has lower systemic toxicity than other amides because of its lower affinity for cardiac channels (1094). Intra-articular local anesthetics may cause chondrotoxicity; however, chondrotoxicity is worse with bupivacaine or mepivacaine. While methemoglobinemia is a major issue with prilocaine, benzocaine and lidocaine can also cause methemoglobinemia (1095,1096).

Local anesthetic toxicity affects 2 organs that inherently are less tolerant of anaerobic metabolism, the heart and brain. Cardiac toxicity is mostly related to accidental intravascular injection, leading to the conduction disturbances, contractile dysfunction, and ventricular arrhythmias that are seen in local anesthetic induced cardiac toxicity (1097). More importantly, for interventional pain physicians, the incidence of cardiac toxicity increases with bupivacaine, a longer acting anesthetic. Bupivacaine blocks inactive sodium channels during the cardiac potential at a concentration of 0.2 mcg. Bupivacaine binding is described as "fast-in, slow-out" fashion as it binds very quickly to a large portion of sodium channels during the cardiac action potential, but releases from the channel slowly during diastole, resulting in a large proportion of medication accumulating at 60 to 150 beats per minute. Local anesthetic toxicity may become a serious issue, even though adverse effects are rare. From minor symptoms to major cardiac or CNS effects, local anesthetic system toxicity is an important consequence in interventional pain management. The epidemiology of local anesthetic toxicity has been reported from zero events to 25 per 10,000 nerve blocks. One study reported seizures of 79 of 10,000 brachial plexus block procedures (1098,1099).

Lidocaine at 5 to 10 mcg/mL will also result in a substantial sodium channel blockade during cardiac action potential. However, in contrast to bupivacaine, lidocaine follows the "fast-in, fast-out" principle, meaning it releases from sodium channels rapidly during diastole. This allows for a quick recovery, and reduced incidence of cardiac toxicity even compared to bupivacaine. Consequently, during a cardiac arrest, it may be crucial to continue resuscitation measures until bupivacaine is completely released. CNS changes include agitation, confusion, dizziness, drowsiness, dysphoria, auditory changes, tinnitus, perioral numbness, metallic taste, and dysarthria. Without adequate recognition and treatment, these signs as symptoms can progress to seizures, respiratory arrest, and/or coma.

Historically, local anesthetic literature suggests that cardiac toxicity is often presented after antecedent CNS toxicity (1097). However, with more potent local anesthetics, cardiac toxicity may precede CNS toxicity. Lidocaine was utilized far more frequently than bupivacaine. Subarachnoid blockade with bupivacaine may turn out to be a disaster, specifically in the cervical spine. Consequently, injections of bupivacaine in the cervical or thoracic spine is contraindicated. Even then, lidocaine is also injected in extremely low concentrations of 0.5%. In the cervical spine, one must still be careful with appropriate visualization of the epidural space without any subdural or subarachnoid filling. Failure to follow basic principles can result in respiratory arrest, as well as cardiac arrest.

10.3 Radiation Safety

Other complications of epidural interventions include radiation exposure, when guided under fluoroscopy. This is a potential problem with damage to eyes, skin, and reproductive organs (1100-1104).

The ALARA principle should be respected when x-ray is used because excessive radiation to a patient or a physician can cause radiation injury or a stochastic effect such as neoplasm and genetic mutation (1104).

Radiation exposure used in pain procedures is related to factors such as the skill of the operator, the distance of the operator from the patient, the orientation of the operator's head, the distance of the image detector from the patient, the beam collimation, the tube configuration, the tube voltage and filtration, and the complexity of the procedure (1102).

Fortunately, most studies regarding radiation exposure during fluoroscopy-guided pain interventions have concluded that exposure levels are below the yearly limit established by the International Commission on Radiological Protection (ICRP). However, exposure to low levels of ionizing radiation over the long-term cannot be accurately predicted. These long-term, low-level ionizing radiation exposures may not acutely destroy cells, but may lead to cell damage and genetic mutations that can lead to sequelae years later (1103).

10.4 Complications of Lysis of Adhesions and Neuroplasty

Lysis of adhesion was first introduced into the lexicon of interventional pain medicine techniques in 1989 (1105). Since that introduction, the procedure has grown in acceptance and application to include epiduroscopy and transforaminal approaches to the