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THE EFFECTS OF ALCOHOL AND DRUG ABUSE
ON THE STERNAL END OF THE FOURTH RIB

by

Katherine Markham Taylor

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A Dissertation Submitted to the Faculty of the
DEPARTMENT OF ANTHROPOLOGY
In Partial Fulfillment of the Requirements
For the Degree of
DOCTOR OF PHILOSOPHY
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2000
As members of the Final Examination Committee, we certify that we have read the dissertation prepared by Katherine Markham Taylor entitled The Effects of Alcohol and Drug Abuse on the Sternal End of the Fourth Rib and recommend that it be accepted as fulfilling the dissertation requirement for the Degree of Doctor of Philosophy.

Stephen L. Zegura
Date 4-10-00

Mary Ellen Morbeck
Date 4/10/00

Walter H. Birkby

Final approval and acceptance of this dissertation is contingent upon the candidate's submission of the final copy of the dissertation to the Graduate College.

I hereby certify that I have read this dissertation prepared under my direction and recommend that it be accepted as fulfilling the dissertation requirement.

Stephen L. Zegura
Dissertation Director
Date 4-10-00
STATEMENT BY AUTHOR

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SIGNED: Katherine Mathews Taylor
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DEDICATION

In Honor of my Grandmother

Dorothy Jane Stecker

Who always said I could be anything I wanted to be
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ABSTRACT

Estimation of skeletal age at death is based on the premise that osseous tissue undergoes predictable and patterned changes through the life of the individual that can be quantified and accurately correlated with skeletal age. The utility of any method of estimating skeletal age at death is dependent on two basic principles. First, the descriptive parameters of the method must account for the range of phenotypic variation observed at the skeletal site. Second, the method must be accurate when applied to an unknown individual, regardless of the individual's unique life history. This study examines the reliability and accuracy of the sternal end of the fourth rib method for the determination of skeletal age at death and explores whether chronic substance abuse alters the pattern of change at the sternal end of the fourth rib. Additional variables considered include gender, race and the presence of thoracic disease. One hundred and fifty five sets of ribs, obtained during forensic autopsy, are examined and age at death determined in two separate trials. All antemortem data, with the exception of gender, are collected following completion of rib examination in order to prevent biasing the observer. Reliability (intra-observer error) and accuracy are computed utilizing the kappa statistic. The results suggest that the sternal end of the fourth rib is a reliable but not an accurate method of determining skeletal age at death. The variables of sex, race, and thoracic disease all influence the accuracy of the method. Chronic substance abuse appears to influence both the reliability and the accuracy of the method.
An anthropologist intensely scrutinizes the fossil casts of SK 48 and KNM-ER 406. They share the common features of a small brain case, robust nuchal and sagittal crests, a well-developed, flat dish-like face, and large posterior teeth. KNM-ER 406 appears similar to, but more robust than, SK 48. The question contemplated by the anthropologist is four-fold. Are the observed differences a reflection of species characteristics, population differences, sexual dimorphism or unique individual characteristics? The interpretation of the differences makes an enormous impact on the reconstruction of hominid phylogeny.

Conversely, consider an anthropologist that has just unearthed five anatomically modern human skeletons at the base of an ancient structure. Skeletal analysis reveals that all five individuals exhibit the unilateral non-fusion of the acromial process of the scapula. Is the trait genetically controlled thereby suggesting that the five individuals are in fact genetically related, or is the trait actually prevalent in the population reflecting a small population size and the effects of genetic drift? Perhaps the trait is not genetically determined at all and instead reflects the fact that all five individuals engaged in similar repetitive behavior during skeletal development. The chosen interpretation of the apparent prevalence of the skeletal trait potentially influences the reconstruction of the newly discovered society.

Lastly, consider the forensic anthropologist faced with an unidentified homicide victim. Law enforcement requests a biological profile of the decedent to include the race,
sex, and age at death. The anthropologist relies upon established methods and knowledge of human skeletal variation to derive what is requested by the investigating agency in an attempt to determine the identity of the victim.

Although thousands, if not millions, of years separate the subjects of analyses in these three scenarios, the underlying premise upon which the analyses are based is the same. The skeleton "mirrors our phylogeny" (Morbeck, 1997:117). The basic plan is a mammalian template, modified by evolutionary adaptations defining the order, family, genus and species, further shaped by traits reflecting population affinity and sexual dimorphism, and overlaid by individual modifications reflecting phenotypic plasticity and response to individual life history events. The challenge to the paleoanthropologist scrutinizing the australopithecines is determining which traits define the species versus the population versus the gender versus the individual. The challenge to the biological anthropologist reconstructing an ancient population is to determine the heritability of the osseous anomaly, thereby establishing if it is evidence of familial relationship or shared occupation. The goal of the forensic anthropologist is to define the individual based on established parameters of "racial" variation, sexual dimorphism, and skeletal changes correlated with advancing age.

Theoretically, the rules are established for the forensic anthropologist. There is documentation of skeletal traits occurring in higher frequency in individuals from specific geographical origin thereby defining "skeletal race". Sexual dimorphism is based on the premise that males are generally bigger than females with a larger muscle mass and therefore more robust areas of muscle attachment. In addition, females exhibit
pelvic modifications associated with child bearing. Lastly, changes in the skeleton occur at the histological as well as the morphological level as an individual ages. Some of these changes are predictable and can be categorized for use in determining skeletal age at death. But are the rules really set in stone? As gene flow increases every generation, aided in large part by modern technology and a breakdown of cultural as well as geographical boundaries, skeletal traits defining population affinity become less distinct. Modern society has produced weight lifting females, physically inactive males, and steroid treated gender converts, challenging the skeletal expression of sexual dimorphism. Disease processes, metabolic and hormonal imbalances, and biomechanical stresses theoretically have the potential to alter the pattern of skeletal aging. So how sound is the methodology?

Any method of skeletal analysis must recognize, account for, and incorporate the above-mentioned variables in order to be applicable. In other words, no methodology can survive if it is static and unchanging. Modifications must occur as new variables are identified and their contribution to phenotypic expression elucidated. The current study examines one method of determining skeletal age at death, phase analysis of the sternal end of the fourth rib, and the effects of one specific individual repetitive behavior, chronic substance abuse.

The determination of skeletal age at death from morphological changes in the skeleton is an exercise in pattern recognition. Given a specific skeletal feature, the pubic symphysis, auricular surface of the ilium or the sternal end of the fourth rib, the premise underlying the method is that the feature undergoes predictable and qualitatively
recognizable changes with time. This permits the assignment of an unknown into a defined phase, which correlates with an age range. The premise is not that every individual of a given age exhibits the exact same phenotype but rather that the range of phenotypic variation exhibited by the trait at any given age is accounted for by the descriptive parameters defining the phase. Thus, applicability of the method is dependent on the recognition of variables contributing to phenotypic variation and a grasp of the extent to which each variable alters morphology.

Variables contributing to phenotypic variation can be either genetic or environmental in origin. The pelvis for example, is recognized as an ideal location for documenting changes with age because it exhibits sexual dimorphism, plays a critical role in locomotion and parturition, and shows phenotypic plasticity (Meindl et al., 1985). In other words, the overall morphological form of the pelvis may be genetically controlled but the environmental stresses induced by locomotion and parturition find expression within the confines of phenotypic plasticity. As one ages, the mechanical stresses result in patterned changes in morphology. Thus, determination of skeletal age at death is dependent on morphological change that occurs with cumulative environmental stresses. The key, however, is to recognize, and to some extent control for, environmental variation. For example, many methods of determining skeletal age at death recognize that hormonal changes associated with parturition and menopause necessitate gender specific parameters. In addition, pathological factors, including past trauma and disease states, can be identified by skeletal analysis and consequently estimates of skeletal age at death can be tempered by the possibility of pathologically induced alterations to the patterned
change. What challenges a method however, is an individual life history characteristic that is only evidenced by an alteration of the patterned changes associated with age.

The current study seeks to analyze the accuracy and reliability of the rib method of determining skeletal age at death and to determine the effects of chronic substance abuse on the morphological indicators employed by the method. Developed by Iscan and associates (1984), the method boasts smaller age ranges than generated by other methods and accuracy and reliability comparable to methods based on the most commonly used feature, the pubic symphysis. The method incorporates gender specific parameters (Iscan et al., 1985) and modifications to account for racial variation (Iscan et al., 1987). The shape, form, texture, and overall quality of the bone at the costal chondral junction define the phases of morphological change (Iscan et al., 1985). Testing the accuracy and reliability of the method in a group of non-substance abusers tests whether or not the change with age at the sternal end of the fourth rib is in fact patterned and if the method in its current version adequately describes the variation present in each phase. Testing the accuracy and reliability of the method when applied to a sample of chronic substance abusers tests whether a repetitive behavior can alter the pattern of skeletal aging and whether such alterations are predictable.

The current study utilizes the sternal end of the fourth rib collected at autopsy at the King County Medical Examiner’s Office in Seattle, Washington. In 1998, King County recorded 234 (Reay, 1998) deaths attributed to recreational overdose. The prevalence of black tar heroin on the streets of Seattle has made understanding the effects of chronic substance abuse on skeletal indicators a paramount concern with regards to the
determination of age at death in unidentified remains. To that end, the current study begins with Chapter 2, a discussion of skeletal biology and the current methods employed for estimating age at death. Chapter 3 details the effects of chronic substance abuse focusing on the most popular drugs of abuse, alcohol, heroin, and cocaine. The chapter also includes a discussion of drug abuse in King County. Chapter 4 details the materials and methods employed for the study. Chapter 5 presents the results and Chapter 6 provides a discussion of the results, the corresponding implications, and the topics for future research.

The implications of the current study for the practice of forensic anthropology are obvious as it directly deals with the reliability of a method currently in use to determine skeletal age at death. But what implication does the current study have for the work of the biological anthropologist debating the origin of acromial non-fusion or the paleoanthropologist debating the phylogeny of the australopithecines?

The biological anthropologist in the previously described example is dealing with an osseous anomaly not the determination of skeletal age at death. However, documented examples of individual behavior resulting in skeletal alterations provide precedence for the existence of behaviorally modified skeletal traits. Perhaps this tips the scales toward an interpretation of shared occupation or shared repetitive behavior during development rather than shared genes. Likewise, the paleoanthropologist in the opening example is dealing with individuals from a population unlikely to be chronic intravenous heroin abusers. However, examples of skeletal traits expressing behavior rather than gender, population or species differences, add one more level of differentiation to the puzzle and
potentially blur the lines that differentiate species characteristics from population and gender specific traits.

The issue of the differentiation of skeletal traits and the interpretation of skeletal morphology is perhaps no better demonstrated than with the court case involving the discovery and disposition of the "Kennewick Man", a single individual from a population 9300 years old. How does one differentiate between population, gender and individual characteristics, and which traits are actually indicative of racial affinity? The legal implications are enormous.

The current study examining one method of aging skeletal remains and the effects of a single repetitive behavior may appear narrow in scope and application. However, each and every example of individual behavior altering skeletal morphology poses challenges to the principles upon which comparative skeletal analysis is based.
The adult skeleton is comprised of 206 bones that function to protect internal organs, provide a mobile lever system for muscle attachment and coordination, act as a calcium and mineral store for the body, and provide a protected area for blood cell production. As a living, dynamic tissue, bone participates in metabolic activity, is influenced by changes in the internal environment including chemical and mechanical signals, and is vulnerable to pathological conditions and homeostatic deviations. As stated by Rogers, "the skeletal system must be thought of as an organ of the body, closely related to other systems, initiated in its growth by hereditary specification but subject to the vagaries of impact by environmental forces" (Rogers, 1982:16). The varied functions of bone tissue are facilitated by the complex structural and functional organization of its components.

Skeletal Organization

Like all connective tissue, bone is made up of a cellular component and an extracellular matrix. The matrix consists of an organic component, chiefly collagenous fibers and glycoproteins, and inorganic mineral salts, predominately calcium and phosphate (Fawcett, 1994). The cellular component of bone consists of three cell types: osteoblasts, osteoclasts, and osteocytes.

Osteoblasts function in the building of bone and the synthesizing of structural proteins and growth factors (Meghji, 1992). Derived from osteoprogenitor cells present in bone, osteoblasts manufacture and secrete collagen and noncollagenous proteins that act
as a framework for the deposition of the mineral matrix. In addition, they secrete alkaline phosphatase, an enzyme involved in the process of mineralization (Rodan, 1992). Bone formation by osteoblasts is modified by systemic hormones and local factors including paracrine, which influence osteoblast number and function (Canalis, 1993).

Osteoclasts, responsible for the breakdown of bone, are derived from a mononuclear phagocyte lineage (Lassus et al., 1998). Osteoclasts initiate bone resorption by dissolving the mineral matrix and hydrolyzing the organic matrix (Dziak, 1993). Rates of resorption are controlled either by changing the number of functional osteoclasts or altering their level of function (Vaananen et al., 1988). Osteoclast activity is controlled in large part by signals from osteoblasts which act to both promote as well as inhibit bone resorption (Vitte et al., 1996).

Maintenance of bone mass and mineral content is dependent on a balance between the activity of osteoblasts and osteoclasts. During childhood and adolescence, bone undergoes modeling, a process by which bone morphology is altered to reflect growth and increasing muscle and body mass. During adult life, bone undergoes remodeling which involves the continual breakdown of old bone and the formation of new bone (Ng et al., 1997). If bone resorption exceeds bone formation, osteopenia results. Characterized by a reduction in bone tissue volume, osteopenia can result from insufficient bone accumulation during growth or by pathological conditions that disrupt the balance between bone formation and bone resorption (Frost, 1985). Osteopenia coupled with mechanical incompetence results in osteoporosis, a disorder characterized by bone
fragility, spinal deformity, and a high incidence of pathological fractures (Kiebzak, 1991).

The balance between bone formation and bone resorption is in part controlled by the stress imposed by mechanical loading. The most recent theory postulates that signals between bone cells are relayed in response to changes in the mechanical environment. Remodeling is increased when loading is reduced or elevated to excess (Martin, 2000). The most probable signaler is the third cell type, the osteocyte.

Osteocytes are mature bone cells enclosed in a protective shell, or lacuna, and surrounded by bone matrix. Osteocytes participate in metabolic activities and are the most likely candidate for the role of signal initiator in mechanotransduction (Burger et al., 1995). Essentially, mechanotransduction involves the recognition of mechanical stimuli, the initiation of a response involving biochemical signaling between the sensor and the effector, and a response by the effector in the form of bone formation or resorption. The purpose is to alter bone architecture in order to counter the imposed stress (Turner and Pavalko, 1998).

The ability of bone to sense and respond to stress is facilitated by its complex microscopic organization. In cortical bone the structural unit is the osteon, which is composed of a central Haversian canal surrounded by concentric layers of matrix referred to as lamellae. Osteocytes enclosed in lacunae are aligned in the lamellae and connected by nutrient channels known as canaliculi (Fawcett, 1994). In cross section, the osteon resembles a bullseye pattern. Cortical bone makes up approximately 80% of osseous tissue and is found predominantly in the appendicular skeleton (Sambrook et al., 1993).
Spongy bone, found predominantly in the axial skeleton, lacks Haversian systems and is instead comprised of lacunae and canaliculi surrounded by plates of matrix referred to as trabeculae. Aligned in conjunction with the direction of stress applied to bone, trabeculae provide the internal scaffolding of spongy bone (Rogers, 1982). In contrast, compact bone is denser and provides strength and mechanical support.

**Bone Maintenance**

Formation of bone begins during fetal development and continues through adulthood in the form of remodeling and repair (Ferguson *et al.*, 1998). The adult morphology and quality of bone reflect a form dictated by genetics but modified by hormonal input, metabolic activity, and environmental stresses. During growth and development, the skeletal system optimizes its architecture in order to meet the functional demands imposed by mechanical loading (Turner and Pavalko, 1998). Maintenance of skeletal tissue is dependent on the ability to sense and respond to mechanical stresses thereby adapting to changing loads and functional demands. The drive to maintain skeletal mass in order to respond to functional demands is challenged by the decrease in bone mineral density that occurs with aging.

Peak bone mass, defined as the amount of bony tissue present at the end of skeletal maturation (Bonjour *et al.*, 1994), is achieved in late adolescence. Annual increases in bone mineral density are most marked in females at the age of menarche, approximately 11-13 years, and in males between the ages of 13 and 17 years (Kroger *et al.*, 1993). The attainment of peak bone mass is earlier in females than males presumably because of an earlier onset of puberty (Lu *et al.*, 1994). The bone mineral levels
associated with peak bone mass are thought to be largely under genetic control but influenced by dietary habits, including calcium intake, and degree of physical activity (Sambrook et al., 1993). Loss of bone density following the attainment of peak bone mass is site specific and occurs in both sexes, with a rapid loss in women associated with the onset of menopause (Sambrook et al., 1993).

Bone mineral density and modification of skeletal morphology are dictated by a form/function relationship. As a dynamic biological system, bone is capable of adapting to the mechanical stresses imposed by daily activities as well as pathological conditions (Katz, 1980). Adaptation occurs through bone remodeling, a combination of bone resorption and bone formation. The goal of adaptation is to maintain an adequate structure to meet functional demands balanced against the cost of excessive bone mass on mobility (Turner, 1991).

Physical Activity

Bone mineral levels of individuals with varying degrees of physical activity perhaps best exhibit the response of bone to changing physical stresses. Young athletes participating in load bearing sports represent an extreme in levels of exercise. When compared to age-matched controls, statistically significant increases in bone mineral content are documented in athletes including female volleyball players (Alfredson et al., 1997), female soccer players (Alfredson et al., 1996; Duppe et al., 1996), female gymnasts (Nichols et al, 1994; Kirchner et al., 1996; Dyson et al., 1997), female tennis players (Haapasalo et al., 1998), male and female college rowers (Cohen et al., 1995), elite junior weight lifters (Conroy et al., 1993), and power lifters (Tsuzuku et al., 1998). In
contrast, swimming, a non-weight bearing activity, is found not to induce osteogenesis in elite female and male athletes (Taaffe \textit{et al}., 1995; Taaffe and Marcus, 1999).

In general, exercise is shown to be positively correlated with bone mineral density in males (Snow-Harter \textit{et al}., 1992), young females (Madsen \textit{et al}., 1998), and premenopausal women (Alekel \textit{et al}., 1995; Dornemann \textit{et al}., 1997). The effects of exercise on the bone mineral content of postmenopausal women are debated. An increase in bone density in response to exercise is documented in older women (Shimegi \textit{et al}., 1994). However, contrary results include a reported difference in bone response between premenopausal and postmenopausal women to the same exercise regime (Bassey \textit{et al}., 1998), an increase in muscle mass but not bone mineral density in postmenopausal resistance trainers (Pruitt \textit{et al}., 1995), and the general consensus that physical activity increases muscle mass but is not sufficient stimulus to prevent the loss of bone with aging (Ryan and Elahi, 1998).

At the opposite extreme from athletes are those individuals limited in physical activity because of immobilization or paralysis. Acute immobilization is associated with a rapid loss of bone (Bauman \textit{et al}., 1999) and bone mass is reduced on the hemiplegic side of stroke patients (Takamoto \textit{et al}., 1995; Sato \textit{et al}., 1998). Spinal cord injured patients exhibit a significant loss of bone in the hip but no significant decrease in spinal bone mineral density (Leslie and Nance, 1993; Goemaere \textit{et al}., 1994; Szollar \textit{et al}., 1997; Szollar \textit{et al}., 1998). Likewise, following pediatric spinal cord injury bone mineral density is reduced in the femoral region but preserved in the lumbar spine (Kannisto \textit{et al}., 1998). Most bone loss occurs below the pelvis in both paraplegic and quadriplegic patients
(Garland et al., 1992). Likewise, bone loss in boys with Duchenne muscular dystrophy is most marked in the lower extremities. Bone density in the spine is slightly decreased when the boys are ambulatory but decreases significantly as the disease progresses and ambulation is reduced (Larson and Henderson, 2000).

