Letters and Corrections

Letters submitted for possible publication must be identified as such and submitted with a signed transfer-of-copyright form (see Table of Contents for location). Text length must not exceed 400 words. No more than five references may be used; reference format must be that specified in “Information for Readers and Authors.” All parts of letters must be typed double-spaced, including references. Letters not typed double-spaced will not be considered for publication. Tables and figures will not be published. Acceptance of letters for publication depends on several factors: newness of information, relevance to current topics and recent content of this journal, clarity of statement, distribution of topics within this section, conformity to acceptable formats, and space available; only about half of the letters submitted can be published. The Editor reserves the right to shorten letters and make changes to our style. Authors of letters to be published will be notified of their acceptance. Unpublished letters are returned only on request.

The Chronic Fatigue Syndrome

To the Editor: Manu and colleagues (1) recently reported that a large proportion of self-selected patients with chronic fatigue have various psychiatric conditions, as determined by the application of a structured interview, the Diagnostic Interview Schedule (DIS), and that only a few patients had well-recognized organic illnesses.

We agree that primary psychiatric disease is common in patients with fatigue and that only an occasional patient seeking medical care for chronic fatigue has a well-recognized organic illness, such as an occult malignancy. We believe that there is another disorder, the chronic fatigue syndrome, that is also likely to be an organic illness, although it has not been definitively established as such (2). The prevalence of the syndrome is unknown.

The results of several studies (1, 3) have clearly suggested that mood disorders frequently occur in patients with chronic fatigue. A critical question, however, has not been addressed by these studies: Are the patients fatigued because they have a primary mood disorder, or has a mood disorder developed as a secondary component of a chronic organic illness? Stated another way, are the patients feeling sick because they are depressed, or are they depressed because they are sick?

In addition, can the diagnosis of chronic fatigue syndrome be considered if a patient has a mood disorder? We and other researchers (4) have previously stated that the chronic fatigue syndrome should not be excluded if the patient meets the working case definition of the syndrome and if the mood disorder appears to have developed after the onset of the chronic fatiguing illness. It is difficult to make the diagnosis of chronic fatigue syndrome in a patient who clearly had a mood disorder before the onset of chronic fatigue. Although we and other researchers (4) have previously argued otherwise, we now feel that such a history should not necessarily exclude the diagnosis of chronic fatigue syndrome. We have seen patients with longstanding mood disorders who developed the syndrome and had findings that suggested organic illness (for example, persistent fevers, adenopathy, or abnormal laboratory results). Indeed, evidence suggests that a patient's neuropsychiatric constitution can make him or her vulnerable to prolonged recovery from acute infections (5). Thus, even if many of the neuropsychiatric problems predate the onset of chronic fatigue, they could be viewed as contributing to the emergence of biochemical responses that perpetuate many of the syndrome's somatic features. We emphasize that we speak only for ourselves and not for the other investigators (2) who joined us in proposing a working case definition of the syndrome.

Finally, we are concerned about the interpretation of data on psychiatric illness in patients with chronic fatigue. Formal diagnostic indices of psychiatric dysfunction such as the DIS generally have been developed on the basis of findings in patients who typically are physically healthy; many neuropsychologists are leery of using these instruments to diagnose a psychiatric disorder in patients who do have or may have an underlying organic illness. Indeed, many of the instruments base the diagnosis of psychiatric disease, in part, on the presence of symptoms that could well reflect an underlying organic illness (for example, fatigue, anorexia, and weight loss).

It would be inappropriate to conclude from the data of Manu and colleagues that 129 of their 135 patients with chronic fatigue had only a primary psychiatric disorder. Indeed, with the chronic fatigue syndrome as with other illnesses, it may be more productive to avoid the kind of "mind-body" dualism that has characterized much past thinking about the pathogenesis of illness.

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Aerosolized Pentamidine

To the Editor: In their editorial on aerosolized pentamidine, Armstrong and Bernard (1) confuse two ethical responsibilities of physicians and thereby render an unethical proscription. The medical community has the responsibility to identify useful therapeutic agents and to encourage their use. The methodology of the double-blind, randomized, controlled clinical trial has passed, but in our self-flagellation for not fulfilling our collective responsibility (that is, conducting a clinical trial), we should not deprive our patients of our best clinical judgment and the best available therapy.

