before becoming pregnant (Hobie et al.).

Many patients are afraid that the doses of chemotherapy or radiation they received as children will cause birth defects
in children conceived later in life. A case study by Stovall et al. (2004) of 25,000 patients post-cancer therapy found no correlation between previous doses of radiotherapy and congenital malformations in patients’ children conceived years after therapy. Blatt (1999) did not find sufficient evidence of increased risk of congenital abnormalities or nonhereditary cancer occurring in children after parents’ childhood chemotherapy or radiation therapy. Stovall et al. also cited other authors who found no correlation between an increase in congenital malformations and patients who received chemotherapy and radiotherapy. These patients are likely to have children that are normal and healthy. However, exceptions do exist, such as patients who can pass on a single gene-inherited cancer, like retinoblastoma, to their children or patients who received abdominal radiation as children because abdominal radiation decreases uterine size, which can lead to birth defects (Green et al., 2002; Hobbie et al., 2002). Patients with a history of a single gene-inherited cancer should be offered genetic counseling and genetic testing (Hobbie et al.). Patients with a history of abdominal radiation should be referred to an obstetrician specializing in high-risk pregnancies.

Some experimental strategies that are being used to preserve female fertility include oocyte cryopreservation, embryo cryopreservation, ovarian tissue harvesting with transplantation, and gonadal suppression by gonadotrophin analogs.

To minimize damage to the ovaries from abdominal radiotherapy, surgical translocation, or oophoropexy, general surgery or laparoscopy may be used to move the ovaries to a location outside of the radiation field or behind the uterus, which can shield the ovaries from intense radiation exposure (Thomson et al., 2002; Visvanathan, Cutner, Cassoni, Gaze, & Davies, 2003). Of note, the procedure may not be useful in prepubertal children because the uterus is small (approximately 1.6 ml in volume compared to 43 ml in a nulliparous adult) and may not be large enough to shield the ovaries (Larsen et al., 2004; Schwartz, 1999). Even with the increased risk of radiation exposure in young children who receive this procedure compared to adults, oophoropexy has been accepted as a means to preserve fertility in female patients who are receiving abdominal irradiation (Thomson et al.). With the procedure, a small risk exists of ovarian cyst formation or disruption of the reproductive system vasculature, causing a decrease in fertility (Meirow & Nugent, 2001). Parents and patients should be informed of the risk before surgery. Parents also should be made aware, before abdominal radiation is initiated, that younger children have a greater chance of having smaller uterine volume as adults (Meirow & Nugent).

Some experimental strategies that are being used to preserve female fertility include oocyte cryopreservation, embryo cryopreservation, ovarian tissue harvesting with transplantation, and gonadal suppression by gonadotrophin analogs.

Embryo cryopreservation has produced positive results in patients post-cancer therapy, but the ethical implications of storing an embryo prior to cancer therapy must be considered, including the risk of patient death or separation from the father during therapy, which may cause legal complications (Oktay & Sonmezer, 2004). The therapy may not be applicable for adolescent or young adult patients because donor sperm is needed to fertilize an oocyte before cryopreservation (Oktay & Sonmezer).

Many people have used ovarian tissue cryopreservation, but at the time of a study by Aubard et al. (2001), only one attempt to reintroduce cryopreserved ovarian tissue to the body had been made. An attempt at moving ovarian tissue to an area outside of the field of radiation, such as the arm, recently produced a pregnancy and live birth after reimplantation of the ovarian tissue around the area of the fallopian tubes (Donnez et al., 2004). Unfortunately, the method is not useful in patients receiving total body irradiation and high-dose chemotherapy (Oktay & Sonmezer, 2004). In addition, transplantation of ovarian tissue has been reported to have a likelihood of reintroducing tumor to the body following reimplantation of the tissue, which is an especially high risk for patients who had reproductive or abdominal cancers (Oktay & Sonmezer).

Gonadal suppression by gonadotropin analogs (GnRHa) concurrent with chemotherapy is the only current nonsurgical technique available to preserve female fertility. A female patient is injected intramuscularly with a GnRHa, such as lupron, once a month during cancer treatment (Blumenfeld, Dann, Avivi, Epelbaum, & Rowe, 2002). The therapy is aimed at reducing the rate of enhanced ovarian follicle atresia during chemotherapy (Blumenfeld et al.). Blumenfeld et al. reported a positive result from the use of GnRHa during chemotherapy; only 3 of 58 patients experienced primary ovarian failure following chemotherapy with concurrent GnRHa therapy versus 32 of 50 control patients who received the same chemotherapy.
and radiotherapy regimen but did not receive GnRHa. A separate study showed similar results to the Blumenfeld et al. study and recommended the use of GnRHa in all pubertal adolescents receiving chemotherapy (Pereyra Pacheco et al., 2001). Yet another study proposed that, although the therapy may be an attractive option for patients receiving chemotherapy, no results have suggested that GnRHa protects ovarian follicles from radiation therapy (Thomson et al., 2002). Of note, the technique is new and more research is needed prior to its widespread use.

**Recommendations for Adolescent and Young Adult Patients With Cancer**

Ovarian failure can affect patients’ quality of life during the teen years. Teenagers may feel social isolation because they are not experiencing typical pubertal changes. Scant research on ovarian failure in adolescence and its effect on quality of life has been done, and more research is needed. Delaying childbirth until later in life is not recommended for female survivors of childhood cancer, especially those who are at high risk of ovarian failure (Hobbie et al., 2002; Larsen, Muller, Schmiegelow, et al., 2003). Hormone-replacement therapy has been indicated to curtail the outward physical effects of menopause, such as hot flashes, mood changes, stress incontinence, and sleep changes (Nachtagall & Nachtagall, 2004). In young adults with premature menopause, hormone-replacement therapy may be initiated to decrease the risk of early-onset osteoporosis associated with menopause and to help maintain regular menstrual cycles, which can improve psychological health (Byrne, 1999; Hobbie et al.). Survivors with early-onset menopause require careful medical follow-up because of an increased risk of osteoporosis and heart disease (Byrne).

For families who may be faced with the tragedy of having a child with cancer, hope that the child may survive and live a healthy and happy adult life is paramount. In fact, hope has been recognized as creating positive health outcomes and an increased ability for families to cope with a chronic illness in small children, patients’ families receive a positive outlook that patients may overcome and survive their devastating illnesses. By understanding the risks of infertility in children who receive cancer therapy, nurses can educate patients and parents appropriately about the risk of future infertility. Nurses also can help adolescents and young adults with cancer understand that alternative methods of having a child exist, such as adoption and surrogacy. Still, although some of the strategies to preserve fertility are experimental, some families may opt to try them. If therapy can be delayed and the family would like to try experimental treatment, they should be supported by the oncology team. Providing patients and families with the hope of a healthy future during the difficult time of induction therapy may help to decrease psychological stress and encourage patients and families to focus on health instead of disease.

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