The increase in bone mass in response to load bearing exercise and the decrease in bone mass in the absence of dynamic mechanical stress support the premise that skeletal tissue adapts to its mechanical environment. However, hormonal factors can magnify, limit, or impede the response of skeletal adaptation.

**Hormones**

The three most prominent hormones associated with bone physiology are the ones responsible for calcium homeostasis: parathyroid hormone, vitamin D hormone, and calcitonin. Additional hormones exerting influence over skeletal tissue include thyroid hormone, growth hormone, insulin, the glucocorticoids, reproductive hormones, and a variety of paracrine and autocrine hormones.

Parathyroid hormone is secreted in response to decreased serum calcium levels. Osteoclast activity increases under the influence of parathyroid hormone, however the hormone has little direct influence over the bone resorbing cells (McSheehy and Chambers, 1986a). Instead, parathyroid hormone targets osteoblasts which in turn stimulate osteoclastic bone resorption (McSheehy and Chambers, 1986b). Hyperparathyroidism is characterized by increased bone mineral loss, especially dramatic in postmenopausal women (Guo et al., 1996). Parathyroid hormone also acts to increase the renal tubular reabsorption of calcium and the renal excretion of phosphate ions.
Decreasing the serum concentration of phosphate ions prevents osseous mineral deposition and maintains serum concentrations of calcium. Lastly, parathyroid hormone influences the biosynthesis of vitamin D, a hormone necessary for the intestinal absorption of calcium (Sampson, 1997).

The metabolic pathway for the biosynthesis of vitamin D begins in the skin with a vitamin D precursor, 7-dehydrocholesterol. When exposed to sunlight, the precursor is converted into cholecalciferol, which travels via transport molecules to the liver where it is converted into 25-hydroxycholecalciferol. The vitamin D metabolite is either stored or converted in the kidney to 1,25-dihydroxycholecalciferol. Calcium absorption across the intestinal epithelium is accomplished by a calcium binding protein stimulated by 1,25-dihydroxycholecalciferol (Hadley, 1992). Like parathyroid hormone, 1,25-dihydroxycholecalciferol stimulates bone resorption through primary action on osteoblastic cells (McSheehy and Chambers, 1987). Vitamin D insufficiency decreases intestinal absorption of calcium and results in hyperparathyroidism, which in turn increases bone resorption (Adams et al., 1999).

The final hormone involved directly in calcium homeostasis is calcitonin. Synthesized by the C-cells of the thyroid gland, calcitonin is secreted in response to elevated plasma levels of calcium ion. Calcitonin functions as an inhibitor of osteoclastic bone resorption (Sampson, 1997). As a treatment for osteoporosis, nasal calcitonin therapy is documented to increase axial and appendicular bone density (Peichl et al., 1999).
Additional systemic hormones influencing skeletal homeostasis include thyroid hormones, growth hormone, insulin, and steroid hormones. Thyroid hormones increase bone turnover (Britto et al., 1994) and when hypersecreted lead to bone mineral depletion and osteoporosis (Jodar Gimeno et al., 1997; Siddiqi et al., 1997; Nuzzo et al., 1998; Olkawa et al., 1999).

Growth hormone, secreted from the pituitary gland, influences osteoblast function and growth (Slootweg, 1993), and stimulates bone turnover (Finkenstedt et al., 1997). When hypersecreted, growth hormone results in increased osteoblast and osteoclast activity (Ezzat et al., 1993). The effect on bone mineral content is debatable however, as studies on acromegalic patients have demonstrated both increased (Lesse et al., 1998) as well as normal (Ho et al., 1992) spine and femoral bone mineral density. Hyposecretion of growth hormone is associated with osteopenia in both children and adults (Bachrach et al., 1998; Kotzmann et al., 1998), and the decrease in growth hormone that occurs naturally with advancing age is correlated with a reduction in bone turnover (Toogood et al., 1997).

Insulin, secreted from the beta cells of the pancreas, is involved in growth and anabolic metabolism. Patients with insulin dependent diabetes mellitus (type I diabetes) have reduced bone mineral density and a higher incidence of osteopenia and osteoporosis when compared to healthy controls (Miazgowski and Czekalski, 1998). However, a decrease in bone mineral density does not appear to be associated with type II, non-insulin dependent diabetes (Sosa et al., 1996; Isaia et al., 1999). Osteopenia associated with type I diabetes may result from a disruption to the hormone axis controlling calcium
homeostasis (Hampson et al., 1998) or it may be associated with bone compromise due to microvascular complications (Munoz-Torres et al., 1996).

The steroid hormones include the glucocorticoids and the reproductive hormones. The glucocorticoids, secreted from the adrenal cortex and involved in a variety of metabolic processes, have negative effects on bone when secreted in excess or for extended periods of time. Patients suffering from Cushing's syndrome, characterized by hypersecretion of glucocorticoids, exhibit reduced osteoblast function, increased bone resorption, and reduced bone mineral density (Chiodini et al., 1998; Godang et al., 1999). Prolonged, high dose corticosteroid therapy is associated with an increased risk of osteoporotic fracture (Sambrook et al., 1990) and glucocorticoid-induced osteoporosis is identified as the second most common form of osteoporosis (Lindsay, 1999).

Reproductive hormones, including the estrogens and androgens, are also steroid hormones. Both androgen and estrogen concentrations are associated with bone loss in perimenopausal women (Johnston and Slemenda, 1995). Estrogen is recognized as a key component in bone maintenance as evidenced by the presence of estrogen receptors on both osteoblasts and osteoclasts (Oursler et al., 1993). Reduction in the serum levels of estradiol is identified as a major factor in the postmenopausal decrease in bone mineral content (Ohta et al., 1993) and estrogen deficiency is stated to be the main causal factor in postmenopausal (type I) osteoporosis (Smith, 1993). The importance of the reproductive hormones is further evidenced by the success of hormone replacement therapy in maintaining bone mineral density at the lumbar spine and hip in postmenopausal women (George et al., 1999). In addition, there are documented cases of decreased axial and
appendicular bone mineral levels in young, amenorrheic athletes who experience bone loss in weight-bearing bone despite the apparent benefits of enhanced physical activity (Myburgh et al., 1993; Rencken et al., 1996).

In men, both estrogen and androgen play an important role in the maintenance of bone mineral density (Mizunuma et al., 1998). For men undergoing androgen deprivation therapy as treatment for prostate cancer, the duration of therapy is correlated with a loss in bone mineral density (Wei et al., 1999). Testosterone levels also are demonstrated to correlate with bone mineral density in men (Choi et al., 1995). In general, hypogonadism is considered a risk factor for the development of osteoporosis in males (Daniell et al., 2000).

In addition to the systemic hormones, a number of paracrine and autocrine hormones are important in the maintenance of skeletal tissue. Insulin-like growth factor I (IGF-I), secreted as a systemic hormone from the liver or as a paracrine from bone cells, is a proposed mediator in the balance between bone formation and bone resorption (Hayden et al., 1995). Serum levels of IGF-I are positively associated with bone mineral density (Vestergaard et al., 1999) and low levels have been detected in young men diagnosed with osteoporosis (Johansson et al., 1992). Cytokines including interleukin-1, interleukin 4, interleukin 6, tumor necrosis factor, gamma-interferon, and transforming factor-beta are demonstrated to influence osteoclast activity (Roodman, 1993).

Prostaglandins, another category of paracrines, are postulated to inhibit (Chambers, 1988) as well as stimulate (Raisz et al., 1993) osteoclast activity.
Diet and Body Mass

In addition to the stresses of mechanical loading and the influence of systemic and local hormones, skeletal integrity is impacted by metabolic factors. The maintenance of calcium homeostasis, for example, is dependent on an adequate dietary intake of calcium. Because calcium is necessary for a variety of life essential processes, including cardiac function and nerve impulse transmission, an inadequate dietary intake forces the body to liberate calcium from bone. Consequently, calcium and vitamin D supplementation, in addition to hormone replacement therapy, is used to combat bone loss in postmenopausal women (Power et al., 1999). Calcium supplementation in young females is demonstrated to have a positive influence on bone mineral density in the spine (Nowson et al., 1997) and consumption of the daily recommended allowance of the mineral is associated with increased bone density at the spine and wrist in middle aged men (Bendavid et al., 1996). However, the benefits of dietary calcium intake are tempered by lifestyle factors as intestinal absorption decreases when activity levels decline (Branca, 1999).

Skeletal health is also impacted by intestinal malabsorption disorders reflecting the integration of the skeletal system with other body systems. Bone loss is documented in inflammatory bowel disease (Pollak et al., 1998), Crohn’s disease (Robinson et al., 1998), Whipple’s disease (Di Stefano et al., 1998) and celiac disease (Gonzalez et al., 1995). Possible causal factors include corticosteroid treatment, disturbances in calcium homeostasis (Bjarnason et al., 1997), reduced levels of vitamin D, or low body weight (Kemppainen et al., 1999).
Body weight history is also identified as the most important predictor of osteoporosis associated with anorexia nervosa (Hotta et al., 1998). In anorexic patients, bone resorption is increased without a matching increase in bone formation (Lennkh et al., 1999). Malnutrition is also a characteristic of cystic fibrosis and, consequently, patients suffering from the disease have decreased bone density when compared to healthy controls. (Gibbens et al., 1988; Grey et al., 1993; Haworth et al., 1999). In contrast, obesity is positively correlated with bone mineral density, particularly in postmenopausal women (Kin et al., 1991).

**Genetics**

The complex interactions between lifestyle, metabolic factors, and hormonal activity are further complicated by the interaction of each with the genetic parameters dictating skeletal phenotype. Research into the underlying genetics of the skeletal system is largely motivated by the drive to define the etiology of osteoporosis. Currently as much as 70% of the variation in bone phenotype is attributed to genetic variation (Eisman, 1999).

One of the first skeletal loci identified is for the vitamin D receptor (VDR) gene. Allelic variations in the gene are correlated with bone density levels (Spector et al., 1995; Lazaretti-Castro et al., 1997; Fujita et al., 1999) and a start codon polymorphism appears to influence peak bone mass (Harris et al., 1997). Other allelic variations correlated with bone density are associated with the genes for the estrogen receptor, insulin-like growth factor-I pathway, interleukin-4 and -6, the interleukin-1 receptor antagonist, calcitonin and the parathyroid receptors (Eisman, 1999).
In general, genetic contribution is identified for bone formation (Kelly et al., 1991), bone mineral density (Pocock et al., 1987; Kelly et al., 1993; Harris et al., 1998), lean body mass, and fat mass (Nguyen et al., 1998). Recognition of the genetic component of skeletal phenotype motivates the assertion in clinical circles that the presence of osteoporosis in a first degree relative is a significant risk factor for the development of the disorder (Yeap et al., 1998).

Clearly there is a genetic contribution to skeletal phenotype. However, the complexity of the interactions between genotype, environmental factors, hormonal and metabolic variables, and lifestyle choices confound efforts to define the etiology of aging bone and associated disorders such as osteoporosis. Researchers urge caution in assigning significance to individual variables or accepting heritability estimates (Slemenda et al., 1991; Hopper et al., 1998).

Aging Bone

Changes in bone quality occur with aging in both males and females. In addition to a decline in bone mineral density, aging bone undergoes a reduction in stiffness and strength, becomes more brittle, and fractures more easily (Martin, 1993). Metabolically, there is a proposed uncoupling of the normal process of bone formation and resorption (Kiebzak, 1991). Hormone levels change as parathyroid hormone secretion increases while secretion of growth hormone and vitamin D hormone decreases. Reproductive hormone levels also decrease with the most significant change occurring in women with the onset of menopause (Nuti et al., 1995). In addition, many elderly individuals experience a decrease in physical activity and dietary insufficiencies (Kiebzak, 1991).
Microscopically, senescent bone exhibits an imbalance in mineralization with a high number of poorly mineralized osteons as well as osteons with hypermineralized lamellae (Nyssen-Behets et al., 1997). Ortner (1975) contends that the increase in the frequency of forming osteons, a decrease in the number of complete osteons, and the variability in the extent of mineralization with age suggest that the underlying etiology is in fact a decrease in the rate of bone formation rather than an increase in the rate of resorption. This hypothesis is supported by cell studies that suggest that both bone precursor cells and osteoblasts undergo age-related changes that affect the proliferative capacity of the cells (Kassem et al., 1997; Long et al., 1999).

Bone quality, and in particular bone density, are directly related to the risk of fracture (Fatayerji et al., 1999). The reduced quality of senescent bone is reflected by the fact that 90% of hip fractures occur in patients over the age of 70 (Dresner-Pollak et al., 1996). In addition, osteoporosis is the most common metabolic bone disorder and a major concern to the world health care community (McGowan, 1993).

In addition to a reduction in bone quality with aging, the skeletal system is impacted by an age-associated decrease in muscle volume and mass (Porter et al., 1995; Evans and Cyr-Campbell, 1997). The resulting loss in muscle strength is implicated in a reduction in load stresses that shifts bone into a disuse mode and stimulates the age-related loss of bone (Frost, 1999). Recent research suggests that senescent bone cells actually may be less responsive to the biomechanical stimulus of heightened physical activity (Stanford et al., 2000).
The biomechanical, hormonal and cellular changes associated with aging translate into the general reduction in bone quality with aging. The desire to establish methods of determining skeletal age at death, however, has necessitated the translation of aging trends into patterns of skeletal change that can be quantified and correlated with age at death.

Determining Skeletal Age at Death

Determination of skeletal age at death is a key component of archaeological as well as forensic analysis. For archaeological endeavors, age at death estimations are relevant to the demographic analysis of sites and the corresponding models derived from such data. In forensic investigations, estimating the age at death of a John or Jane Doe facilitates relevant comparisons to lists of missing persons.

Estimation of skeletal age at death is based on the premise that osseous tissue undergoes predictable and patterned changes through the life of the individual that can be quantified and accurately correlated with skeletal age. An essential premise is that the range of observed changes are accounted for by the descriptive parameters of the method. Given that in a majority of cases the identity of the individual under examination is unknown or unconfirmed at the time of analysis, estimation of age at death must be independent of variables reflecting the individual’s health, behavior, medical history or lifestyle choices. Additionally, differences in the pattern of aging which are sex or population specific must be identified and accounted for by the method in order to render the technique broadly applicable. The usefulness of any method for the determination of
skeletal age at death is also dependent on the ease with which it can be applied to skeletal material and the reliability as well as the accuracy of the method.

Given that skeletal remains in both an archaeological and forensic context are often incomplete, the need for multiple methods utilizing different regions of the skeleton has influenced anthropological research with regards to age at death estimations. Currently methods in use by anthropologists include histological examination of osseous tissue, radiographic techniques, and gross morphological analysis of skeletal features. Anthropological research continues to refine current methods and develop new ones, as no method yet developed is universally applicable or immune to criticism.

The method utilized in the current study is a relatively new method that offers narrow estimated age ranges and confidence intervals equivalent to the methods most commonly in use. Understanding the appeal of the method requires a discussion of the other methods currently utilized by anthropologists to determine skeletal age at death. The methods for aging sub-adults are numerous and generally more accurate than those available for aging adults. Only methods used for aging individuals 16 years or older will be considered in the discussion that follows.

**Histological Methods**

In an effort to find a method of determining skeletal age at death that provides high accuracy and precision and is not based on subjective analysis, researchers turned to the microscopic analysis of osseous tissue and teeth.
Bone

Kerley (1965) was the first to devise a method of determining skeletal age at death based on histological changes that occur with advancing skeletal age. His method utilizes ground cross-sections of the femora, tibiae, and fibulae and involves the counting of complete osteons, fragmentary osteons, and non-Haversian canals in four separate microscopic fields. He reports standard errors ranging from five to 13 years.

Ahlqvist and Damsten (1969) propose a slightly different method based on the reported difficulty in distinguishing between intact and fragmentary osteons. Based instead on percent osteonal bone, the method of Ahlqvist and Damsten has the reported additional advantage of repositioning the fields in order to avoid the linea aspera of the femur, an area thought to be influenced by additional, non-age related variables.

The two methods have been tested against each other (Bouvier and Ubelaker, 1977; Stout and Gehlert, 1980) and found to be comparable in reliability, but with Kerley’s method superior in overall accuracy. The use of total osteon counts rather than percentage of osteonal bone is supported as the variable of choice in histological estimation of age at death (Stout and Stanley, 1991). Kerley’s method has been refined by Kerley and Ubelaker (1978) to include a correction factor for different microscopic field sizes. Despite the correction factor, accuracy is reported to be greatest when a field size similar to that used by Kerley is employed (Stout and Gehlert, 1982).

A third method is reported by Singh and Gunberg (1970) which utilizes stained and decalcified sections of the anterior midshaft of the femur and tibia. Despite a claimed
accuracy of within six years in 95% of the males examined, researchers have found the method prone to considerable error (Stout and Gehlert, 1980).

Recognizing that established histological methods require a complete cross sectioning of bone, more recent research focuses on the possible derivation of a less destructive and therefore more applicable method of microscopic estimation of age at death. Thompson (1979) proposes a method that utilizes a small core of bone with associated regression formulae for the humerus, ulna, femur and tibia. Likewise, Ericksen (1991) provides regression formulae for osteon counts from thin sections of the anterior cortex of the femur. The use of partial sections is supported by recent research which reports that sub-areas as small as 15% predict up to 95% of the variation in total osteon density in an anterior mid-diaphyseal femoral section (Iwaniec et al., 1998).

In order to avoid the possible biomechanical influence of weight bearing on osteon remodeling in the lower extremities, Stout and Paine (1992) have examined the histology of the rib and clavicle. They report good correlation between age at death and the histology of each bone individually. However, the best overall accuracy and reliability are reportedly accomplished with a formula utilizing the microscopic examination of both bones together. They assert that the use of the rib and clavicle is beneficial in cases in which long bones are not available or in which long bones are damaged by poor preservation, animal activity, or malicious intent. A similar attempt to chronicle microscopic age changes in the occipital bone yields random variation too high to be of use in the accurate estimation of age at death (Cool et al., 1995).
Although reported to be both reliable and accurate, methods of determining skeletal age at death by microscopic examination of osseous tissue are hampered by the complexity of the procedure, the equipment required, and the necessary time expenditure. In addition, Stout (1988) points out the number of environmental and genetic factors that influence osteonal remodeling. The list includes, but is not limited to, hormonal effects, electrolyte, metabolic and genetic functional disorders, toxic agents, radiation damage, regional trauma, paralysis, mechanical usage, nutrition, drugs, and genetic structural disorders. He asserts that the intent of pointing out such variables is to introduce a measure of caution when employing histological aging methods.

**Teeth**

In addition to bone tissue, researchers have looked at microscopic changes in teeth in an effort to find and quantify changes that progress with age. During the 1980's a great deal of research effort was expended on developing a forensically applicable method of determining age at death from cementum annulation. First described by Stott and coworkers (1982), the method involves the counting of incremental cementum layers allegedly laid down on the root of the tooth in a predictable temporal sequence. The layers appear microscopically as alternating light and dark bands (Condon et al., 1986) which can be counted. The mean count of the root section is added to the number of years from birth to the eruption of the tooth in question to obtain an estimated age at death (Naylor et al., 1985). The method is reported to be more accurate than methods utilizing morphological changes at the pubic symphysis and sacro-iliac joint (Charles et al., 1986).
The method is not immune to criticism, however. Miller and colleagues (1988) asserts that although the method is potentially useful in wildlife studies it is without validity in human applications because humans lack seasonal variations in nutrition and metabolism and have a life expectancy longer than the wild animals thus far studied. Condon and colleagues (1986) point out that although tooth decay may not affect the root it may create pain that subsequently alters the use of the tooth. The change in the mechanical environment may alter the process of cementogenesis and thus negatively impact estimations of age at death obtained from incremental counts.