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Reference

Magnetic Resonance Scanning in the Sjögren Syndrome

To the Editor: Alexander and colleagues (1) recently described cerebral lesions visualized by magnetic resonance (MR) imaging in patients with the Sjögren syndrome. We caution against assuming that all MR abnormalities in cerebral white matter are due to an underlying collagen vascular disease. We report a case of the Sjögren syndrome and progressive multifocal leukoencephalopathy in an 18-year-old patient who had white matter lesions on an MR scan. These lesions were similar in appearance to some of those described by Alexander and colleagues.

A computed tomographic (CT) scan done without use of an intravenous contrast agent showed generalized atrophy and a hypodense area in the right middle cerebellar peduncle and deep white matter of the right cerebellum. An MR scan showed a linear zone of high signal on T2-weighted images that extended into the right lower mesencephalon. An area of high signal was also noted in the anterior temporal lobe. Demyelinating lesions characteristic of progressive multifocal leukoencephalopathy were visualized during the postmortem examination, and viral particles consistent with a diagnosis of JC virus were seen on transmission electron microscopy.

Progressive multifocal leukoencephalopathy is a rapidly progressive demyelinating disease that has been reported in patients with a wide spectrum of disorders of immunosuppression, including patients with systemic lupus erythematosus receiving steroid treatment (2, 3). Lesions are asymmetric and are located predominantly in the subcortical white matter, although the cerebellum and brainstem may be involved. The lesions are not related to the distribution of the cerebral blood vessels or the ventricular system.

In these patients, the CT scan characteristically shows low-density lesions in the white matter that follow the contours of the gray-white junction and infrequently reveals involvement of the cortical gray matter. Contrast enhancement or mass effect is rare. The MR scan shows multiple lesions and decreased signal intensity on T1-weighted images (long T1) and in-

References
creased signal intensity on T2-weighted images (long T2) (4, 5). The lesions may initially be round or oval, and may later become large and confluent. In our patient, the differential diagnosis also included other demyelinating conditions, infection, infection, and the vasculopathy of the Sjögren syndrome.

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Carboplatin and Renal Dysfunction

To the Editor: Carboplatin is a second-generation platinum analogue that has activity in refractory ovarian cancer similar to that of cisplatin (1). Carboplatin appears to have a spectrum of clinical toxicity different from that of cisplatin: at therapeutically equivalent doses, the dose-limiting toxicity for carboplatin is myelosuppression, with comparatively little renal or neurologic toxicity (1-3). For this reason, we have begun to study the use of high-dose carboplatin therapy (800 mg/m² body surface area - cycle) in combination with granulocyte-macrophage colony-stimulating factor (GM-CSF) in patients with refractory ovarian cancer. It is anticipated that GM-CSF may blunt carboplatin-induced bone marrow suppression, as has been observed in preliminary studies with other cytotoxic chemotherapy (4).

Unexpectedly, the first two patients treated with this regimen had decreases of 58% and 36%, respectively, in creatinine clearance after the first cycle of carboplatin chemotherapy. The third patient had a decrease in creatinine clearance of 61%. In all three patients, the carboplatin dose associated with the change in creatinine clearance was 800 mg/m², which was given as a 30-minute intravenous infusion in 250 mL of 5% dextrose in water. Hydration was not given before or after the carboplatin dose to any of the patients. The peak serum creatinine level measured in the patients did not indicate the severity of renal compromise. (Further information is available from the author upon request.) Patient 1 received gentamicin during the leukocyte nadir of cycle 1 as treatment for febrile neutropenia.

All patients subsequently treated with this regimen have been given vigorous intravenous hydration with 250 mL/h of normal saline (0.9% sodium chloride) for 3 hours before and 3 hours after carboplatin therapy. No reductions in creatinine clearance have been noted in the nine additional patients who were treated with hydration in this fashion. Each of the three patients with progressive renal compromise had pre-existing stable cisplatin-related renal dysfunction of 1 to 3 years’ duration. The other nine patients given vigorous intravenous hydration also had pre-existing stable cisplatin-related renal dysfunction of durations ranging from 6 months to 5 years.

Although cisplatin and carboplatin have different spectrums of toxicity, their mechanisms of action are the same, with the formation of identical DNA lesions. The results of preclinical studies suggest that the chronic phase of cisplatin-related renal toxicity may be related to platinum-DNA adduct formation in kidney tissue. The additional platinum-DNA adduct formed in renal tissue by carboplatin may play a role in increasing the degree of renal dysfunction.

Whatever molecular mechanism is operative, we feel that carboplatin is less nephrotoxic than cisplatin but must be administered with caution to patients who have stable cisplatin-related renal dysfunction. Carboplatin can be safely administered to such patients on an outpatient basis when vigorous normal saline hydration is given for several hours before and several hours after the actual administration of the drug.

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References

Testicular Cancer, Clinical Stage I

To the Editor: Stephens and Williamson (1) reviewed seven surveillance series and cited these experiences as a mandate for abandonment of surgical staging of testicular cancer, clinical stage I. The authors reviewed the literature and did not report their own experience. From my experience in a single series of more than 1000 cases that occurred over a 20-year period, I have found that testis tumor is still treacherous and is perhaps best staged early and accurately. If such staging can be done without any long-term morbidity, it is both prudent and rational to do so, at least for the time being, while clinical staging is still insensitive to at least the 30th percentile (2).
The authors possibly presumed that the position of urologists on node dissection in low-stage disease is fixed. This is not the case. Responsible urologic oncologists do not maintain the position that all patients with clinical stage I disease should have surgery now and forever. Yet discriminating between patients who do and patients who do not have positive nodes is still difficult. Ideally, we should have an increased sensitivity of clinical staging and a predictive validity of our clinical and primary tumor staging approaching 100%; that day is not yet here. The large collected series in Great Britain reported several times shows clearly that the potential for relapse is 32% over a 4-year period (2). The alarming feature of this study was that some patients had relapses more than 2 years later; previously, it had been assumed that patients would not have a relapse, if they had not already had one by the 24th month. Yet, 4% of patients a year continue to have relapses; thus, the relapse rate after 4 years is up to 32% from 24% at 2 years.

When clinical staging remains relatively insensitive, an appropriate intervention is nerve-sparing retroperitoneal lymph node dissection. Use of this technique would eliminate the one significant morbidity of radical retroperitoneal lymph node dissection. Virtually all patients who have had the nerve-sparing technique ejaculate after the operation (3). Chemotherapy also has long-term morbidity, not the least of which is long-term infertility in approximately 50% of patients (4). Therefore, when by-passing early pathologic staging (retroperitoneal lymph node dissection), the tradeoff may be significant long-term chemotherapy toxicity, such as permanent infertility, in a large number of patients who may require such therapy during a clinical relapse.

Overall, Stephens and Williamson did a good job of reviewing several series, but it should be noted that several more deaths have occurred in patients with relapses in some of the studies cited. From my experience with a large series of patients, I have found that testis cancer is still treacherous and is best managed early, accurately, and aggressively. Again, because such management can now be done without any long-term morbidity, it is quite rational to do modified nerve-sparing retroperitoneal lymph node dissection in clinical stage I nonseminoma.

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References

Excessive Drug Challenge

To the Editor: Sanford and colleagues (1) provided incontrovertible evidence of an association between metronidazole and attacks of acute pancreatitis. The patient developed acute pancreatitis each of the four separate times that she received the drug. Although impressive, their findings are also distressing. How did it happen that three physicians each challenged the patient with metronidazole after her first attack of pancreatitis? Was no one aware of any of the patient's previous experiences? Some explanation is in order.

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In response: We appreciated Dr. Donaldson's comments and query. The clear association of our patient's pancreatitis with metronidazole therapy was not recognized sooner for logistical reasons due to the all-too-frequent problems of fragmented care. The patient's inpatient and outpatient health care were rendered in several locations and by numerous physicians. Her case was further complicated by poor compliance and inconsistent follow-up. The association of metronidazole with her recurrent pancreatitis was made after her third episode of pancreatitis, but metronidazole was subsequently again prescribed by an unsuspecting physician in a walk-in clinic.