Regardless of its validity, use of the method is restricted by the skill required to master the technique, the equipment required to prepare a root section, and the numerous steps involved in its application.

**Radiographic Methods**

In contrast to histological methods, radiographic methods of determining skeletal age at death are non-destructive and only require access to x-ray equipment, a standard feature in most autopsy facilities. In addition, radiographs provide a record of skeletal morphology that can be filed indefinitely for future use. Attempts to utilize radiographs to determine age at death focus primarily on the teeth, clavicle, long bones, and the bones of the anterior chest wall.

*Teeth*

Because comparative dental radiography is a frequently employed method of establishing positive identification in a forensic setting, the possibility that dental radiography might also provide a means of determining skeletal age at death is an
enticing notion. Prapanpoch and colleagues (1992) explore the possibility that the degree of pulp chamber recession can be positively correlated with advancing age. They find that genetic background and environmental factors such as diet introduce too much variability relevant to pulp chamber recession. Consequently, although the pulp chamber does recede with age, the degree of recession cannot be quantitatively correlated with age at death.

*Long Bones*

Walker and Lovejoy (1985) examined and visually seriated the radiographs of the proximal femur, proximal humerus, clavicle and calcaneus of 130 individuals based on the degree of trabecular involution. They report that the clavicle has the highest rank order correlation to age in both sexes. Males have a higher correlation than females at all sites studied. Interestingly, they find that anatomically adjacent sites, the distal clavicle and proximal humerus for example, often yield vastly different results with regards to relative trabecular involution. Although unlikely to be highly useful in the aging of single individuals, seriating by relative radiolucency can be useful in the aging of populations for demographic purposes.

*Anterior Chest Plate*

Radiographic observation of morphologic changes in the anterior chest plate is a recognized indicator of skeletal age at death. McCormick (1980) points out that mineralization of the costal cartilages, viewed radiographically, allows for a broad estimation of age based on the premise that the degree of mineralization increases with increasing age. He states, however, that this method does not provide the precision
possible with other methods of determining skeletal age at death and should thus be used as a "screening" method, or when a broad estimation is all that is required.

In a later publication, McCormick and Stewart (1988) report on the examination of the plastron, or chest plate, of 1,965 cadavers. The variables studied include the location, pattern, density and extent of costal cartilage ossification, the amount of degenerative changes in the sternal head of the clavicles, the contour of the costomanubrial junction, and the cupping of the sternal rib ends. They find that the first rib ossifies in a unique pattern and at a unique rate relative to the remainder of the rib cage. They also document sex-specific patterns in the ossification of the costal cartilages of ribs two through seven. In addition, the rib ends become progressively more "cupped" as the depth of the depression at the costal cartilage junction increases with age. Changes in the costomanubrial border progress from a smooth, slightly concave border in the teens to a distinctively irregular border at ages in excess of 35. Degenerative, osteoarthritic changes are noted with progressing age at the sternal end of the clavicle. The authors assemble an aging method utilizing all the variables that can be evaluated from a plastron roentgenogram and assert that the technique is useful in the determination of skeletal age at death on modern, autopsy specimens.

A similar study (Barres et al., 1989) uses chest radiographs to evaluate bone demineralization, fusion of the pieces of the manubrium, rib-to-cartilage attachment changes, cartilage mineralization, and cartilage-to-sternum attachment changes. The authors find that for males, the three variables with the highest correlation to age are cartilage-to-sternum attachment, rib-to-cartilage attachment, and sternal fusion. For
females, the two variables that appear to be the best predictors of age are bone
demineralization and cartilage mineralization. Noteworthy, however, is the fact that the
female sample is smaller (10 versus 41) and has a higher mean age at death (45 versus 31
years). A regression equation derived from the data reportedly provides an accuracy of
age estimation within nine years and is “faster and simpler” than macroscopic methods of
determining skeletal age at death.

**Gross Morphological Change**

The use of morphological change as an indicator of age at death is a qualitative
method that relies on pattern recognition. Based on the premise that certain regions of the
skeleton undergo predictable changes with time, methods utilizing gross morphological
change depend on the recognition of a sequence of changes that can be divided into
phases and correlated with age at death. Regions of the skeleton studied for age related
morphological change in the adult include the epiphyses of the medial clavicle and iliac
crest, the dentition, cranial and maxillary sutures, the clavicle and sternum, the pubic
symphysis, sacro-iliac joint, and the sternal end of the fourth ribs.

**Epiphyseal Closure**

One of the most obvious patterns of change associated with age is the timing of
epiphyseal closure. Initially documented by Stevenson (1924), the timing of epiphyseal
closure is used primarily for the aging of juveniles. However, the iliac crest and median
clavicle remain patent until the early to mid-twenties and are thus valuable for the aging
of young adults. The timing of epiphyseal closure at both sites has been re-evaluated by
Webb and Suchey (1985) under the assumption that cultural changes including oral
contraceptives for women and vitamin additives in food, may have altered the progress of epiphyseal union thereby rendering the older standards inaccurate.

By analyzing 605 males and 254 females aged 11 to 40 years at death, Webb and Suchey (1985) find that females show more variability in the timing of epiphyseal closure than males and consequently, the authors provide broader age ranges than reported in previous studies. Their research design includes recording information on height, weight, nutritional status, physical affliction, dysfunction, and disease with the recognition that such variables may affect bone development and consequently negatively impact correlation with age at death.

Dentition

Gustafson (1950) documents six changes in the dentition associated with advancing age: attrition, periodontosis, deposition of secondary dentin, cemental apposition, root resorption, and root transparency. Maples (1978) reports that multiple regression analyses with each of the six variables reveals that root transparency is the most reliable. Drusini and colleagues (1991) report that as age increases the dentinal tubules become occluded by minerals. When the refraction indices of the dentin are equivalent to those of the surrounding tissues, the dentin becomes invisible to transmitted light. They compare two methods for measuring dentine transparency, use of calipers versus use of a computerized densitometric analyzer, and find both methods equally applicable with a reasonable degree of accuracy. They conclude that root dentin transparency is an age dependent variable that can be used to generate broad estimates of age at death in recent and historical samples.
Perhaps the most obvious change in the dentition with age is dental attrition, or wear. Presumably, an individual with excessive molar wear is older than an individual with minimal wear. This assumption, however, fails to take into account possible differences in the forces causing the wear. This fact is demonstrated by the work of Molnar (1971) on the skeletal remains of Native Americans from three distinct areas: California, the Southwest United States and the Valley of Mexico. Molnar documents significant differences in the type and degree of wear with positive correlation between tooth wear and cultural factors.

Additional factors influencing dental wear include individual habits and chewing patterns. Use of dental wear to assess age at death assumes that dental wear in continuous during the functional life of the tooth and that the wear and diet are effectively uniform within the population (Nowell, 1978). Consequently, the method is of little use in a forensic setting. The use of dental wear, although not applicable to the determination of age at death of a single individual, can be used systematically on skeletal populations to provide relative age at death estimations (Lovejoy, 1985).

*Cranial and Maxillary Sutures*

One of the first researchers to document the use of cranial suture closure for the determination of skeletal age at death was Dwight (1890). Later, Todd and Lyon (1924) found human variability in the timing of suture closure too excessive to be useful in the determination of age at death, a statement reiterated by McKern and Stewart (1957). The method has been refined, and the accuracy improved, by Meindl and Lovejoy (1985). These authors report on a study of 236 crania in the Hamann-Todd collection and the
finding that a high correlation exists between suture closure and age at death that is independent of sex and race.

Application of the method requires the analysis of 10 separate sites on the cranium. The sites are evaluated for the degree of suture closure with scores ranging from 0 for completely patent to 3 for completely obliterated. The sum of the scores is used to determine the estimated age at death. Although relatively easy to apply, the method is dependent on subjective analysis. In addition, the weathering of the cranium and associated exfoliation can prevent an accurate interpretation of the degree of suture closure.

In a similar approach, Mann and colleagues (1987) report on the analysis of 36 maxillae from individuals aged 13-79 years at death. They utilize all four sutures of the superior palate, the incisive, anterior median palatine, transverse palatine and posterior median palatine, and assign a score from 0 to 4 based on the percentage of suture obliteration. Although variability in suture obliteration between individuals is documented, they find that the general pattern of closure is consistent with broad age categories. A later study (Mann et al., 1991) documents sexual dimorphism in the timing and pattern of maxillary suture closure, and reiterates that the method is useful for broad age determinations designated as child, adolescent, young, middle-aged, and old adult.

Gruspier and Mullen (1991) have tested the Mann method on 83 male maxillae. They report extreme inaccuracy in the predicted ages reflecting a non-linear relationship between age and total suture closure. They further point out that the assumption that
quantitative methods are somehow more “scientific” and accurate than qualitative methods for the determination of skeletal age at death is dangerous and misleading.

**Clavicle and Sternum**

Given that the radiographic changes in the clavicle are correlated with age at death (Walker and Lovejoy, 1985), the possibility that gross morphological changes exist that also correlate with age is a valid possibility. Kaur and Jit (1990) analyzed the cortical index, defined as the proportion of cortical thickness to total diameter of the bone. They utilized sections of the clavicle, some cut horizontally and some cut parasagitally. They report that the index increases between the ages of 15-30 years, sharply decreases between the ages of 31 and 40 in both sexes, and then continues a sharp decrease after age 40 in females and a moderate decrease in males. The authors present cortical indexes for males and females for different age groups and assert the usefulness of the method in a forensic context.

Morphological changes in the sternum are also documented radiographically (McCormick and Stewart, 1988). Xiao and colleagues (1987) have derived a method of age determination based on the observed degree of change in morphological characteristics of the male sternum. Sun and colleagues (1995) have derived a similar method for females. Although reported to be “simple and accurate”, the methods utilize a regression formula with multiple variables, some of which are subjective in nature.

**Pubic Symphysis**

The anatomical feature most often used for the determination of skeletal age at death is the pubic symphyseal face (Meindl *et al.*, 1985). A cartilaginous joint separated
by a pad of fibrocartilage, the pubic symphysis permits little or no motion under normal conditions. During pregnancy, however, the joint becomes more mobile under the influence of reproductive hormones. Additionally, the effects of late term pregnancy on the area surrounding the joint is evidenced by scars of parturition, or birth scars, that appear as pitting on the dorsal surface of the pubic bone (Angel, 1969). Clearly, the functional adaptations of the pubic symphysis associated with child bearing suggest that age-correlated morphological change at the joint is likely to be sexually dimorphic.

Todd was the first researcher to devise a method of age at death estimation based on morphological change at the pubic symphysis for males (Todd, 1920) and for females (Todd, 1921). The method is applicable to individuals from 18 to 50+ years of age at the time of death. Todd’s method involves evaluating nine morphological features or areas of the pubic symphyseal face that change with age and placing the pubic symphysis into one of 10 phases. The sequential phases correspond to the predictable, age-correlated changes that occur at the joint.

McKem and Stewart (1957) published a new symphyseal method based on component analysis. Alleging that Todd’s method is too static and consequently fails to recognize the variability associated with each feature, McKem and Stewart propose that a method utilizing three basic components is easier to use and therefore more applicable. The authors separate the symphyseal face into three component parts, the dorsal demi-face, ventral rampart, and symphyseal rim, and document a succession of metamorphic changes for each component. When evaluating a symphyseal face, a researcher must determine a score for each component. The sum of all component scores then correlates
with a range for estimated age at death. The method of McKern and Stewart was derived from analysis of 450 Korean War dead and consisted largely of young, white males. A similar formula, utilizing component analysis, was subsequently derived for use on females (Gilbert and McKern, 1973).

Hanihara and Suzuki (1978) use regression analysis to derive a linear function for symphyseal age estimation. The authors state the method is only applicable for individuals 18-38 years of age as changes to the pubic symphyseal face exhibit large variations after the age of 40. Snow (1983) also uses regression analysis to substitute a predicted age for the mean age in the method of McKern and Stewart.

Returning to the Todd methodology of reliance on a single pattern rather than component analysis, Katz and Suchey (1986) examined morphological change of the male Os-pubis. The method they derived was refined for application by Suchey and Brooks for males (1988a) and for females (1988b). The method entails the recognition of six phases of morphological change that have separate associated mean ages but overlapping ranges. Applicability of the method is enhanced by the availability of casts of each phase with corresponding descriptions of the key features for each. Katz and Suchey (1989) have refined the method further with the assessment of mean age and standard deviations according to race.

The various methods of determining skeletal age at death utilizing morphological change at the pubic symphysis have been tested individually and against each other for reliability, accuracy and ease of applicability. The Todd method is reportedly problematic when applied to older specimens (Meindl et al., 1983; Meindl et al., 1985; Katz and
Suchey, 1986). However, when compared to the method of McKern and Stewart, the Todd method is reported to be preferable to component analysis (Meindl et al., 1985; Katz and Suchey, 1986). In addition, Bocquet-Appel and Masset (1982) assert that the limited population on which the method of McKern and Stewart is based introduces systematic error with all populations aged using the method exhibiting a distribution similar to the Korean war dead. The method of Hanihara and Suzuki using regression analysis is reportedly more accurate than other component systems but is limited in application to the 18-38 year age range (Meindl et al., 1985). The method of Gilbert and McKern, a component analysis for the aging of females, is alleged to be highly unreliable and inaccurate with inherent problems of inter-observer error (Suchey, 1979). Klepinger and colleagues (1992) found that in a blind test of 202 females and 116 males using the McKern and Stewart, Gilbert and McKern, and Suchey-Brooks methods, all aging methods are “disappointing” with regard to accuracy and precision. They do conclude, however, that the methods of Suchey-Brooks fares the best with regards to both males and females and that the racially specific refinements should be the method of choice for the aging of males.

Katz and Suchey are not the first researchers to consider the variable of race. Todd (1921) finds race to be an insignificant variable. McKern and Stewart (1957) find no racial differences but admit that their sample contains only 10 blacks. To their credit, they do suggest the possibility that human groups with different nutritional backgrounds may exhibit different rates or patterns of morphological change. Klepinger and colleagues
(1992) also point out that physical trauma or a lack of physical activity may alter the normal pattern of morphological change.

*Auricular Surface of the Ilium*

Lovejoy and colleagues (1985) have designed a method utilizing the auricular surface of the ilium that is similar to the pubic symphysis method. Although they admit that changes in the auricular surface are more complex than changes at the pubic symphysis, the authors assert that the ilium undergoes changes beyond the age of 50 and is in fact equally accurate as a predictor of skeletal age at death. In addition, the ilium is often better preserved than the pubic symphysis.

The method divides the auricular surface into 2 demi-facets and a retroauricular area. The variables analyzed include porosity, surface grain, billowing and density. The complexity of the method arises from the fact that the two demi-faces may exhibit combinations of features from more than one phase. Consequently, the researcher must determine which features best reflect the aging process and choose a corresponding designated phase. Changes to the retroauricular area are designed to refine the phase estimation. The authors acknowledge the complexity and assert that practice and experience will make the method clearer and more applicable both forensically and archaeologically.

Lovejoy and colleagues (1985) further propose that the age changes at the auricular surface of the ilium are usually independent of osteoarthritic degeneration. Although pregnancy often results in the formation of a pre-auricular sulcus, this introduces only minimal bias and therefore separate standards for males and females are
not warranted. The method is stated to compare favorably with other systems utilizing the pubic symphyseal face with regards to accuracy and reliability.

In a test of the method, Murray and Murray (1991) find that the degenerative change is not dependent on race or sex for any given age category. However, they report that the degenerative changes are far too variable across individuals for the method to be useful as a single predictor of skeletal age at death. The accuracy of the method is further questioned by recent work in the medical community that recognizes anatomical variants of the sacroiliac joint including accessory joints, bipartite iliac bony plate, crescent-like iliac bony plate and semicircular defects to the iliac surface (Prassopoulos et al., 1999). In addition, the sacroiliac joint is subject to a high incidence of rheumatoid and inflammatory diseases (Kampen and Tillmann, 1998).

*Sternal End of the Fourth Rib*

Iscan and colleagues (1984) have introduced a new site for the estimation of skeletal age at death, the sternal end of the fourth rib. The sample on which the method is based consists of 118 white males from whom the sternal end of the fourth rib and associated cartilage was removed at autopsy. The fourth rib was chosen because of the ease with which it could be removed during a postmortem examination. All ribs were collected from individuals of known sex, age, and race. The rib pairs were soaked in water for several weeks and then gently boiled to remove soft tissue without damage to the bone at the costochondral junction.

The ribs are separated into nine groups based on changes at the costochondral junction. The variables considered include formation of the pit, shape and depth of the
pit, configuration of the walls and the rim surrounding the pit, and the overall quality and
texture of the bone. For a detailed description of each phase, see Chapter 4, table 4.1.

Statistical analysis including CROSSTABS, BREAKDOWN and ONEWAY
analysis of variance run on the data demonstrates a causal relationship between age and
phase. The most rapid and uniform change occurs from the ages of 17 to 28. Variation
increases after age 39, a fact which is reflected by greater age ranges in the later phases.
Phase 6, corresponding to an age range of 43-58, shows the most variability. The authors
assert that 85% of the changes occurring with age can be accounted for by the
characteristics defining each phase (Iscan et al., 1984).

Recognizing that women are under different hormonal influences and potentially
subject to different biomechanical stresses when compared to men, the authors have
developed a separate aging method for females utilizing the sternal end of the fourth rib
(Iscan et al., 1985). The method is derived from the study of 86 white, female rib pairs of
known age, sex, and race. Like the male sample, the female ribs are divided into nine
phases based on the morphology of the costochondral junction. In contrast to the males,
the females demonstrate earlier pit formation and differences in the overall pattern of
change. For a detailed description of each phase, see Chapter 4, table 4.2.

The data for females are subjected to the same statistical analysis as the males and
yield slightly different age ranges for each phase based on 95% confidence intervals. The
most rapid change is noted to occur in phases 1 through 4 corresponding to an age range
of 15 to 28 years. The rate of change slows after the age of 28 years. Generally, females
from the age of 20 years on exhibited thinner and less dense bone than males of the same age (Iscan et al., 1985).

Iscan and Loth tested both the female and male standards in a multi-observer test enlisting physical and forensic anthropologists. In the test of the method for aging males (Iscan and Loth, 1986a), 15 sets of ribs were judged by 25 anthropologists utilizing only color plates of each phase. The judges did not have a written description of each phase so that assessment essentially relied on visual pattern recognition. For 9 sets of ribs, 80% or more of the estimates were within 1 phase of the actual. Nearly all participants correctly aged some rib pairs while others were almost universally missed. This implies that the variation was greater among the ribs than among the judges (Iscan and Loth, 1986a). The authors report no correlation between the variation and individual characteristics such as cause of death, medical history, substance abuse, or occupation. Overall the tendency was to underage the rib pairs. Interestingly, the ability to apply the method was not dependent on educational background as in several cases judges with predoctoral degrees or recent Ph.Ds performed better than judges with many years of postdoctoral experience.

The method for aging females was tested using 10 sets of ribs and 28 volunteer judges (Iscan and Loth, 1986b). The test was set up in the same manner with judges supplied with photographic plates illustrating the rib phases. Collectively, rib assessment averaged well within one phase of the ideal. The specimen least accurately aged was from an individual who died of drug intoxication. The specimen most accurately aged died of arteriosclerotic cardiovascular disease (Iscan and Loth, 1986b). Overall, the
results were slightly better for the females tested than for the males with a mean deviation of .82 phases versus .97 phases, respectively.