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Lovastatin and Visual Changes

To the Editor: Lovastatin is an effective and well-tolerated medication for patients with hypercholesterolemia (1). A potent inhibitor of the rate-limiting enzyme 3-hydroxy-3-methylglutaryl enzyme A in cholesterol synthesis, it has not been associated with cataract formation, unlike earlier investigative drugs that blocked cholesterol synthesis (2). Although new lens opacities have developed in some patients receiving lovastatin, others “lost” opacities, and drug therapy was not discontinued in any of the patients because of ocular side effects (3). We report a case of significant visual changes that occurred in a patient soon after the institution of lovastatin therapy.

A 52-year-old man had coronary artery disease, hypertension, and hypercholesterolemia. His hypertension was being treated with enalapril maleate, labetalol hydrochloride, and indapamide. He was given gemfibrozil therapy in September 1987; in early November,
his therapy was switched to lovastatin, 20 mg at bedtime.

The patient had been found to have unremarkable findings on slit-lamp biomicroscopy and a refraction in May 1987. He had amblyopia and a congenital hypertropia in the left eye. His best corrected visual acuity was 20/30 in the left eye and 20/20 in the right eye; he also had a corneal “with-the-rule” astigmatism. From 1980 to 1987, his correction for myopia had decreased by only 0.50 diopters.

On 23 December 1987, 5 weeks after he was given lovastatin therapy, the patient had another eye exam because of blurred vision, loss of acuity, and problems with bright sunlight; he denied headaches or loss of vision. His correction for myopia had decreased 0.50 diopters in the 6-month interval, and slit-lamp biomicroscopy showed the first time posterior polar yellowing and slight frosting at the anterior zone of disjunction. No other abnormalities were seen. In January 1988, the lovastatin therapy was discontinued because of the significance of the ocular findings. A repeat eye examination 6 months later showed that no findings had changed since 23 December.

The results of studies of changes in refractive errors and ophthalmologic acumen suggest that the patient’s ocular changes are abnormal (4). Because changes in refraction must be due to changes in the cornea, aqueous and vitreous humor, lens, or retina, the results of the patient’s otherwise normal ophthalmologic examination suggest that the refractive change was due to the formation of the lenticular capacities. Because these changes occurred soon after the institution of lovastatin therapy, it is difficult not to ascribe them to the medication.

Although visual disturbances are reported in 1% of patients receiving labetalol hydrochloride, and although blurred vision has been listed as a side effect in less than 5% of patients receiving indapamide and in less than 1% of patients receiving enalapril maleate, not one of these agents has been reported to cause changes in the lens or refractive index (5). (The patient previously had no problems with the agents before the institution of lovastatin therapy).

Because of the association of hypercholesterolemia with other diseases requiring drug therapy and because of the rising prevalence of “naturally” occurring cataracts in middle-aged patients, it is difficult to interpret the incidence of new opacities with lovastatin. A clinical trial in younger hypercholesterolemic persons may have to be done to resolve this question. Until then, physicians prescribing lovastatin should be aware of the potential for visual changes, they should follow the recommendations for regular ophthalmologic examinations outlined by the manufacturer, and they should report adverse findings promptly.

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References

Bladder Neuropathy in Periarteritis Nodosa

To the Editor: Neuromuscular complications of periarteritis nodosa have been well described (1); however, the occurrence of bladder paralysis has not been reported.

A 74-year-old woman was hospitalized in February 1987 for a gait disturbance and generalized weakness. Since September 1986, she had noted the absence of voiding sensation and used to urinate once daily when she thought her bladder was full. Later, voiding became long and difficult but remained painless, and she used abdominal pressure. Her symptoms progressively worsened until a severe motor deficit from mononeuropathies multiplex prompted her hospitalization. Perineal examination showed the loss of all conus reflexes and hypotonia of the anal sphincter. Periarteritis nodosa was diagnosed on the basis of the usual clinical, biologic, and neuropathologic criteria, including a biopsy specimen showing a superficial perineal nerve. The results of cystomanometry showed a hypotonic, underactive, and hyposensitive detrusor with increased compliance, resulting in indolent overdistention of the bladder. An overactive urethral closure was noted. Electromyography showed signs of denervation in the perineal muscles and an increase of the sacral-evoked potential latencies (58.4 milliseconds; normal, <44 milliseconds); the latency of the cortical-somatosensory-evoked potential obtained by stimulation of the pudendal nerve was normal.

On the sixth day of steroid therapy, her weakness, neuropathy, and dysuria had dramatically improved. Normal bladder sensation was recovered. On the seventh day, the patient died suddenly. Autopsy findings showed pancreatitis and arteritis, with fibrinoid necrosis in the kidneys. The results of a complete pathologic examination of the brain, spinal cord (with special attention to its sacral segment), cauda equina, and the available fragment of the pudendal nerve were normal. Inflammatory infiltrates were found in the right sacral plexus and in the muscular wall of the bladder; in the latter, giant cell arteritis was found and no fibrinoid necrosis. The main finding was the actual occurrence of panarteritis and fibrinoid necrosis involving the medium-sized arteries in the perivesical fat where autonomic and somatic nervous affereances ramify.

Bladder neuropathy was proved by the urodynamic pattern (2) and the electrophysiologic findings showing pudendal nerve involvement. The same pattern may occur in other peripheral neuropathies (3, 4) or in giant cell temporal arteritis (5). In our patient, the
occurrence of bladder paralysis and its dramatic improvement with steroid therapy favored a specific complication of the vasculitis. On the basis of the increased latency of the sacral-evoked potential, we expected anatomic involvement of the pudendal nerve; its normal pathologic aspect may be explained by the rapid effect of corticosteroid therapy because the urinary symptoms disappeared before the patient died.

References

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Wide QRS-Complex Tachycardia

To the Editor: The article on wide QRS-complex tachycardia by Akhtar and colleagues (1) is a welcome treatise on the electrocardiographic diagnosis of this complex and often enigmatic arrhythmia. Although I agree with most of their conclusions, I must take issue with two points. First, that “ventricular tachycardia makes up an overwhelming majority of cases” is not definitively answered by a study made up of patients referred to cardiologists experienced in electrophysiologic evaluation. There is an inherent bias in this tertiary referral that cannot be easily explained away. Although this supposition may be correct, a large trial of consecutive patients from providers of primary care would be required to answer this question. I agree, however, that it is safer to assume ventricular tachycardia as the underlying mechanism when doubt exists, and to treat patients accordingly.

Secondly, although wide complex tachycardias are very common (for example, in the coronary care unit), the finding of wide complex tachycardia in patients presenting to an emergency department who are not in full cardiac arrest is probably relatively uncommon in most primary care settings. For physicians who are less experienced at interpreting electrocardiographic findings and who see these rhythms infrequently, knowing the specific criteria for diagnosis may not be that important. Most patients with this rhythm present to an emergency room staffed by such physicians, who must provide the initial care. It would seem appropriate to manage patients according to the following three-step protocol:

1. If the patient is asymptomatic, or minimally symptomatic and hemodynamically stable, then obtain a 12-lead electrocardiogram and call an experienced electrocardiographer, or look up criteria such as those proposed by Akhtar and colleagues (1) or Wellens (2) while observing the patient. Rational therapy can then be given.

2. If the patient is hemodynamically unstable, immediate synchronized cardioversion or procainamide at a loading dose of 20 mg/min to 1 g as guided by blood pressure, QRS and QT interval, monitoring may be tried.

In this protocol, the need to remember specific criteria are minimized, and the emphasis is placed on appropriate therapy with an eye to avoiding pitfalls, such as the use of verapamil (1, 4). It seems unreasonable to expect noncardiologists to remember specific criteria for a relatively uncommon entity. Perhaps this is why misdiagnosis is so common (4).