The authors assert that the small sample size in the female test sample precludes analysis of possible correlation between antemortem factors such as cause of death, medical history, height, weight or occupation, and the accuracy of age determination generated from analysis of rib morphology. For males, however, they state that because the original sample on which the method was based is composed of a diverse group with varied medical histories and medical backgrounds, the resulting phase descriptions necessarily incorporate such variation (Iscan and Loth, 1986b). They do, however, admit that because the original sample, for both males and females, is composed of American whites, the possibility that the method is not applicable across populations is a valid concern.

In an effort to address the applicability of the method to a non-white population, Iscan and colleagues (1987) applied the standard for white males and females to a sample of 53 male and 20 female American blacks. In general, black ribs are found to look older than white ribs after the age of 30. Consequently, black ribs aged using the white standards are consistently over-aged from three to ten years for phases 5 through 7. The authors conclude that although blacks exhibit a different rate and pattern of morphological change from those observed in whites, separate standards are not warranted. Instead, the authors suggest modifications to the white standards when applying the method to the aging of a black decedent.
In an independent test of the fourth rib aging technique, Russell and co-workers (1993) found the methods derived from white males equally applicable to black males, although they do report a non-significant tendency to underage blacks, which is in contrast to the tendency to overage reported by Iscan and colleagues (1987). The method is reported to have "produced results similar to those obtained from radiographic changes in the proximal femur and lateral-anterior suture closure but not as accurate as the revised pubic symphysis, auricular surface, or cementum annulation" (Russell et al., 1993:56). They conclude by asserting the usefulness of the method, particularly when used in conjunction with other aging methods.

The method was also applied to the aging of the Spitalfields Cemetery population and found to be equally applicable to an historic population (Loth, 1995). In addition, application of the method to ribs other than rib number four, produces results suggesting that the method can be "cautiously applied" to ribs two through nine if rib four is not available (Dudar, 1993). Dudar and colleagues (1993) assert that the best results, however, are obtained when histological methods utilizing the ribs are used in concert with morphological methods. Use of the two techniques together accounts for more of the biomechanically induced variability and is thus preferable to either indicator alone.

Despite the reported accuracy and applicability, the original authors of the method acknowledge that factors affecting bone growth and remodeling include nutrition, disease, occupational stress, general health, endocrine disorders, medication, and the degree of physical activity (Iscan et al., 1984; Iscan et al., 1985). Given that the method is
a relative newcomer to the arena of skeletal aging, many of the listed variables and their effects on the morphology of the sternal end of the fourth rib remain untested.

**Variation in Costal Cartilage Ossification**

A review of the literature reveals the use of both the term “calcification” as well as the term “ossification” in reference to changes in the costal cartilages. Because calcification (mineralization of the cartilage matrix) precedes ossification (formation of bone), the two procedures are simply stages of the same overall process. In addition, it is presumed that the use of the term calcification is actually a misnomer and should be thought of as ossification (Semine and Damon, 1975). The two terms are used in the discussion that follows in accordance with their use by the authors of the works cited. For all intents and purposes, the two terms should be considered synonymous.

Semine and Damon (1975) report the results of a study of costal cartilage ossification in five distinct populations: European Americans, Lebanese, Solomon Islanders (the Lau and the Baegu), and a group of white male US veterans. The first rib, noted to ossify differently from the lower ribs, is considered separately. The study utilizes an accepted method of evaluation, ranking ossification on a scale of zero (no ossification) to four (extensive or complete ossification). They find that Caucasians exhibit greater ossification than the Baegu and offer nutritional differences as a possible causal factor. The Caucasians consume a mineral rich diet that includes salt and seafood while the Baegu diet is rich in carbohydrates. Interesting, although genetically indistinguishable, the Baegu and the Lau also exhibit differing degrees of ossification. While the Baegu consume predominately carbohydrates, the Lau are cited as supplementing the Baegu diet
with seafood and salt. The results of this study, and in particular the comparison between the genetically homogenous Baegu and Lau, suggest that nutritional differences do indeed affect the rate and degree of costal cartilage ossification. This raises the possibility that population specific standards would be required for any method utilizing the rate of ossification as a determinant of age.

In addition to the conclusions regarding the impact of nutritional variance, the above-cited study also finds differences between males and females. In all populations studied, the first rib in males ossifies significantly more than the first rib in females. For the lower ribs, males exhibit the greatest changes before the age of 45 and females after the age of 45. This suggests a possible hormonal influence tied to the changing levels associated with menopause in females. Males also are examined relative to chest circumference with regards to the hypothesis that larger physique imposes a greater strain on the first rib and may thus incite ossification. They do, indeed, find a correlation between the variables suggesting a possible biomechanical component to ossification of the first rib (Semine and Damon, 1975).

In a longitudinal study of 13 healthy male military pilots, Barchilon and co-workers (1996) report great variability in the ossification process of the first costochondrosternal joint. They conclude that the degree of ossification of the first costal cartilage is not precise enough for prediction of age. However, they also point out that as military pilots, the observed individuals are subjected to challenging physical pressures on the chest that may accelerate the ossification process. They theorize that microfissures resulting from repeated stress to the joint may stimulate calcification of the cartilage. If
true, this would constitute an example of biomechanical stresses influencing costal cartilage ossification.

McCormick and Stewart (1983) and Stewart and McCormick (1984) report on the identification of a sex specific pattern of ossification. The pattern, characterized by spherical or globular foci of ossification occurring in the central portion of the rib cartilage, is reportedly unique to postmenopausal women. Interestingly, the pattern does not involve changes at the sternal end of the rib.

Teale and colleagues (1989) also have found differences between males and females with costal calcification more common in men than in women. After studying 700 chest radiographs of men and women from the third through seventh decades of life, they conclude that “although the prevalence of calcification on chest radiographs increases with age, it is not a specific sign of aging in an individual” (Teale et al., 1989:335). They further express puzzlement as to why the costal cartilage should be prone to calcification in the first place, and why such processes would be more common in men than in women.

In support of a predictable pattern of ossification in the costal cartilages, studies of aberrant ossification in children have demonstrated a pattern consistent with what is seen in older individuals. Calcification is reported in the costal cartilages of adolescent hyperthyroidism patients (Senac et al., 1985) and in a single patient undergoing prolonged prednisone treatment (Dearden and Mosier, 1975). In both cases, the ossification is reported to be aberrant in timing but not in pattern. Although not specifically analyzed with regard to the method of Iscan and Loth (Iscan et al., 1984; Iscan et al., 1985), the
authors nevertheless report that the pattern is consistent with what is seen in older individuals.

Although supportive of the premise of a recognizable pattern of ossification, cases of early ossification in children also raise the possibility of variation stemming from environmental or hormonal influences. Haddad and co-workers (1993) report on two children with tracheobronchial, laryngeal, and costochondral cartilage calcification. Although associated with cardiovascular anomalies, the condition is otherwise idiopathic in origin. Clearly, environmental variations do exist that can influence the rate, if not the pattern, of costal cartilage ossification.

Chronic substance abuse, whether considered purely as an environmental (behavioral) force or as a behavior with an underlying genetic predisposition, must be considered as a possible factor influencing the ossification of the costal cartilages in a manner comparable to nutritional variances, biomechanical insult, disease processes, and hormonal imbalances.
The phenomenon of drug abuse is multifaceted with biological, psychological, epidemiological, social, cultural, and legal components. In addition to marijuana, the most frequently abused drugs in the United States are alcohol, heroin, and cocaine (DiMaio and DiMaio, 1989). Alcohol is the most commonly abused drug in the United States and the most frequently encountered causal factor in violent or unnatural deaths (Freimuth, 1993). Heroin and cocaine offer the lure of euphoric highs for the users and the potential of large profits to the drug traffickers. Unfortunately, drug use is most common among the poor who "do not take precautions against the spread of disease, can least afford the habit and seem to have less perception of the long term risks involved" (Stephens, 1993: 734). The effects of drug abuse are seen in hospital emergency rooms, jails and courtrooms, treatment centers, homeless shelters, and the county morgue.

Drug abuse is not a modern cultural phenomenon. In the 19th century, the supposed "three curses of mankind" were said to be alcohol, morphine, and cocaine (DiMaio and DiMaio, 1989). Heroin was first manufactured from morphine in 1898 and proclaimed to be non-addictive (Stephens, 1993). Cocaine was first isolated and named by a German chemist in 1859. Proponents of its use, in one form or another, included kings, queens, two popes, Thomas Edison, H.G. Wells, Jules Verne, and Sigmund Freud (Weiss and Mirin, 1987). Between the years of 1886 and 1900, Coca-Cola was manufactured using a combination of caffeine and cocaine (Stephens, 1993). The use of
cocaine is not confined to recent history, as coca leaves have been discovered at Peruvian gravesites dating to approximately 500 AD (Weiss and Mirin, 1987).

In 1970 the U.S. Controlled Substance Act provided the legal foundation for governmental control and enforcement of drug manufacturing and use (Stephens, 1993). Since then, heroin and cocaine consumption has continued as illicit substance abuse. Recognition of the effects of chronic substance abuse on the human body, and most notably on skeletal morphology, is paramount to the medico-legal investigation of death. In addition, the apparent antiquity of drug abuse suggests skeletal manifestations of the behavior could be encountered in an archaeological as well as modern context.

This chapter reviews the known effects of chronic substance abuse and includes sections on heroin abuse, cocaine abuse, intravenous drug use, and alcohol abuse. The chapter concludes with a discussion of drug abuse in King County, Washington, the population from which the study sample is derived.

The Effects of Heroin Abuse

Heroin, diacetylmorphine, is classified as a schedule I substance in the United States based on its high abuse potential in the absence of any recognized medicinal value (Baselt and Cravey, 1995). It is almost always prepared by a reaction of acetic anhydride and morphine, with the only natural source of the latter being the botanical Papaver somniferum and its cultivars, known more commonly as the garden poppy, oil poppy, and the opium poppy (Cooper, 1999). Street grade heroin exists most commonly as either “China White”, a white powder, or “Black or Mexican Tar”, a dark gummy substance that in larger quantities resembles black roofing tar. Although China White can be
administered intranasally by insufflation ("snorting"), a majority of heroin use in the King County area is by intravenous injection. This undoubtedly reflects the fact that the majority of the heroin available on the streets of Seattle and surrounding environs is Black Tar (Jackson et al., 1998).

The preparation of Black tar involves dissolving the drug in water to facilitate injection. This is accomplished by heating the drug in a "cooker" which most commonly consists of a kitchen spoon and hand held cigarette lighter. Once dissolved, the drug is drawn into the syringe using a cotton wad as a filter. The sources of contamination are numerous and include the water source (often toilet water or saliva) and the possibility of cotton fibers or infiltrates mixing with the solvent during the loading of the syringe. Injection is either intravenous or subcutaneous. The veins of choice tend to be those located in the antecubital fossa (inner elbow), however injection also occurs in the groin, ankle, neck, or any accessible vein. Long-term injection can lead to the sclerosing or thrombosing of veins resulting in "track marks". Once the utility of the vein is lost, addicts will seek a new injection site or resort to subcutaneous injection ("skin popping"), usually in the buttocks or thighs.

As an opiate derivative, heroin is classified as a narcotic or central nervous system depressant. In large doses it can incite respiratory depression and coma. The mechanism of heroin death is not known, although pulmonary edema is the most frequent complication of heroin overdose (Ellenhorn and Barceloux, 1988). Informants at death scenes routinely describe the decedent as "shooting up", going to sleep, often times with a characteristically "wet snore", and then becoming unresponsive. Death can result from
true pharmacological overdose, allergic or idiosyncratic reactions, adulterants, or drug interactions (Ellenhorn and Barceloux, 1988).

Medical complications from heroin abuse are divided into two categories, those resulting from the heroin itself and those resulting from the unsterile practices surrounding its administration (Stern et al., 1968). The former category is restricted exclusively to the syndrome of acute heroin intoxication and centers largely on the pulmonary manifestations and complications of heroin overdose (Stern et al., 1968). The latter category will be considered in conjunction with the effects of the parenteral use of cocaine.

The Effects of Cocaine Abuse

Cocaine, derived from the leaves of *Erythroxyylon coca*, a South American shrub, is one of the most potent of the naturally occurring central nervous system stimulants (Baselt and Cravey, 1995). Medicinally, cocaine is used as a stimulant, topical anesthetic, and vasoconstrictor (Gordon et al., 1990). In addition, cocaine creates patient “euphoria” and is easily tolerated by the patient making it the anesthetic of choice for use in intranasal surgery (Fairbanks and Fairbanks, 1983). Despite its medical value, cocaine has, in previous years, been identified as America’s leading illicit drug of abuse (Tarr and Macklin, 1987).

Street cocaine, or cocaine hydrochloride, is a white powdery substance that is often times administered by insufflation. “Free base” cocaine is the alkaloidal form of the drug which is more volatile than that chloride salt and thus suitable for smoking (Ellenhorn and Barceloux, 1988). Freebase cocaine, also known as crack because of the
noise produced when it is heated, is a solid form of the drug descriptively known as "rock cocaine". Crack cocaine is smoked using a crack pipe, which is often a crude appliance composed of a glass tube and a makeshift filter. For those abusers low on funds, a crack pipe can easily be manufactured from the glass tube encasing a single stemmed rose purchased at a flower shop or grocery store. A filter is easily constructed using a small piece of copper Brillo pad marketed for the scrubbing of pots and pans. Like heroin, cocaine can also be intravenously injected.

As a central nervous system stimulant, cocaine creates an euphoric high followed by a rebound dysphoria, or "crash" (Ellenhorn and Barceloux, 1988). Some recreational drug users choose to use cocaine and heroin simultaneously, a habit known as "speedballing", reportedly in an effort to diminish the down side of the cocaine high by mellowing it out with the depressant effects of heroin. Taken in excessive quantities, cocaine can cause myocardial infarction, ventricular tachycardia, fibrillation, pulmonary dysfunction, and intracranial bleeding (Baselt and Cravey, 1995), all of which can be fatal.

In addition to the immediate effects of cocaine, published case reports suggest that vasoconstriction from cocaine use can induce rhabdomyolysis leading to acute renal failure (Whooley et al., 1990; McCrea et al., 1992). Additional skeletal involvement is most notable for the cartilaginous and bony destruction of the support structures of the nose, pharynx, and palate. Published case reports include the bony destruction of the eye orbit (Underdahl and Chiou, 1998), septal perforation with pharyngeal wall ulceration (Deutsch and Millard, 1989), and perforation of the septum and palate, and nasal dorsum
collapse (Mattson-Gates et al., 1990). The bony and cartilaginous manifestations of cocaine snorting are a reflection of the high dosage administered by recreational cocaine “snorters”. A dosage of 2 to 3 mg/kg is the recommended maximum dose for topical anesthesia. Cocaine abusers may use 1,000 mg/kg or more on a daily basis (Schweitzer, 1986).

The most severe effects on the skeletal system of chronic cocaine abusers result from the parenteral use of the drug. Like heroin, the complex of infectious diseases resulting from the non-sterile practices of intravenous drug abuse leads to several notable skeletal disorders. Consequently, the parenteral use of the two drugs will be considered together in the following section.

The Effects of Intravenous Drug Abuse

Louria (1974) summarized the most common infections accompanying heroin injection and included the following: endocarditis, hepatitis, embolic pneumonia, cellulitis, myostitis, arthritis, osteomyelitis, brain abscess or meningitis, malaria, tetanus, and upper respiratory acquired pneumonia. He hypothesized that “the increased incidence of infection in the addict certainly results from putting organisms directly into the bloodstream or establishing local lesions in the skin, subcutaneous tissues, veins or muscles that form the nidus for severe focal or systemic infections” (Louria, 1974: 228). Furthermore, the phenomenon is not limited to heroin users as the apparent total disregard for sterile preparation extends to the use of other injected drugs of abuse.

In addition to the unsterile practices of drug preparation, there is also the possibility that the drug itself is contaminated. Although the contamination of heroin
would be impossible to prove in the absence of a widespread and longitudinal sampling of street grade heroin, it has been documented that heroin (white powder) is usually "cut" with mannitol, inositol, quinine, procaine, lidocaine, diphenhydramine, or sometimes talc (Villareal et al., 1993). Interestingly, the spread of malaria by the sharing of contaminated needles has decreased since the 1960’s, presumably because of the inclusion of quinine as a common adulterant of street grade heroin (Louria et al., 1967).

Although many of the above listed infections result from the hematogenous dissemination of contaminated material, the practice of subcutaneous injection is no safer. The practice of so called "skin popping" produces interconnecting abscesses that create the perfect environment for the growth and proliferation of Clostridium tetani (Louria et al., 1967). Similarly, "skin poppers" may succumb to necrotizing fascititis, an aggressive and often times lethal disease.

Of the numerous infectious diseases associated with intravenous drug abuse, three in particular require further mention. Osteomyelitis, septic arthritis, and osteosclerosis all manifest in skeletal tissue and therefore have the potential to alter, either directly or indirectly, the skeletal indicators of age at death. The three diseases are considered separately in the discussion that follows.

Osteomyelitis

Osteomyelitis, inflammation of the bone caused by pathogenic organisms, is a disease classically described in children, usually in reference to the long bones of the lower extremities (Waldvogel et al., 1970). When it is described in adults, it is seen most commonly in the lower cervical and lumbar spine (Fishbach et al., 1973). Case reports in
the literature describe the lytic destruction of bone and the erosion of the articular margins owing to the erosive effects of osteomyelitis.

In a published report of 14 intravenous heroin abusers, Endress and colleagues (1990) describe cervical osteomyelitis characterized by advanced vertebral body destruction, disk space infection, prevertebral abscess, and anterior cervical inflammatory reaction. Zucker and colleagues (1974) likewise describe several patients with pyogenic vertebral osteomyelitis of the lumbar spine. One patient exhibited the partial destruction of L4 and L5 with the corresponding narrowing of the intervertebral space. A second patient presented with a similar manifestation involving L3 and L4. The causal mechanism is assumed to be septic emboli from intravenous injection of heroin. Wiesseman and co-workers (1973) outline the post treatment follow-up of five patients with vertebral osteomyelitis that exhibited the proliferation of new bone with the narrowing of the intervertebral spaces indicating progressive fusion. Clearly, osteomyelitis of the spine is a potentially destructive process infecting individuals reported to have no predisposing condition except intravenous drug abuse.

In addition to the spine, osteomyelitis of hematogenous origin in intravenous drug abusers has been reported in the sternum (Walker and Pate 1991) and sternoclavicular joint (Holzman and Bishko, 1971). Taylor and Lawson (1986) report periostitis and osteomyelitis in the bones of the forearms either as a result of the injection of bacteria into the periosteum or by introduction through infected skin and subcutaneous tissue. Likewise, Ashby (1976) reports on a case of non-nosocomial infection of the ulna presumed to be the result of the contiguous spread from a soft tissue abscess.
The medical literature also includes case reports of disseminated candidiasis as a distinct syndrome in heroin users. Candidiasis, an infection of the skin or mucous membranes caused by a yeast, is reported to involve costal cartilage and/or bone in numerous reported cases (Collignon and Sorrell, 1983; Sorrell et al., 1984; Dupont and Drouhet, 1985; Miro et al., 1988). *Candida* is also noted as a causal mechanism in osteomyelitis (Holzman and Bishko, 1971; Lafont et al., 1994).

**Septic arthritis**

Septic arthritis, like osteomyelitis, results in the erosion of joint margins. It is most often associated with fibrocartilaginous joints including the sacroiliac, sternoclavicular, sternocostal, and pubic symphysis. Appearance is uncommon in larger joints (Lopez-Longo et al., 1986).