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References

Doctor, Patient, and Family

To the Editor: Hahn and colleagues (1) on the doctor-patient-family relationship are to be commended for acknowledging and discussing the dysfunctional situations that may underlie the behavior of patients. Also important is their recognition of patients' unreasonable demands that place physicians in difficult positions. I would disagree, however, with the authors' suggestions that the primary physician draw in the family and focus on the patient's dysfunctional behavior within the family unit. The primary physician should concentrate on the patient and on the patient's responses to the demands made on him or her. For example, the pregnant diabetic patient who insists that her 14-year-old daughter give her insulin injections...
should be recognized as not responding in an acceptable fashion to a problem. The physician must address the patient's response to her diabetes. There are many options available other than referral to a family therapist, including individual psychotherapy, group therapy, or a diabetic support group.

It is also important to recognize that dysfunctional families can result from many situations: living with an alcoholic; surviving childhood physical, emotional or sexual abuse (2); or living with an abusive spouse. Patients need to trust their physician to share emotionally difficult information. If the patient's primary physician convenes the family for discussion, patient-physician trust may not develop, and therapy may be directed away from the patient's own issues.

It is disappointing that the authors do not present research data substantiating their hypothesis or discuss the outcomes of patients. The only outcome mentioned is that the patient's complaints against family members are viewed as "distorted" by the physician. Who decides whose perceptions are distorted? Is it the family members whose behavior has been identified as dysfunctional or the clinician who sees a snapshot of family dynamics? Can a clinician who "may disagree with a patient's attitude or behavior toward other family members ... maintain an atmosphere of mutual trust and assurance that enables the patient to rely on the clinician"?

The difficult patient may be involved in dysfunctional relationships within or outside of the family unit (for example, work). The physician must be able to recognize unreasonable requests from the patient. The decision by the primary physician to transgress the dyadic relationship and "call in others," however, should be made cautiously and selectively, rather than early and frequently as the authors suggest.

The opinions and assertions herein are the private views of the author and are not to be construed as reflecting the views of the Department of the Navy or the Department of Defense.

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References

Ethical Decisions in Small Hospitals

To the Editor: Niemira's article (1) on ethics committees at rural hospitals is interesting, but I fear that she misses a deeper issue. She appears to assume that the committee's role in a large institution is similar to that in a small one, although she alludes to the possibility that their functions may be different. I agree strongly with the latter point from my experience as chairman of the ethics committee of our local 54-bed district hospital for the last 5 years. Its central role is resolving conflict among the patient (or family), the physician, nurses, or, rarely, administrators. A small hospital in a relatively stable community has several characteristics that greatly facilitate conflict resolution and are different from those of an urban hospital.

First, the hospital frequently has a longstanding relationship with the patient and family, and terminal care has often been discussed before. The implications of lengthy illnesses have been discussed with the family physician, and there are fewer surprises for the family, the physician, and nurses. This relationship is especially so in the last few years because of the popularization of the durable power of attorney. Second, many persons from rural populations (at least if our community is a valid example) are not used to and frequently do not want prolonged survival under difficult or expensive circumstances. Use of the ultimate medical technology is frequently not only not expected, but also actively eschewed, and both personal and community resources generally allow more limited alternatives than in an urban setting. Also, although there are always exceptions, life and death are on the whole, closer neighbors in a rural setting, and the acceptance of death is easier, given reasonably irrevocable circumstances. Third, I have found that consultations provided in an informal "curbside" fashion provide better results than a more formal one. Rural physicians, generally tend to view their patients more autonomously than physicians in larger, multispecialty centers, and hence ethical issues are better resolved by providing educational information for thoughtful use by each individual physician or nurse.

My points might be construed almost as an 'anti-committee' position. There is some truth to this perception, because I suspect that the current popularity of "relegating" ethical decision and advising is not always healthy from the viewpoint of physician-patient interactions. At the same time, this position attempts to make the patient, nurse, and physician the actual "committee" by providing informal and frequently indirect tools and information for conflict-resolution. This kind of process, however, can probably only occur in a small rural setting, in which the background of patient-physician interaction, and the self-selected nature of the physicians and nurses who choose to be in such communities allow this kind of interaction to exist in a fairly routine manner.

In conclusion, applying our current understanding of the role of ethics committees from larger institutions to rural hospitals should not necessarily follow the same model. The general guidelines and information base for approaching ethical decision-making must be shared by all, but the manner of their application in a rural center is very different.

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Reference