Goldin and co-workers (1973) report on four heroin users diagnosed with sternoclavicular septic arthritis characterized by demineralization of the head of the clavicle with associated osteomyelitis of the sternum and/or erosion of the inferior sternum. Brittini and colleagues (1985) report on nine cases of primary septic arthritis of which four affected the sternoarticular joint, three the sacroiliac, one the sacroccocygeal and one the knee. Likewise, in a survey of 37 cases, Lopez-Longo and colleagues (1987) found 39% affected the sacroiliac joint while 37% involved the chondrosternocostal joints. A repeating theme in published case reports regarding primary septic arthritis in intravenous drug abusers is the probable causal role of drug contaminants or unsterile injecting paraphernalia (Goldin et al., 1973; Ross et al., 1975; Oh, 1977).
Osteosclerosis

While osteomyelitis and septic arthritis involve the destruction of bone, osteosclerosis is characterized by an increase in bone mass and skeletal radiodensity (Villareal et al., 1992). The condition accompanies a variety of disorders including inherited osteopetroses and a wide array of developmental conditions. When it is acquired, it is rarely idiopathic (Villareal et al., 1992). Villareal and colleagues (1992) describe two intravenous drug abusers who presented with pain in the lower extremities. Both patients exhibited elevated alkaline phosphatase levels (a marker of bone mineralization), elevated osteocalcin levels (a marker of bone formation), elevated urine excretion of hydroxyproline (a marker of bone resorption), decreased urine excretion of calcium, moderately elevated levels of 1,25 dihydroxycholecalciferol, and normal parathyroid levels. The symptoms suggested an elevated rate of skeletal remodeling characteristic of osteosclerosis. The authors speculate that the pathogenesis involved an increase in bone formation rather than a decrease in bone resorption and put forth that the causal mechanism could be the parenteral transmission of a virus that directly or indirectly stimulates osteoblast activity.

In a response to Villareal et al., (1992), Beyer and colleagues (1993) describe a patient with similar symptoms who exhibited cortical thickening of the lower extremities. As an alternative causal mechanism, they propose a chemical substance introduced as a contaminant of the drug injected. Interestingly, both sets of authors shy away from hepatitis as a causal virus although other authors have cited osteosclerosis as a rare complication of Hepatitis C infection (Diamond and Depczynski, 1996). Regardless of
the causal mechanism, osteosclerosis is a documented complication of intravenous drug abuse that potentially alters skeletal morphology.

The Effects of Ethanol Abuse

Ethanol (alcohol) is a social drug that is rarely used therapeutically, although it is found in medicinal liquids such as cough syrups and mouthwashes. In comparison to illicit drugs, alcohol is cheap, readily available, and easily consumed. Consequently, alcohol is the most common single drug taken by patients and determination of blood alcohol level the most frequently performed medicolegal test (Ellenhorn and Barceloux, 1988).

In large quantities, alcohol acts as a central nervous system depressant. Chronic alcohol abuse is associated with systemic disorders affecting the integumentary, gastrointestinal, respiratory, cardiovascular, genitourinary, endocrine, nervous, muscular, and skeletal systems (Ellenhorn and Barceloux, 1988). Like illicit drugs, chronic alcohol use is implicated in the etiology of several skeletal pathologies. The exact effect of alcohol on bone physiology and skeletal morphology, however, is debated.

Perhaps the most recognized skeletal disorder associated with chronic alcoholism is an increase in the incidence of fractures, particularly compression fractures of the spine. A study by Peris and colleagues (1995) found that in a sample of 76 chronic alcoholic males, 36% had evidence of vertebral fractures. The fractures occurred with the highest frequency in T5, T8, T9, T11, T12 and L1. In addition, 61% of those exhibiting vertebral fractures had a history of one or more non-vertebral fractures including rib fractures. In comparison, only 3% of the nonalcoholic age matched control group
exhibited peripheral fractures. These researchers further state that most of the alcoholic patients developed the vertebral fractures in the absence of decreased bone mineral density. They therefore postulate that trauma is the most likely factor responsible for the observed vertebral fractures.

An earlier report by Israel and co-workers (1980) found that in a sample of 198 alcoholic males, 57 (28.9%) exhibited vertebral fractures, while only four of 218 (1.8%) control subjects had them. They, too, speculate that trauma is the probable cause of alcoholic vertebral fractures. In fact, they suggest that the incidence of such fractures is so common that chest radiographs may be useful in the screening for alcoholism. Other researchers acknowledge the higher prevalence of vertebral fractures in alcoholics but denounce the usefulness of chest radiographs as a tool for the diagnosis of excessive alcohol consumption (Keso et al., 1988).

In a large population based study with repeated assessments of alcohol intake and the measurement of other factors that may affect bone mineral density, Felson and colleagues (1995) found that moderate alcohol consumption actually increased bone density. Females who consumed seven oz. or more of alcohol per week averaged 5 -10% greater bone densities with the most notable sites being the femoral neck, distal radius, and lumbar spine. Males who consumed a quantity of alcohol equal to or in excess of 14 oz. per week exhibited a marginal positive correlation with bone density. The authors do acknowledge, however, that their subjects were generally well nourished so that any nutritional consequences of chronic alcohol consumption were absent. Beneficial effects associated with moderate alcohol consumption have also been reported in cases of
duodenal ulcer, gallstones, enteric infections, rheumatoid arthritis, atherosclerosis, osteoporosis, and type II diabetes (Goldberg et al., 1999).

Not all researchers agree that fractures in alcoholics are traumatic in origin or that alcohol consumption increases bone density. Utilizing samples of the iliac crest taken at autopsy, Saville (1965) found that young alcoholics had a decreased bone mass similar to that observed in post-menopausal women. Nilsson and Westlin (1973) and Johnell and co-workers (1982) both found a decrease in the bone mineral content in the forearms of alcoholics when compared to age matched controls. Additionally, Johnell and co-workers (1982) found an increase in osteoclast number in iliac crest biopsies suggesting increased bone resorption. Bikle and co-workers (1985) report a mean density of vertebral cancellous bone in the alcoholics they studied that was only 58% of the normal value and a mean density of appendicular bone that was 90% of the normal value. Spencer and colleagues (1986) also report a high incidence of excessive bone loss in the 96 young and middle aged chronic alcoholic men they studied and equate such loss to what is seen in osteoporosis.

The causal mechanism and definitive etiology of bone mineral loss in alcoholics remain speculative. Nilsson and Westlin (1973) suggest nutritional factors may be involved because the alcoholic subjects they studied matched the controls for mean height but averaged five kilograms less in weight than the control subjects. When their sample was dissected, they found no significant difference in bone mass in the young alcoholics when compared to the controls but there was a significant drop of nearly 25% in the older alcoholics. This suggests age and/or duration of alcohol abuse may be
relevant factors. Kimball (1997) suggests that hepatic cirrhosis (common in alcoholics) may influence levels of cytokines involved in bone resorption. Other researchers have suggested additional possible causal mechanisms including hormone imbalance and the direct toxic affects of alcohol on bone.

The reported behavior of hormones in response to alcohol consumption is varied and often contradictory. A study of 38 noncirrhotic male alcoholics found normal bone mineral density of the lumbar spine when compared to the controls but a 40% reduction in the serum levels of 25-hydroxycholecalciferol and 1,25-dihydroxycholecalciferol (Laitinen et al., 1990). Decreased levels of vitamin D metabolites have been reported by other researchers as well (Pitts and Van Thiel, 1986; Abbott et al., 1994). However, a study of 17 chronic alcoholics found lower serum levels of 25-hydroxycholecalciferol but increased levels of 1,25 dihydroxycholecalciferol concurrent with increased levels of parathyroid hormone (Feitelberg et al., 1987). In contrast, other researchers report normal parathyroid levels in alcoholics (Crilly et al., 1988; Labib et al., 1989; Laitinen et al., 1994). Clearly there is a lack of consensus regarding the hormonal response to alcohol consumption.

Similarly, Bikle and colleagues (1993) conducted a thorough study of 27 chronic ethanol abusers which included bone density measurements by CT scan of the lumbar spine, histomorphometry analysis of iliac crest biopsies, and measurement of serum levels of vitamin D metabolites, parathyroid hormone, and bone minerals. They found that age and/or duration of alcohol abuse substantially influenced the degree to which bone remodeling was affected by alcohol and postulated that parathyroid hormone may
stimulate bone resorption in younger alcoholics but that with time the cumulative effects of alcohol abuse cause a decrease in bone remodeling regardless of parathyroid hormone levels. They interpreted this as possible evidence that alcohol is toxic to bone cells and stated their conclusions as:

An early ethanol induced increase in PTH to initiate the imbalance between bone resorption and formation coupled with a cumulative toxic effect of ethanol on bone cell activity, possibly affecting osteoblasts more than osteoclasts, would explain the results of this and other studies” (Bikle et al., 1993: 694).

A cumulative toxic effect of alcohol would be consistent with a decrease in spinal bone density and an increase in spinal fractures with age.

The possibility that ethanol is directly toxic to bone cells is a theory that is repeated throughout the literature with regard to the skeletal effects of alcohol abuse. In an effort to measure osteoblast activity, researchers rely on the measurement of serum levels of proteins synthesized by osteoblasts including osteocalcin and bone Gla-protein (BGP). Elevated levels of either protein reflect a high rate of bone synthesis while decreased levels indicate reduced osteoblast activity. Low serum levels of osteocalcin in alcoholic subjects are cited as evidence of a toxic effect of alcohol on osteoblasts (Rico et al., 1987; Labib et al., 1989; Laitinen and Valimaki, 1991). Likewise, low levels of serum BGP in conjunction with histomorphometric analysis and decreased bone mineral density are cited as evidence of the suppressant effect of alcohol on osteoblasts (Diamond et al., 1989; Gonzalez-Calvin et al., 1993).

Additional evidence of the direct toxic effect of ethanol on osteoblasts is provided by the results of an in vitro study exposing osteoblasts to ethanol (Chavassieux et al.,
A measurable decrease in osteocalcin levels suggests that ethanol may decrease osteoblast activity and proliferation. The suppression of osteoblasts would lead to a decrease in bone formation and defective mineralization (Rico, 1990).

In an effort to sort out the often contradictory results of studies on human subjects, some researchers have turned to an animal model utilizing rats. While human studies are complicated by numerous variables including, but not limited to, differential exposure to sunlight, the degree of malnutrition, duration of alcoholism, extent and frequency of drinking, chronological age, and the possibility of malabsorption disorders (Turner et al., 1988), rat models allow the researcher greater uniformity between subjects.

Studies utilizing rat models have demonstrated normal levels of calcium regulating hormones (Sampson et al., 1998) and disturbed vitamin D metabolism (Turner et al., 1988) in chronic alcohol fed rats in comparison to controls. In addition, alcohol-fed rats exhibit an intrinsic difference in bone quality with a higher fat percentage and lower mineral content (Sampson et al., 1998). Osteoblast activity is decreased in both chronic and acute exposure (Diez et al., 1997; Sampson et al., 1998). Increased bone resorption is evidenced by an increase in the diameter of the medullary cavity of long bones and the loss of trabecular bone (Turner et al., 1988). In addition, trabecular bone in the femur is noted to be thinner and more columnar in alcohol-fed rats resulting in the increased likelihood of fracture (Peng et al., 1988).

Although the obvious drawback of rat models is the uncertainty of exact congruence between human and rat physiology, it is nonetheless interesting to note many similarities between the two models. Both suggest that chronic alcohol abuse decreases
bone mineral content, stressing calcium homeostatic mechanisms and exposing alcoholics to increased risk of pathological fractures.

A final skeletal pathology related to chronic alcohol abuse and therefore worthy of mention is nontraumatic aseptic osteonecrosis. Thought to result from the obstruction of the vascular supply by a fat embolus and linked to co-existent hyperlipidemia (Jacobs, 1992), nontraumatic osteonecrosis is most common in the femoral head but may also occur in the humeral head or femoral condyle (Saville, 1975). In a study of 1157 chronic alcohol abusers, 5.3% were found to have lesions consistent with aseptic necrosis (Orlic et al., 1990). Allowed to progress, the disorder results in serious disability and restriction of motion (Saville, 1975). Although not common, osteonecrosis must nonetheless be considered as a possible skeletal pathology resulting from chronic alcohol abuse.

**Drug Abuse In King County**

King County Washington spans an area of 2,130 square miles and includes the entire Seattle area as well as 32 other cities and towns. The Washington State Office of Financial Management estimates the 1998 population of King County at 1,665,800 individuals. Unless otherwise noted, all data regarding deaths in King County are obtained from the King County Medical Examiner’s Office 1998 Annual Report.

The office reported the investigation of 1507 deaths in 1998, of which 427 (28.3%) were deemed accidental. In King County, deaths caused by the recreational overdose of illicit or prescription drugs are not considered homicides, suicides (unless evidence of intent exists), or natural deaths. They are therefore ruled accidental. In 1998, 179 of the 427 (42%) accidental deaths were attributed to drugs. Eighty-one percent of
the decedents were between the ages of 30 and 49 years. Ten percent were between 20 and 29 years, and two individuals were between the ages of 16 and 19 years. The number of accidental overdoses (179) represents a 12% increase compared to the 155 deaths investigated in 1997. When suicide and undetermined deaths attributed to drugs are added to accidental deaths, the total for 1998 jumps to 234 which is the highest number ever recorded for King County.

With regard to the total number of deaths caused by drugs (234) in 1998, 144 (62%) involved opiates, either alone or in combination with other drugs. This is a greater than 400% increase in opiate involved deaths since 1985. Cocaine, either alone or in combination with other drugs, was involved in 70 of the 234 deaths (30%) while ethanol in combination with other drugs was implicated in 95 deaths (41%). Acute ethanol intoxication in the absence of any other drugs was the cause of death in 11 cases (5%).

Epidemiologists for the King County Department of Public Health studying opiate deaths in the county report that most decedents are white, male, and between the ages of 25 and 54. Most are county residents residing in the central area of Seattle (Solet, 1999). The increase in opiate deaths in King County is perhaps best represented by the fact that only 47 opiate deaths were recorded in 1990 as compared to 144 in 1998. This represents a 206% increase despite the fact that the county population only increased by 11% during the same time frame (Solet, 1999).

The increase in drug use in the King County area is further reflected by the fact that seven counties in Puget Sound, including King County, have been designated as a high intensity drug trafficking area by the United States Federal Government (Larson,
1999). In addition, needle exchange programs, run by the King County Department of Public Health and designed to decrease the transmission of blood borne diseases, average 55,000 to 65,000 needle exchanges per month. In 1998, they recorded 1.7 million exchanged syringes and 90,000 users served (Solet, 1999). Intravenous use of heroin is so endemic to the Seattle area that in street vernacular a fatal heroin overdose is referred to as a "Seattle natural" death.

Heroin and cocaine are both readily available on the streets of Seattle with "flake" cocaine selling for as low as $35-$40 per weighed gram, "Crack" cocaine for $20 per 1/10 to 1/8 gram rock, and Mexican tar heroin for $80-$100 per gram. Prescription opiates which include codeine, fentanyl, hydrocodone (Vicodin, Lorcet and Anexsia), hydromorphone (Diluadid), meperidine (Demerol), methadone, oxycodone (Percodan, Percocet), pentazocine (Talwin), and propoxyphene (Darvon) carry a street value of $3 to $5 per tablet (Jackson et al., 1998).

The paraphernalia needed to administer illicit drugs are also not difficult to obtain. Needles for injection are sold in packs of 10, marketed primarily for diabetics, and available over the counter in any drug store. "Cookers" are constructed utilizing household items, most notably kitchen spoons or the sawed off bottom of an aluminum can. As discussed previously, crack pipes are manufactured utilizing items that are easily obtained at the neighborhood grocery store or florist. Indeed, the availability of the drugs, and the simplicity of the paraphernalia, suggest that illicit drug abuse in King County will continue to be a problem in the years to come.
The continued abuse of illicit drugs suggests that cases handled by the Medical
Examiner's Office will continue to involve a high frequency of chronic drug abusers.
This serves to illuminate the urgency of determining the contribution of chronic
substance abuse to the alteration of skeletal morphology. In cases of unidentified
remains, medical history is obviously not known. Therefore, the forensic practitioner
must be able to rely upon methods of determining age at death that are independent of the
possibility that the decedent was a chronic substance abuser.
CHAPTER 4
MATERIALS AND METHODS

The Sample

The sample consists of 173 pairs of the sternal end of the fourth rib removed during autopsy from cases under the jurisdiction of the King County Medical Examiner’s Office in Seattle, Washington. Removal of a segment or segments of ribs is a routine procedure during autopsy. The rib specimens are retained with other tissue specimens indefinitely for future analysis including, but not limited to, DNA testing, nutritional assessment, disease analysis, and age determination. This study, therefore, does not require collection of material outside of the tissue procurement that is standard autopsy procedure.

Deaths which come under the jurisdiction of the Medical Examiner are defined by state statute (Revised Code of Washington 68.5) and include, but are not limited to, the following circumstances:

1. Persons who die suddenly when in apparent good health and without medical attendance within thirty-six hours preceding death.
2. Circumstances which indicate death was caused in part or entirely by unnatural or unlawful means.
3. Suspicious circumstances.
4. Unknown or obscure causes.
5. Deaths caused by any violence whatsoever, whether the primary cause or any contributory factors in the death.
6. Contagious disease.
The manners of death investigated by the Medical Examiner's office include natural, suicide, homicide, accidental, and undetermined. Deaths attributed to acute intoxication of a controlled substance, and resulting from recreational use, are deemed accidental.

When the King County Medical Examiner's Office assumes jurisdiction over a death, an investigator's report is initiated regardless of whether the death involves a scene investigation. The investigator's report includes an accounting of the terminal event, vital statistical information, and a summary of medical history. To this end, investigators routinely interview family members, physicians, employers, and friends of the decedent, request and review medical records, and perform thorough scene investigations that illuminate the life quality and personal habits of the decedent. The investigator's report also includes a section for an indication of drug and/or alcohol abuse. Standard procedure is to indicate the number of years, if known, of substance abuse and the drug(s) of choice. This is routinely recorded as "years" and all 10 investigators active at the time of data collection for this study report that a notation on the report is only made if abuse is known to be chronic (loosely defined to be in excess of 3 years). There are, however, cases in which medical history (and specifically drug abuse history) is not pursued either because of unavailability of information or because the cause of death is obvious and not dependent on past medical history (most notably traffic related deaths, suicides by hanging or gunshot wounds).

Medical records obtained on the decedents almost always list a medical history section that provides excellent information regarding the addictive behaviors of the
decedent. Obtainment of the medical records is part of the standard investigative protocol and is not achieved solely for the purpose of this study.

Following autopsy, a report is generated by the pathologist and is considered confidential information, available only to immediate family members or by court order. The autopsy report discusses the integrity and overall health of each and every organ system, noting pathological conditions which may be caused by chronic drug abuse. Chief among them is the observation of healed track marks on the antecubital fossae, a finding consistent with chronic intravenous drug abuse. Also noteworthy is a finding of micronodular cirrhosis and/or fatty liver. These pathological liver conditions are consistent with, although not exclusively caused by, chronic alcohol abuse.

The decision to assume jurisdiction on a death is made by the professional staff of the King County Medical Examiner’s Office in accordance with RCW 68.50. Permission of the legal next of kin is not required. Opposition to autopsy on the part of family members is considered but the decision to perform a full postmortem exam rests solely with the Medical Examiner. Likewise, medical records are obtained via subpoena and do not require a release by the legal next of kin.

This study was performed with the complete knowledge and support of the King County Medical Examiner’s Office.

Collection

The sternal end of the right and left fourth rib consisting of approximately 1-4 inches of bone and 1-2 inches of associated cartilage was removed by pathology assistants at autopsy and packaged individually by case number. The cases for which a
fourth rib was removed (per autopsy protocol) rather than another rib segment were
dependent on the condition of the remains and accessibility of the fourth rib. Extensive
thoracic trauma including rib fracturing as is common in restrained drivers in motor
vehicle collisions, for example, negated the usefulness of the fourth rib for study. When
the fourth ribs were collected, they were labeled and placed in a box in the freezer for
later maceration. The ribs utilized in this study were collected over a 7-month period.

Maceration

Each set of ribs was placed in an elastic banded, woven fabric hair net, cinched
and secured with a rubber band. A number tag bearing the case number was tied around
the package. Fifteen sets of ribs, each individually packaged and labeled, were placed in a
metal container filled with water and non-bleach detergent. The container was placed on
a hotplate and allowed to reach a temperature of approximately 130 degrees Celsius for 7
hours. Following the cooking process, the ribs were removed from their fabric baggies
and individually cleaned and scraped to remove soft tissue. The cartilage at the costal
cartilage junction usually became disassociated during cooking. If, however, cartilage
remained, it was removed delicately with a dental pick. Care was taken not to alter the
morphology of the sternal end of the bone.

Following cleaning, the ribs were laid out on a towel with their associated case
number. The following morning, Nicole Bourque, the Forensic Anthropology Intern at
the King County Medical Examiner’s Office, packaged each set of ribs and assigned each
a code number. The original tag bearing the case number was placed in a sealed
envelope, which was packaged with the rib set. From that moment on the ribs were
identified only by the code number. Code numbers ranged from 1 to 173. Ms. Bourque kept the running list of the code numbers and associated case numbers.

**Examination of Ribs**

Ribs were analyzed for estimated age at death utilizing the method of Iscan, Loth and Wright for males (Iscan *et al.*, 1984) and females (Iscan *et al.*, 1985). A description of rib phases and their corresponding age ranges is found in Table 4.1 and Table 4.2 for males and females, respectively. The age ranges are the ones in the original publications reporting the method for males (Iscan *et al.*, 1984) and females (Iscan *et al.*, 1985). The age ranges reflect 95% confidence intervals derived from the statistical analysis of their data. Consequently, several age ranges overlap and a few ages are excluded.

Analysis involved the visual assessment of the morphology of each rib set and the subsequent placement of the ribs into a defined phase. In cases in which the ribs exhibited features consistent with two different, but sequential phases, both phases were reported. Each set of ribs was examined twice with the results recorded as “trial 1” and “trial 2”. A span of at least 5 days separated the two trials. Ribs were examined in groups of 50 and each group, with the exception of the first, consisted of ribs undergoing trial 1 examination as well as ribs undergoing trial 2 examination.

The process of examination consisted of Ms. Bourque, official data recorder, handing me, the observer, a set of ribs. Both ribs, left and right, were visually assessed to determine an appropriate phase. The only information given to me at the time of observation was whether the ribs were those of a male or female. At no time was I told whether I had previously examined the rib set or what my prior findings were.

<table>
<thead>
<tr>
<th>Ph</th>
<th>Yr</th>
<th>Shape of Pit</th>
<th>Morphology of Rim (walls of pit)</th>
<th>Condition of Bone</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>16-18</td>
<td>Amorphous indentation in articular surface. Billowing may still be present</td>
<td>Rounded, regular walls. May see beginning of scalloping</td>
<td>Firm and solid</td>
</tr>
<tr>
<td>2</td>
<td>20-23</td>
<td>“V” shaped</td>
<td>Thick and smooth walls. Scalloped or slightly wavy rim with rounded edges</td>
<td>Firm and solid</td>
</tr>
<tr>
<td>3</td>
<td>24-28</td>
<td>Narrow to moderate “U” shape</td>
<td>Thick walls with rounded edges. Some scallops may remain but more irregular rim.</td>
<td>Firm and solid</td>
</tr>
<tr>
<td>4</td>
<td>26-31</td>
<td>Narrow to moderately wide “U” shape but deeper pit than previous phase.</td>
<td>Thinner walls, rounded edges. Irregular edges with no regular scalloping remaining</td>
<td>Good condition but decrease in weight and firmness.</td>
</tr>
<tr>
<td>5</td>
<td>34-42</td>
<td>Moderately wide “U” shape. No change in pit depth.</td>
<td>Thinner walls with sharp edges. Scalloped pattern gone – replaced by irregular bony projections</td>
<td>Signs of deterioration with evidence of porosity and loss of density.</td>
</tr>
<tr>
<td>6</td>
<td>43-56</td>
<td>Deep wide “U” shape</td>
<td>Thin and sharp edges. Irregular rim with bony projections that are frequently more pronounced at the superior and inferior borders</td>
<td>Light in weight, thinner and more porous, especially inside the pit.</td>
</tr>
<tr>
<td>7</td>
<td>54-64</td>
<td>Deep, wide to very wide “U” shape</td>
<td>Walls thin and fragile. Sharp irregular edges with bony projections.</td>
<td>Light in weight, brittle and with obvious porosity.</td>
</tr>
<tr>
<td>8</td>
<td>65+</td>
<td>Very deep and widely “U” shaped. Floor may be absent or filled with bony projections</td>
<td>Extremely thin, fragile, and brittle with sharp, highly irregular edges and bony projections</td>
<td>Lightweight, thick, brittle, friable and porous. “Window” formation sometimes seen in walls.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Ph</th>
<th>Yr</th>
<th>Shape of Pit</th>
<th>Morphology of Rim (walls of pit)</th>
<th>Condition of Bone</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15-</td>
<td>Initial pit indentation with billowing of articular surface</td>
<td>Smooth, rounded, slightly wavy rim.</td>
<td>Firm and solid.</td>
</tr>
<tr>
<td>4</td>
<td>26- 31</td>
<td>Deeper flared “V” or “U” shape.</td>
<td>Central arc. Rounded rim with less pronounced scallops remaining.</td>
<td>Thinner walls with plaque like deposits in pit.</td>
</tr>
<tr>
<td>5</td>
<td>34- 46</td>
<td>Very flared “V” or “U”.</td>
<td>Irregular rim, sharpening edges. No regular scalloping remaining.</td>
<td>Appreciably thinner walls with plaque like deposits covering most of the pit.</td>
</tr>
<tr>
<td>6</td>
<td>43- 58</td>
<td>Deeper, wider “U” shape.</td>
<td>Central arc is less obvious. Sharp rim with start of irregular projections of bone.</td>
<td>Thinning walls with increased roughening and porosity inside the pit.</td>
</tr>
<tr>
<td>8</td>
<td>70+</td>
<td>Widely shaped “U”.</td>
<td>Extremely sharp, irregular rim with brittle projections of bone now prominent from superior and/or inferior margins of rib and floor of pit.</td>
<td>Thin, badly deteriorated, porous walls with window formation.</td>
</tr>
</tbody>
</table>
All data were recorded by Ms. Bourque on a preprinted Excel spreadsheet. The number of ribs examined in each sitting as well as the time span separating examination sessions facilitated a completely blind study in which I, the observer, had absolutely no way of recognizing rib sets or of knowing the results of previous trials.

Ribs, which were damaged during the maceration process thereby preventing analysis, were rejected and recorded as an “R” on the data sheet. A majority of the rejects came from the first batch of macerated ribs as the process was yet to be perfected. During the first maceration attempt, the ribs were cooked longer and at a higher temperature. This resulted in the softening of the bone and destruction of the morphological characteristics used to phase the ribs. Consequently, the maceration process was modified in subsequent cook downs and the problem was thereby eliminated.

In addition, two sets of ribs were determined by Ms. Bourque to be female based on a feminine first name. They were thus aged utilizing the female standards. At the completion of the aging trials, the investigator’s reports revealed that the decedents were actually male. This necessitated the rejection of both subjects.

Collection of drug abuse data

Following completion of trial 1 and trial 2 for each set of ribs, a list of case numbers used was assembled and the investigator’s report, death certificate, and autopsy findings were collected for each case. Determination of drug abuse history as well as pertinent autopsy findings was done independently of the aging trials. In other words, a determination of chronic drug abuse was made solely on information provided in the
Medical Examiner record and was not in any way based on, or influenced by, the data collected from the aging trials.

Substance abuse history included the drug of choice if known, and the length of time of suspected abuse. A drug was only recorded under “history”, if its use was known to be chronic. Cases ruled “no abuse history” had specific mention in the medical examiners record of “no known alcohol or drug abuse”. This information was related by family members, significant others, or friends, and substantiated by scene investigation, medical records, and autopsy findings. Cases that lacked any mention of abuse history (either its existence or its absence) were recorded as “unknown”. If drug or alcohol abuse was suspected but not confirmed, the case was still recorded as “unknown” under “history” but a comment was made to that effect in the “comments” section of the data sheet. Data distribution is displayed in Table 4.3.

<table>
<thead>
<tr>
<th>Males</th>
<th>Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unknowns</td>
<td>Abuse History</td>
<td>No Abuse History</td>
</tr>
<tr>
<td>27</td>
<td>46</td>
<td>32</td>
</tr>
<tr>
<td>8</td>
<td>19</td>
<td>23</td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td>65</td>
</tr>
</tbody>
</table>

In addition to abuse history, information regarding cause of death was obtained from the death certificate. A separate data column was utilized to record the specific drug or drugs when substances of abuse appeared on the death certificate as either the primary or contributing cause of death. If no drugs were directly involved in death, “none” was recorded in the column.
Every effort was made to specify the drug of choice when chronic illicit substance abuse was noted in the medical record. Non-illicit drugs of abuse, however, were grouped into a prescription drug abuse category. In addition, alcohol (EtOH) abuse was recognized and recorded separately.

Additional Variables

In addition to sex and drug abuse history, the "race" of each subject was obtained from the investigator's report. Determination of "race" follows medico-legal protocols and includes the following options: White, Black, Asian, Hispanic, Native American, and Other.

Data were also collected regarding the existence or absence of cardiac and pulmonary disease. Cardiac disease included arteriosclerotic cardiovascular disease (ASCVD), coronary atherosclerosis, hypertensive cardiovascular disease, cardiomyopathy, cardiac hypertrophy, or cardiomegaly. Pulmonary disease included chronic obstructive pulmonary disease (COPD), emphysema, or asthma. Data regarding cardiac disease, COPD, and emphysema were obtained from the autopsy report and only listed if the findings indicated moderate to severe disease. Data on asthma were obtained from the medical history. Existence of cardiac disease was indicated by a "C" on the data sheet; the type of disease was only specified in the comments section if it involved cardiac enlargement (cardiac hypertrophy or cardiomegaly). Existence of pulmonary disease was indicated by a "P" on the data sheet. The type of pulmonary disease was always specified in the "comments" section as all types can conceivably result in
enlargement of the thoracic cage. When disease was only “mild” or no disease was
detected at all, a recording of “none” was made on the data sheet.

All data were collated and recorded on an Excel spread sheet. Data used for
statistical analysis are presented in Appendix A. Information recorded in the “comments”
section and data relevant to the cause of death are included in the subject profiles
recorded in Appendix B.

Statistics

The goal of this study is to determine what factors, most notably chronic drug
abuse, influence a rater’s ability to establish an estimated age at death utilizing the sternal
end of the fourth rib. Consequently, the dependent variable is the assigned rib phase (and
associated age range). The independent variables include time (trial 1 and trial 2), drug
abuse history, race, sex, and thoracic disease. The break down of the sample by each
variable is presented in Table 4.4 at the end of the chapter.

The data are analyzed using the SPSS/PC 8.0 statistical package. Cross tabulation
tests are used to derive kappa (κ) values to measure agreement between trial 1 and trial 2
(intra-observer reliability) and between trial 1 and actual phase (accuracy). Thus, the
kappa statistic is used to determine the reliability of the method, defined as the
consistency or reproducibility of the phase estimations, and the validity of the method,
defined as how accurately the estimated phase compares to the actual phase.

Kappa (κ) is defined as agreement actually observed (Pa) minus the agreement
expected by chance (Pc), divided by one minus the agreement expected by chance (Fisher
and van Belle, 1993:257),
\[ \kappa = \frac{P_a - P_c}{1 - P_c} \]

A kappa value in excess of .75 indicates excellent agreement beyond chance. A kappa value between .40 and .75 indicates fair to good agreement and values below .4 indicate poor agreement. Negative values of kappa indicate less agreement than by chance and are interpreted as zero agreement.

The determination of accuracy or validity requires the use of either trial 1 or trial 2 phase estimations to compare to actual phase. Trial 1 is chosen and is used consistently throughout the statistical analysis.

Cross tabulation tables are generated for the overall data set and for each individual independent variable in order to determine what variables predict or interfere with an observer’s assessment of age utilizing the fourth rib method.

**Limitations**

The greatest limitation of the current study is the use of only one observer. This prevents a measurement of inter-observer reliability and assumes that the problems encountered by the observer are in fact universal in the application of the method.

The method for the determination of chronic drug abuse history also poses a limitation. Although every effort is made to obtain and verify the information as a standard procedure within the Medical Examiner’s office, there are possible sources of erroneous information. For example, when an adult male is reported to have had no history of drug or alcohol abuse by his wife, the assumption is made that he does not have any history that lies outside of her sphere of knowledge. The possibility exists that he had a history of substance abuse prior to meeting his wife or is engaged in covert use of illicit
substances. In other words, the information is only as good as its source. The hope is that by accessing as many sources as possible, the information obtained is in fact accurate.

The final limitation encountered by the current study involves the sample size. While the overall sample size is statistically stable, the individual subgroups within each variable, individual drugs or specific race for example, do not post numbers high enough to allow statistical analysis. This necessitates the combining of subgroups within each independent variable.

Table 4.4: Break-down of sample

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SEX</strong></td>
<td></td>
</tr>
<tr>
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CHAPTER 5
RESULTS

The data are subjected to crosstabulation tests to compute kappa values and
determine the reliability and accuracy of the method when applied to the entire data set
and each data subset. T-tests are performed for the individual variables of race, abuse
history, and thoracic disease in order to determine if a statistically significant difference
exists between the mean age in each data subset.

Complete Data Set

The crosstabulation table for trial 1 to trial 2 and associated kappa value for the
complete data set are recorded in Table 5.1. The crosstabulation results for trial 1 to
actual phase are recorded in Table 5.2. The kappa value of .661 for trial 1 to trial 2
indicates good agreement, or reliability. The kappa value of .177 for trial 1 to actual
phase indicates marginal agreement, or lack of accuracy.

Sex

The crosstabulation table for trial 1 to trial 2 and associated kappa values for
males and females are recorded in Table 5.3 and 5.5, respectively. The crosstabulation
tables for trial 1 to actual phase are recorded in Table 5.4 for males and Table 5.6 for
females.

The data supported use of all 8 phases in the computation of kappa for both males
and females. The kappa values of .611 and .754 for trial 1 to trial 2 for males and
females, respectively indicate good agreement, or reliability, for both sexes. The kappa
values of .147 for males and .237 for females indicate marginal agreement between
estimated phase and actual phase for both sexes.

Race

The sample sizes for Blacks, Asians, Hispanics, and Other were too small to allow
statistical analysis. Thus, Black, Asians, Hispanics, and Other were grouped into a “non-
White” category and statistics were run on the subsets of “White” versus “non-White”. In
addition, a lack of “non-White” individuals in phase 1 and phase 2 resulted in empty cells
in the crosstabulation tables and necessitated the use of phase 3 through phase 8 only in
the derivation of kappa for White versus Non-White. A t-test for equality of means
demonstrates a non-significant difference between the mean age of the two data sets
(p=.575).

The crosstabulation tables for trial 1 to trial 2 for Whites and Non-Whites are
recorded in Table 5.7 and 5.9, respectively. The kappa values of .658 for Whites and .641
for Non-Whites indicate good agreement for both groups. The crosstabulation tables for
trial 1 to actual age for Whites are presented in Table 5.8. The associated kappa value of
.180 indicates marginal agreement. The crosstabulation results for Non-Whites are
presented in Table 5.10. The kappa value of -.023 indicates less agreement than by
chance alone. This is interpreted as zero agreement between estimated phase and actual
phase for Non-Whites.

Abuse History

The requirement that drug abuse be chronic in order to constitute a positive
history resulted in a lack of drug abusers in the lower age ranges. Consequently,
crosstabulation tests could only be run utilizing phases 4 through 8. In addition, the sample size for each individual drug of abuse was too small to allow statistical analysis. Consequently, all drugs of abuse were considered equally and individuals with a positive history of abuse were grouped together as “drug abusers” and compared to “non-drug abusers”. A t-test for equality of means demonstrates a non-significant difference between the mean age of the two data sets (p=.799).

The results for trial 1 to trial 2 for drug abusers are recorded in Table 5.11. The crosstabulation results for trial 1 to trial 2 for Non-drug abusers are recorded in Table 5.13. The kappa value of .493 for drug abusers falls on the lower end of what is considered good reliability. The kappa value of .864 for non-drug abusers indicates excellent reliability or reproducibility.

The crosstabulation tables for trial 1 to actual phase for drug abusers and non-drug abusers are recorded in Table 5.12 and 5.14, respectively. Kappa values of .077 and .220 indicate marginal agreement between estimated phase and actual phase for each group.

**Thoracic Disease**

Because thoracic disease is associated only with the older subjects in the data set, statistical analysis could only be performed utilizing the data for phases 4 through 8. A t-test for equality of means demonstrates a significant difference between the mean age of the two data sets (p=.000).

The crosstabulation tables for trial 1 to trial 2 for subjects with thoracic disease are recorded in Table 5.15. An associated kappa value of .677 indicates good agreement.
between the two aging trials. The crosstabulation tables for trial 1 to trial 2 for subjects without thoracic disease are recorded in Table 5.17. An associated kappa value of .594 indicates good reliability.

The crosstabulation tables for trial 1 to actual phase for thoracic disease and no thoracic disease are recorded in Table 5.16 and Table 5.18 respectively. The kappa values of .042 and .180 reflect marginal agreement between estimated age phase and actual age phase for the two data sets.

A summary table listing all kappa values and corresponding sample sizes for each data set is presented in Table 5.19. Cases sorted by accuracy of assigned phase are presented in Table 5.20 and cases sorted by reliability of assigned phase are presented in Table 5.21. In table 5.20, one case is recorded in several rows under the column representing cases “missed by 4 phases”. The data represent a single case of a white, male, drug abuser with thoracic disease. This is the only case in the entire data set aged incorrectly by four phases.
TABLE 5.1: Trial 1 * Trial 2 Crosstabulation (Complete Data Set)

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<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
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Kappa = .661

TABLE 5.2: Trial 1 * Actual Phase Crosstabulation (Complete Data Set)

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TABLE 5.3: Trial 1 * Trial 2 Crosstabulation (Males)

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TABLE 5.4: Trial 1 * Actual Phase Crosstabulation (Males)

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**TABLE 5.5: Trial 1 * Trial 2 Crosstabulation (Females)**

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**TABLE 5.6: Trial 1 * Actual Phase Crosstabulation (Females)**

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TABLE 5.8: Trial 1 * Actual Phase Crosstabulation (Whites)

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### TABLE 5.9: Trial 1 * Trial 2 Crosstabulation (Non-Whites)

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Kappa = 0.641

### TABLE 5.10: Trial 1 * Actual Phase Crosstabulation (Non-Whites)

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Kappa = -0.023
### TABLE 5.11: Trial 1 * Trial 2 Crosstabulation (Drug Abusers)

<table>
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### TABLE 5.12: Trial 1 * Actual Phase Crosstabulation (Drug Abusers)

<table>
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TABLE 5.13: Trial 1 * Trial 2 Crosstabulation (Non-Drug Abusers)

<table>
<thead>
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TABLE 5.14: Trial 1 * Actual Phase Crosstabulation (Non-Drug Abusers)

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**TABLE 5.15: Trial 1 * Trial 2 Crosstabulation (Thoracic Disease)**

<table>
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**TABLE 5.16: Trial 1 * Actual Phase Crosstabulation (Thoracic Disease)**

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<td>3</td>
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<td>4</td>
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TABLE 5.17: Trial 1 * Trial 2 Crosstabulation (No Thoracic Disease)

<table>
<thead>
<tr>
<th>Kappa = .594</th>
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<tbody>
<tr>
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<td>7</td>
<td>3</td>
<td>11</td>
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<tr>
<td></td>
<td>Phase 7</td>
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<td>4</td>
<td>1</td>
<td>6</td>
</tr>
<tr>
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<td>Phase 8</td>
<td></td>
<td></td>
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<td>3</td>
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<tr>
<td>Total</td>
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<td>23</td>
<td>10</td>
<td>7</td>
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TABLE 5.18: Trial 1 * Actual Phase Crosstabulation (No Thoracic Disease)

<table>
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<td>11</td>
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<td></td>
<td>Phase 7</td>
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<td>1</td>
<td>4</td>
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<td></td>
<td>Phase 8</td>
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<tr>
<td>Total</td>
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<td>25</td>
<td>18</td>
<td>2</td>
<td>57</td>
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TABLE 5.19: Kappa Values and Sample Sizes

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<th>#T1xAA</th>
<th>K T1xAA</th>
</tr>
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<td>105</td>
<td>.147</td>
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<td>50</td>
<td>.237</td>
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<td>105</td>
<td>.180</td>
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<td>.641</td>
<td>28</td>
<td>-.023</td>
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<td>.180</td>
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(Note: The discrepancy in sample size between “T1xT2” and “T1xAA” for “Whites”, “Non-Drug Abusers”, and “No Thoracic Disease” reflects the fact that some cases were placed in the included phases in T1 and T2, however the actual phase fell below the phase cut-off. Consequently, several cases were lost in the comparison of “T1 to AA”).
Table 5.20: Cases Sorted by Accuracy of Assigned Phase.

<table>
<thead>
<tr>
<th>ACTUAL VERSUS PREDICTED</th>
<th>Correctly Phased</th>
<th>Missed by 1 Phase</th>
<th>Missed by 2 Phases</th>
<th>Missed by 3 Phases</th>
<th>Missed by 4 Phases</th>
<th>N</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>%</td>
<td>N</td>
<td>%</td>
<td>N</td>
<td>%</td>
</tr>
<tr>
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<td>28.6</td>
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<td>42.9</td>
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<td>18.1</td>
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<td>39.0</td>
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<td>18.1</td>
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<td>7.1</td>
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<td>15.5</td>
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<td>25.9</td>
<td>23</td>
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<td>17.2</td>
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</table>
### Table 5.21: Cases Sorted by Reliability of Assigned Phase

<table>
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<th>TRIAL 1 VERSUS TRIAL 2</th>
<th>Concordantly Phased</th>
<th>Missed by 1 Phase</th>
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<th>Missed by 3 Phases</th>
<th>Missed by 4 Phases</th>
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</tr>
</thead>
<tbody>
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<td></td>
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</tr>
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<td>1 1.0</td>
<td>0 0</td>
<td>0 0</td>
<td>105</td>
</tr>
<tr>
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<td>40 80.0</td>
<td>9 18.0</td>
<td>0 0</td>
<td>1 2.0</td>
<td>0 0</td>
<td>50</td>
</tr>
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<td>28 26.4</td>
<td>1 0.9</td>
<td>0 0</td>
<td>0 0</td>
<td>106</td>
</tr>
<tr>
<td>Non-Whites</td>
<td>20 71.4</td>
<td>8 28.6</td>
<td>0 0</td>
<td>0 0</td>
<td>0 0</td>
<td>28</td>
</tr>
<tr>
<td>Drug Abusers</td>
<td>36 62.1</td>
<td>22 37.9</td>
<td>0 0</td>
<td>0 0</td>
<td>0 0</td>
<td>58</td>
</tr>
<tr>
<td>Non-Drug Abusers</td>
<td>35 89.8</td>
<td>4 10.3</td>
<td>0 0</td>
<td>0 0</td>
<td>0 0</td>
<td>39</td>
</tr>
<tr>
<td>Thoracic Disease</td>
<td>44 75.9</td>
<td>14 24.1</td>
<td>0 0</td>
<td>0 0</td>
<td>0 0</td>
<td>58</td>
</tr>
<tr>
<td>No Thoracic Disease</td>
<td>43 70.5</td>
<td>18 29.5</td>
<td>0 0</td>
<td>0 0</td>
<td>0 0</td>
<td>61</td>
</tr>
</tbody>
</table>
Statistical analysis of the data set suggests that the utilization of the sternal end of the fourth rib is a reliable but not an accurate method of determining skeletal age at death. The high kappa (κ) values in the trial 1 to trial 2 analyses suggest that the method is descriptive enough to insure a low intra-observer error but not accurate enough to result in even moderate values of κ when comparing estimates to actual age. The variables of sex, race, and thoracic disease all appear to influence the accuracy of the method. Chronic substance abuse history appears to influence both the reliability and the accuracy of the method.

**Overall Data Set**

Table 5.1 illustrates that the method, when applied to a sample of 155 sets of ribs, results in high intra-observer reliability. This supports the premise that a range of variation exists in the morphology of the sternal end of the fourth rib and that visual assessment can be used to reliably assign the morphology to a given phase.

As illustrated in Table 5.21, discrepancies in predicted phase between trial 1 and trial 2 occurred in 44 of the 155 cases. In only two of the 44 cases did the prediction differ by more than one phase. Once again this supports the reliability of the method but suggests that the lines delineating the phases are not clear boundaries. This is illustrated by noting the difference in descriptive terminology between phase 5 and phase 6 for males. The features characteristic of phase 6 are the same as those of phase 5, they are only more extreme. Thus, an assignment of phase 5 versus phase 6 is conceivably
dependent on the observer’s experience and may understandably differ between trials. Most notably, the discrepancy of a single phase suggests that the phases may be too compartmentalized and that a combining of phases not clearly delineated may increase intra-observer reliability. The drawback to such an approach, however, is that it would likewise increase the associated age ranges thereby rendering the method less useful for practical applications.

As an alternative to combining phases, weighting the features reported for each phase could enhance the method. For example, if the ribs exhibit a pit shape consistent with phase 3 but a central arc consistent with phase 4, the researcher must necessarily question which feature is more indicative of actual phase. In one trial the observer may focus on the pit and thus assign the ribs to phase 3, while in the next trial the same observer may note the central arc and choose phase 4. A re-evaluation of the features most characteristic of each phase may provide better descriptive guidelines so that when such a case is encountered, the same features are recognized and utilized each time a trial is undertaken. Theoretically, this would decrease the number of single-phase discrepancies and result in increased intra-observer reliability.

Although the analysis of the overall data set demonstrates good intra-observer reliability, it unfortunately demonstrates poor agreement between estimated phase and actual phase. As illustrated in Table 5.2, only 49 of the 155 cases fell on the diagonal. As detailed in Table 5.20, 70 of the 155 cases (45.2%) differ by one phase between the estimated and the actual, while 36 cases (23.2%) differ by more than one phase.
Of those cases differing by more than one phase, eight are notable for having been assigned to phase 8 when actually belonging to phase 6, and seven for having been assigned to phase 8 but actually belonging to phase 5. Only one of the 15 cases was a female. Seven of the 15 cases, including the female, were drug abusers, which may introduce an additional variable. If analyzed in a practical context, the remaining eight individuals each would have been aged at over 65 years based solely on the morphology of the sternal end of the fourth rib. In actuality, the eight individuals were 34, 38, 38, 42, 42, 48, 48, and 51 years of age. This raises the question of why relatively young and healthy individuals are exhibiting traits characteristic of phase 8.

The ribs of the eight non-drug abusers in question were re-evaluated following the statistical analysis of the data. Although each set of ribs lacked the characteristically deteriorated bone comprising the floor and walls of the pit, they each exhibited bony extensions, sometimes extensive, that are characteristic only of phase 8. One of the eight cases was, by profession, a motorcycle rider suggesting possible biomechanical stresses resulting from repetitive posturing or motion. The other seven cases lack anything in their individual medical histories that would suggest a causal factor for the apparent advanced ossification of the costal cartilage. This implies that either the method inadequately accounts for the variation that exists within a single phase, or that life history factors independent of skeletal aging are acting to alter the morphology of the sternal end of the fourth rib. The latter is contrary to the premise upon which all aging methods are based. Specifically, skeletal aging methods rely upon a known, and therefore predictable, range of phenotypic variation associated with any given age range.
Interestingly, the same apparent problem was encountered by Loth (1995) when she applied the method to the Spitalfields cemetery population. She notes that for “numerous” specimens, an apparent contradiction between bone mass and morphology was observed. She cites her own notations as stating “rib looks phase 8, but feels younger” and concludes that “when this is the case in modern ribs, experience has shown that the firmness of the bone is significant and justifies assigning a younger phase despite older morphology” (Loth, 1995: 469). Unfortunately, no such explanation is offered in the descriptive parameters of the method as originally published (Iscan et al., 1984).

**Effects of Chronic Drug Abuse**

In following the trend established by the overall data set, the agreement between estimated phase and actual phase is poor for both drug abusers and non-drug abusers. This is illustrated in Tables 5.12 and 5.14, respectively. As demonstrated in Table 5.20, 15 of the 38 non-drug abusers (39.5%) differed by a single phase between the predicted phase and the actual phase. Only eight of the 38 cases (21.1%) differed by more than one phase. The eight cases are the males, previously discussed, that were all aged as a phase 8 based on contradictory skeletal indicators. Results for the drug abusers, on the other hand, are more variable with 27 of the 58 cases (46.6%) differing by a single phase, and 13 (22.4%) differing by more than one phase. This discrepancy is reflected by the kappa values of .220 for non-drug abusers versus .077 for drug abusers.

Data for drug abusers include six individuals that exhibit a phase 8 phenotype that are actually phase 5 or phase 6, two individuals aged at phase 4 or 5 that are actually phase 8, and four individuals aged at phase 4 that are actually phase 6. Additionally, the
data indicate that chronic drug abuse does not affect the accuracy with a consistent bias. Of the 40 cases that fall off the diagonal, 50% are over-aged and 50% are under-aged.

Chronic drug abuse is notable for affecting the reliability as well as the accuracy of the method. For non-drug abusers, cross tabulation of trial 1 to trial 2, illustrated in Table 5.13, generates a k value of .864. For drug abusers, however, the k drops to .493. As illustrated in Table 5.21, all discrepancies for drug abusers differ by a single phase, with ten cases under-aged in trial 2 versus trial 1 and 12 cases over-aged. This suggests that chronic drug abuse affects the morphology of the sternal end of the fourth rib in such a way as to influence the reliable placement of a set of ribs into a specific phase. The possibility that addictive behavior alters skeletal morphology of the sternal end of the fourth rib potentially invalidates the method which is based on a supposedly established and documented range of variation for any given phase. In addition, the demonstrated influence of an individual life history characteristic on a skeletal indicator of age at death poses questions regarding the validity of other aging methods based on morphological change.

Given that chronic drug abuse is associated with a wide array of infectious diseases and pathological disorders, it is not inconceivable that such behavior could indirectly result in alterations to the morphology of one or more structural units of the thoracic cage. Because the individual components of the thoracic cage (including the vertebrae, sternum, ribs, and costal cartilages) form an integrated and interdependent kinematic chain, the assumption is that stresses to one component may indirectly affect other structural units. For example, the bone mineral loss associated with chronic
alcoholism frequently occurs in the lumbar vertebrae resulting in vertebral crushing and pathological fractures (Bikle et al., 1985). The resulting loss of stability in the vertebral column may result in compensatory mechanisms that may impact the biomechanical stresses acting on the costal cartilages. Osteomyelitis and septic arthritis, both a documented complication of intravenous drug abuse, can manifest in the spine, costal cartilages, ribs, sternum, and sternal-clavicular joint (Holtzman and Bishko, 1971; Fishback et al., 1973; Goldin et al., 1973; Wiesseman et al., 1973; Zucker et al., 1974; Brittini et al., 1985; Lopez-Longo et al., 1986; Lopez-Longo et al., 1987; Endress et al., 1990; Walker and Pate, 1991). The other skeletal disorder associated with intravenous drug abuse, osteosclerosis, has been documented in the bones of the lower extremity (Villareal et al., 1992; Beyer et al., 1993). While not directly affecting the bones of the thoracic cage, the change in the morphology of the lower extremities may alter posture and locomotion thereby resulting in compensatory changes in the vertebral column which may in turn result in altered forces on the rib cage.

At the physiological level, alcohol abuse has been implicated in the reduction of bone mineral density and alterations in the plasma levels of the hormones most responsible for calcium homeostasis (Pitts and Van Thiel, 1986; Feitelberg et al., 1987; Laitinen et al., 1990; Abbott et al., 1994). In addition, alcohol abuse has been associated with a direct toxic effect on osteoblasts (Rico et al., 1987; Diamond et al., 1989; Labib et al., 1989; Laitinen and Valimaki, 1991; Chavassieux et al., 1993; Gonzalez-Calvin et al., 1993). Intravenous drug abuse of opiates in conjunction with altered immune defenses has been associated with changes in parathyroid hormone secretion (Teichmann et
114

al., 1997) and chronic abuse of opiates has also been implicated in altered bone metabolism and reduced trabecular bone mass (Pedrazzoni et al., 1993).

The sternal end of the fourth rib should not be considered the only skeletal age indicator susceptible to changes resulting from chronic drug abuse. Among intravenous drug users, septic arthritis is most common in the sternoclavicular, sternocostal, sacroiliac and pubic symphyseal joints (Lopez-Longo et al., 1987). All four joints are age indicators in the human skeleton. In addition, osteomyelitis has been reported in the pubic symphysis in connection with parenteral drug abuse (del Busto et al., 1982).

On a positive note, osteosclerosis associated with intravenous drug abuse has been put forth as evidence that skeletal mass can be increased with normal, structurally sound bone during adult life (Whyte et al., 1996). This has enormous implications for the study of osteoporosis (Villareal et al., 1992). Although rare in clinical circles, osteosclerosis has been postulated to be associated with the hepatitis C virus. If true, then the incidence of osteosclerosis may actually increase. The Seattle King County Department of Public Health reports that 83% of King County's intravenous drug abusers are hepatitis C positive.

Given that hepatitis can result in hepatic insult and given that chronic alcoholism is associated with liver cirrhosis, the impact of liver dysfunction is another variable for consideration in chronic substance abusers. Hepatic osteodystrophy is reported to occur in 50% of patients with chronic liver disease (Crosbie et al., 1999). Bone mineral density and levels of 25-hydroxycholecalciferol and 1,25-dihydroxycholecalciferol have been documented to be lower in patients with liver cirrhosis and patients with chronic
hepatitis, than in healthy controls (Tsuneoka et al., 1996). Even in the absence of chronic alcohol abuse, viral cirrhosis is associated with osteoporosis (Gallego-Rojo et al., 1998). In general, osteopenia and osteoporosis are clinically recognized complications of liver disease (Hussaini et al., 1999; Shiomi et al., 1999).

From a broader anthropological perspective, chronic drug abusers represent a subculture within the population worthy of additional study. The fact that intravenous drug abusers are infected by organisms not normally associated with hematogenous bone and joint infections (Holzman and Bishko, 1971) suggests they may suffer from decreased or suppressed immunological function. For example, *Serratia*, a bacterium recognized as a causal agent in osteomyelitis in intravenous drug abusers, was originally felt to be so harmless that it was intentionally used in hospitals as a marker organism to track bacterial movement (Ashby, 1976). Roca and Yoshikawa (1979) comment that osteomyelitis and septic arthritis are usually seen in older individuals suffering from chronic infections yet when related to heroin use, the disorders are seen in a younger and generally healthier subset of the population. A possible decreased immune capacity is further reflected by documented cases of osteomyelitis in heroin users seen as a secondary complication of trauma. Mandal and co-workers (1976) report on two cases of sternal osteomyelitis following a motor vehicle accident while Fox and Brady (1997) report on acute hematogenous osteomyelitis in a closed metatarsal fracture. Clearly, drug abusers are either more exposed to the causal agents than the general population, less immune to their effects, or a combination of both.
The possibility exists that the use of heroin or its adulterants in some way decreases humoral or cell-mediated immunity (Gifford et al., 1975). Shafer and colleagues (1990, 1991) have reported on diminished DNA repair, reduced immunoresponsiveness, and possible genotoxic damage in response to opiate abuse. Nutritional deficiencies may also play a role, as heroin abusers as a general rule practice poor health habits and exhibit an often high degree of dental decay (Pillari and Narus, 1973; Gambera and Clarke, 1976; Himmelgreen et al., 1998). Body weight is positively correlated with bone mineral density, particularly in postmenopausal women (Dawson-Hughes et al., 1987; Kin et al., 1991; Compston et al., 1992; Blanchet et al., 1998; Mazess and Barden, 1999). Crilly and Delaquerriere-Richardson (1990) propose that the maintenance of body weight protects against bone mineral loss and fracture in chronic alcoholics.

The increased incidence of infection presumably results in increased antibiotic use among intravenous drug abusers. Indeed, the possibility exists that the susceptibility of drug abusers to otherwise innocuous organisms may be the result of repeated antibiotic treatment in a hospital setting (Fishbach et al., 1973). Because many chronic drug abusers are seen on an urgent care basis and lack a regular physician for repeated follow-up care, many are non-antibiotic compliant. A common finding at death scenes involving a chronic drug abusing decedent is a medicine cabinet full of partially full antibiotic prescriptions. A lack of antibiotic compliance among drug users raises the possibility that as a subculture, chronic drug abusers may be contributing to the antibiotic resistant crisis currently facing the medical community.
Another interesting facet of the drug abuse culture is the possible social stratification that exists relative to awareness and acknowledgment of the behavior. During the collection of data regarding substance abuse history, a trend was noted whereby family members tended to be quite open about illicit substance abuse but often failed to admit or acknowledge chronic alcohol abuse. In several cases, autopsy findings suggested alcohol abuse although the family adamantly denied any significant abuse history. In other cases, information regarding alcohol abuse obtained from medical records directly contradicted family statements regarding substance abuse. The possible explanations are that either alcohol is not considered a drug of abuse or that alcoholism has different socio-economic connotations and is thus not openly discussed. Many decedents medically considered to have a history of alcoholism were otherwise high functioning individuals with a lucrative income and high social standing. Such is not the case with a majority of the heroin abusers in King County.

Sex as a variable contributing to phenotypic variation

Although males and females are evaluated using different phase descriptions and corresponding age ranges, the results for the two sexes divided are not unlike those of the overall data set. Both sexes exhibit high intra-observer reliability and low agreement between estimated phase and actual phase.

As demonstrated in Table 5.21, only one out of 105 male cases differs by more than one phase between the two trials. Likewise, only one of the 50 female cases differs by more than one phase. Interestingly, both cases are an actual phase 5 aged as a phase 2
for the female and a phase 3 for the male. Tables 5.4 and 5.6 illustrate the low agreement between estimated phase and actual phase for males and females, respectively.

As a subjective conclusion, this author found the phase descriptions for females less ambiguous, and therefore easier to use, than for males. This is potentially reflected by higher intra-observer reliability for females than for males. Likewise, while agreement between estimated and actual age is poor for both sexes, it is higher for females than for males.

In practical application, the need for sex-specific standards is only problematic if the sex of the individual is unknown. Determination of sex is required for many of the other aging methods, and thus is not a restriction unique to the rib aging technique. Why should a difference in costal ossification rates and/or patterns exist between males and females?

Perhaps the most obvious potential difference between males and females with regards to costal ossification is the relative effects of hormone levels. The identification of a specific pattern of ossification unique to postmenopausal women (McCormick and Stewart, 1983; Stewart and McCormick, 1984) suggests that ossification not only differs between the sexes, but also between premenopausal and postmenopausal women. Differences are also noted in the ossification rates in the cartilage of the first rib, which may reflect differential endocrine involvement (Semine and Damon, 1975). The effect of estrogen on bone density in general is reflected by the fact that bone loss of the whole skeleton and the spine in woman through life is calculated to be more than double when
compared to males (Nuti et al., 1995). Postmenopausal osteoporosis, involving rapid resorption and bone loss, is a condition seen only in women (Galloway, 1997).

In addition to the reproductive hormones, IGF-1 levels are reported to be sexually dimorphic, with males recording higher levels than females (Barrett-Connor and Goodman-Gruen, 1998). The significance of this is debated, however, as one study demonstrated a positive correlation between IGF-1 levels in the hip and spine in women but not men (Barrett-Connor and Goodman-Gruen, 1998), while another study reported a correlation between IGF-1 levels and bone mineral density of the spine in elderly men but not women (Janssen et al., 1998).

With regards to chronic substance abuse, studies suggest that moderate alcohol consumption actually increases bone mineral density in postmenopausal women (Felson et al., 1995). This conclusion is supported by a similar study employing an animal (rat) model (Fanti et al., 1997). This suggests the possibility that alcohol differentially affects men and women at the physiological level.

From a biomechanical standpoint, the possibility also exists that the stresses on the rib cage are directionally different in women as a result of the difference in body mass distribution. Semine and Damon (1975) demonstrated a correlation between chest circumference and ossification of the cartilage of the first rib in males and concluded that the correlation reflected a biomechanical stimulus for ossification. Likewise, Barchilon and colleagues (1996) suggested the same thing relative to the observed ossification in military pilots.
An additional biomechanical consideration is the difference between the sexes in the morphology of the structural units of the thoracic cage. Women reportedly have smaller vertebral bodies than men even after accounting for differences in body size (Gilsanz et al., 1997). A difference in vertebral body cross-sectional area is also observed between boys and girls throughout childhood and adolescence (Gilsanz et al., 1994a). The cross-sectional area is reported to increase 25-30% in adult males with aging. No such increase is reported for females (Mosekilde and Mosekilde, 1990). The smaller vertebral size in women may be biomechanically disadvantageous and related to the higher incidence of vertebral fractures in women (Gilsanz et al., 1994b).

Whether related to differential hormone levels, differences in body size and muscle mass, biomechanical factors or a combination of all three, the observed differences in costal cartilage ossification patterns necessitate separate methods for the aging of males and females.

**Race as a variable contributing to phenotypic variation**

Iscan and colleagues (1987) stated that modifications to the original method must be employed in the aging of blacks. This conclusion is supported by the results of the current study. The $\kappa$ values for trial 1 versus trial 2 are good for both groups (.641 for Non-Whites, .658 for Whites). The $\kappa$ values for rating 1 versus actual phase, however, differ between the two groups with a value of .180 for Whites and -.023 for Non-Whites. While agreement between estimated phase and actual phase is considered poor for Whites, agreement is zero for Non-Whites. As illustrated in Table 5.20, only 6 of the 28 (21.4%) Non-Whites were correctly phased while 17 (60.7%) were off by a single phase.
For Whites, 37 of the 105 (35.2%) were correctly phased while 41 (39%) were off by one phase. The high percentage of single-phase discrepancy between actual and predicted for Non-Whites supports the supposition of Iscan and colleagues (1987).

Recognition of race specific standards is not new in the field of anthropology (Trotter and Gleser, 1958). Michelson (1934) suggested a difference between blacks and whites in the rate of ossification of the first rib but admitted his inability to comment on the possibility that the results were actually a reflection of differing socio-economic factors. Odvina and colleagues (1995) proposed that prolonged alcohol abuse results in greater bone loss in whites than in blacks, suggesting a fundamental difference in physiological responsiveness to alcohol or in bone mineral densities.

The incidence of osteoporosis and pathological hip fractures is reported to be lower in blacks than in whites (Liel et al., 1988; DeSimone et al., 1989; Patal et al., 1992; Bell et al., 1995; Gilsanz et al., 1998). In an effort to elucidate the apparent "racial" differences, researchers have reported greater muscle mass and resulting skeletal mass in black women than in white women (Gasperino et al., 1995), and higher bone density (Luckey et al., 1989; Aloia et al., 1996; Pratt et al., 1996, Ettinger et al., 1997), better calcium retention (Anderson and Pollitzer, 1994; Daniels et al., 1997), and lower bone turnover (Weinstein and Bell, 1988) in blacks than in whites. Bone mineral content is also reported to be higher in black children than in white children (Li et al., 1989; McCormick et al., 1991). Lastly, bone mineral density is reported to be higher in black patients with end stage renal disease than in white patients (Stehman-Breen et al., 1999).
The differences between blacks and whites observed in a clinical and experimental setting may contribute to the apparent differences in the pattern of costal cartilage ossification. However, Semine and Damon (1975) found nutritional differences accounting for differences in cartilage ossification in genetically homogenous populations. In addition, recent work has demonstrated a possible correlation between bone aging and climatic factors (Belkin et al., 1998).

Arguably, the probable multifactorial etiology of costal cartilage ossification defies an explanation as simple as “racial” variation. Additional research is no doubt necessary to elucidate the contribution of ancestral affiliation to the phenotypic variance observed at the sternal end of the fourth rib.

**Thoracic Disease as a variable contributing to phenotypic variation**

Tables 5.15 and 5.17 present the results of cross tabulation for rating 1 versus rating 2 for cases of thoracic disease and cases without thoracic disease, respectively. The $\kappa$ values for both show good agreement, with .677 for thoracic disease and .594 in the absence of thoracic disease. Although both data sets show poor accuracy, the $\kappa$ value for cases of thoracic disease is only .042 compared to .180 for cases of no thoracic disease. What, then, is the contribution of thoracic disease to the ossification of the costal cartilages?

Pulmonary diseases, including asthma, emphysema, and chronic obstructive pulmonary disease, decrease the efficiency of ventilation and potentially stress the entire breathing mechanism. This conceivably may result in changes to the biomechanical stresses applied to the thoracic cage, and consequently may alter the pattern and/or timing
of costal cartilage ossification. In addition, the effects of prolonged prednisone treatment (Dearden and Mosier, 1975) suggest that the drugs used to treat chronic respiratory disease, many of which are steroid based, may also impact costal cartilage ossification. Reduction in bone mineral density has been documented in children with asthma who use corticosteroid therapy daily (Boot et al., 1997) and duration of oral glucocorticoid treatment has been found to be negatively correlated with bone mineral density of the rib and proximal femur (Ebeling et al., 1998).

Cardiovascular disease has also been associated with aberrant bone mineral density levels. In a study of 101 patients with severe congestive heart failure, Shane and colleagues (1997) found that approximately half exhibited osteopenia or osteoporosis. In addition, cardiovascular disease is often associated with a compromise in the vascular supply to tissues. This may influence bone at the metabolic level and potentially increase the rate or severity of age related changes. In addition, cardiovascular disease is often positively correlated with a decrease in activity levels and other age related disorders. Nordin and co-workers summarize the importance to the issue of age-related changes in bone,

On the other side of the bone turnover equation are those factors that tend to lower bone formation, notably declining muscular strength and activity possibly in turn related to declining androgen production and/or to vitamin D insufficiency and/or to such disabilities such as arthritis and heart disease (Nordin et al., 1998: 348).

The correlation between age and cardiovascular disease is reflected in the current study by a statistically significant difference in the mean age between the thoracic disease
group and the group without thoracic disease. This suggests that although differences in the accuracy of the method exist when applied to age determination in the two groups, the difference in mean age between the two groups cannot be discounted as a possible contributing factor.

Utility of the method

There are two underlying premises of the sternal end of the fourth rib method for the determination of age at death. First is that the costal cartilages ossify in a predictable and sequential pattern with advancing age. Second, the descriptive indicators defining each age phase must be all encompassing. In other words, the phase must account for the full range of phenotypic variation observed within the specified age range. The results of the current study refute both of the underlying premises of the method developed by Iscan and associates (1984) for the determination of age at death utilizing the sternal end of the fourth rib.

The assumption that descriptive indicators can adequately describe the full range of variation at any given phase assumes that individual life history events, including chronic substance abuse, do not significantly affect age estimates. Indeed, this is a fundamental premise of all methods of determining skeletal age at death. The finding that the method is less reliable and less accurate when applied to chronic substance abusers refutes the fundamental premise and suggests that individual life history events significantly contribute to the phenotypic plasticity of the sternal of the fourth rib. Thus, the full range of phenotypic variation, including what is encountered with chronic
substance abusers, would have to be documented and incorporated into the method in order to render the aging technique applicable in a practical context.

Despite the disappointing results of the current study, application of the method in a forensic as well as archaeological context is still plausible. When reporting the results of the multi-observer test of the method on white females, Iscan and Loth state that "individually, 22 of the 28 judges averaged 1 phase or less from ideal, and no one missed by more than 1.6 phases" (Iscan and Loth, 1986b:993). Similarly, in the test of the method as applied to males, the authors state that "fifteen of the twenty-five judges averaged within one phase of the ideal" (Iscan and Loth, 1986a: 125). Although not directly comparable, the proportion of cases in the current study in which the correct phase or a single phase discrepancy was predicted ranged from 63.8% for the thoracic disease group to 91.2% for the group without thoracic disease (see Table 5.20). Even the results for the drug abusers and non-drug abusers were similar in percentage at 77.6% and 79.0%, respectively. Thus, the poor results of the current study may reflect the stringent research design. Kappa values in the current study reflect correlations between the actual phase and the predicted phase with no allowance for estimates deviating by a single phase.

In a similar study, Baccino and Souaiby (2000) report that when the method is utilized to determine age at death on a French sample of 131 Caucasian male forensic cases, the kappa values are similar to those reported here (κ=.38 for observer 1, κ=.29 for observer 2). The authors conclude that the method is not fit for identification purposes in a French sample. However, they report that the combining of phases (i.e., using a phase
3-4 or 4-5) significantly increases the accuracy of the method. The obvious drawback, however, is that accuracy is increased at the expense of smaller age ranges. Given that in the current study 50% of drug abusers are over-aged and 50% are under-aged, such an approach would necessitate the combining of three phases (the one above and the one below) in order to accommodate the possibility that the decedent is a chronic substance abuser.

Another consideration regarding the utility of the method concerns the comparison of the experimental conditions to those encountered in practical situations. The current study relied upon the ribs, and the ribs alone, to estimate skeletal age at death. Rarely is this the chosen method in practical applications. Usually, the condition of the overall skeleton is noted in conjunction with as many skeletal indicators as are present. One must question if the eight males incorrectly aged as a phase 8 had been evaluated in a practical context, would analysis of the skeleton as a whole have altered the age assessment? In other words, would the knowledge that the remainder of the skeleton lacked indicators of advanced age somehow have tempered the way in which the contradictory features of the sternal end of the fourth rib were interpreted? While this supposition does not exonerate the lack of accuracy generated by utilizing the method alone, it does suggest that the method would be more useful when used in conjunction with other skeletal indicators of age at death.

Lastly, this technique is not entirely without merit. The sternal end of the fourth rib does indeed change morphology as an individual ages. Thus, a greater understanding of the full range of phenotypic variation at each phase coupled with an exploration of the
underlying sources of that variation may serve to transform the method into a useful tool
in a multi-factorial approach to estimating skeletal age at death.

Future Research

As the current study has demonstrated, a greater understanding of the full range of
phenotypic variation in the sternal end of the fourth rib is necessary in order to make any
aging method utilizing the fourth rib useful in practical applications. Given that the
method utilized in this study and devised by Iscan and colleagues was only based on a
sample size of 118 males (Iscan et al., 1984) and 86 females (Iscan et al., 1985), the
possibility of re-evaluating the method utilizing a larger sample size would be a
worthwhile endeavor. A collection of several thousand sets of ribs with a known age at
death and known history would enable the researcher to better describe the full range of
phenotypic variation that exists in each age phase. The hope that the pattern of change at
the sternal of the fourth rib can be better documented and described motivates the
continued collection and evaluation of ribs as the next phase of the current study.
Additionally, the current study was limited by a small sample size that necessitated a
grouping of cases of alcohol abuse with cases of illicit substance abuse. A larger sample
size will facilitate the separate consideration of the variables imposed by a history of
illicit drug use versus chronic alcohol abuse.

The contribution of biomechanical forces to the patterned change at the
costochondral junction could be further explored by examining the ribs of individuals
with limited upper body mobility (i.e., quadriplegics), as well as those with extreme load
bearing stress (i.e., weight-lifters). Likewise, the effects of steroid hormones on the
ossification of costal cartilages may be demonstrated by individuals undergoing extreme hormone therapy (i.e., gender converts). Rib collection from each of the above sample categories is incorporated into the ongoing research design.

Given that a decrease in bone mineral density is a documented change with advancing skeletal age, particularly in women, the question arises as to the correlation between bone mineral loss and age-related morphological change. Iscan and associates discuss the periosteal deposition of bone, possibly accompanied by perichondral mineralization, and endosteal resorption as possible causal factors involved in the morphological change of the sternal end of the fourth rib (Iscan et al., 1984). How, though, does an overall decrease in bone mass, a decrease in vertebral bone mineral density, or an imbalance in calcium homeostasis influence the patterned change in morphology at the costochondral junction? A research protocol in which the ribs are examined in conjunction with measurement of bone mineral content at the rib and/or vertebrae may elucidate the relationship between the two. This in turn would shed light on the accuracy of the method when applied to individuals likely to be exhibiting decreased bone mineral density because of disease or chronic alcoholism.

Two areas of interest not addressed by the current study include the effects of substance abuse on developing bone and the long-term effects of drug abuse following abstinence. Data from an animal model suggest that young alcohol fed-rats exhibit a decrease in bone mass (Sampson et al., 1996), a decrease in bone stiffness and strength (Hogan et al., 1997), and a dramatic depression in growth, growth rate, and hypertrophic cell proliferation at the growth plate (Sampson et al., 1997). The fact that alcohol is a
popular substance of abuse among American youth suggests that studies examining the
effects of alcohol on growing bone may have important implications for the analysis of
skeletal morphology later in life.

Alcohol has also been studied relative to its effects following cessation of abuse.
Pepersack and colleagues (1992) found a rapid increase in bone Gla protein following
ethanol withdrawal, suggesting that the toxic effect of alcohol on osteoblasts ceases when
alcohol intake ceases. Although disturbances in bone metabolism relative to alcohol
abuse are reported to be reversible (Lindholm et al., 1991), bone loss in the lumbar spine
and femoral neck is reported in abstinent alcoholics four months (Chon et al., 1992) and
two years following cessation of consumption (Peris et al., 1994). Nyquist and co-workers
(1996) report that the high bone turnover rate associated with alcohol abuse may still be
present five years after withdrawal. The long term effects of alcohol abuse following
withdrawal have enormous implications for the interpretation of skeletal morphology in
individuals reported with no current substance abuse, or in those for whom medical
history is unknown.

Another variable that may possibly impact skeletal change at the costochondral
junction is the use of tobacco. Heavy smoking has been associated with bone loss in men
(Ortego-Centeno et al., 1997) and women (Hopper and Seeman, 1994). Smoking also has
an apparent affect on calcium and vitamin D metabolism (Brot et al., 1999). The current
study did not consider smoking as a variable, but perhaps future studies need to consider
smoking duration and quantity as variables possibly impacting skeletal health.
In addition to the determination of age at death, the skeletal effects of chronic substance abuse may impact other areas of forensic and/or anthropological investigation. For example, a current study at the King County Medical Examiner's Office is looking at the validity of chest radiographs for the determination of positive identification. Many of the subjects studied are in fact chronic alcoholics. Because alcohol abuse is demonstrated to impact the bone mineral density of the lumbar vertebrae, and because morphological characteristics of the spine are often used in identification, the possibility exists that chronic alcoholism will prove to be a hindrance to this method of positive identification. The results of the study will hopefully offer additional insights into the importance of alcohol abuse in a forensic setting.

The current research has also raised many questions regarding the prevalence and etiology of drug abuse. Williams and Neese (1991) have expounded on the benefits of applying a research paradigm based on evolutionary theory to the study of disease control. Undoubtedly, the study of the complex social, medical and biocultural phenomenon of drug abuse would benefit from such an approach. Generally, there are three possibilities: (1) addictive behavior is genetically based with drug addiction as one possible phenotypic expression, (2) addictive behavior has no genetic contribution in which case such behavior is solely a manifestation of environmental (including cultural) forces, or (3) addictive behavior results from an interaction between genetic factors and environmental variables. If number one is the case, then a comparison of the relative fitnesses between drug abusers and non-drug abusers may help explain the prevalence of the behavior. If number two is the case, then drug abuse is a cultural manifestation and
attempts at intervention should recognize it as such. If number three is the case, then the
transmission of cultural factors, including ideas regarding the acceptance of substance
abuse, may track with the incidence of addictive behavior. In other words, drug abuse
may be an example of co-evolution, which would in turn explain why drug abuse within a
family is often a multi-generational phenomenon.

Clearly, additional studies on the effects of alcohol and illicit drug abuse are
necessary to elucidate the contributions of addictive behavior to phenotypic variation.
The possibility that chronic substance abuse alters skeletal anatomy and the morphology
of the skeletal indicators of age at death has implications for paleodemographic studies
and forensic investigations. The possibility that behavior can alter skeletal indicators
must be considered by every researcher engaged in the interpretation of skeletal
morphology, regardless of whether the specimen in question is an Australopithecine or an
anatomically modern human.
APPENDIX A
DATA SHEET

Column 1
Code number assigned by Ms. Bourque following maceration but before analysis. Missing code numbers correspond to cases rejected because of damage incurred during maceration or incorrect initial sex identification.

Columns 2-4
Phase number based on the age ranges for rib phases as follows:

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Column 2
Chosen phase for the rib pair during the first analysis (trial 1).

Column 3
Chosen phase for the rib pair during the second analysis (trial 2).

Column 4
Actual phase based on the actual age of the decedent. A designation of "void" indicates that the actual age does not fall into the specified age range for any listed phase.

Column 5
Actual age of the decedent (in years).

Column 6
Sex of the decedent:
M=Male
F=Female.
**Column 7**
Race of the decedent:
- W=White
- B=Black
- A=Asian
- H=Hispanic
- N=Native American
- O=Other

**Column 8**
History of chronic (in excess of 3 years) substance abuse with substances coded as follows:
- A=Alcohol (EtOH)
- C=Cocaine
- D=Drug abuse (not EtOH); drug not specified
- I=Intravenous drug abuse (IVDA); drug not specified
- J=Marijuana
- M=Methamphetamine
- N=None
- O=Opiate
- P=Prescription medications
- X=Other (includes OTC drugs, ethyl chloride)
- U=Unknown history (i.e., Cannot be substantiated by the statements of family or friends, medical records or autopsy findings). If drug abuse is suspected (but cannot be confirmed), a mention is included in the subject profile (Appendix B).

**Column 9**
Thoracic disease coded as follows:
- C=Cardiac disease (includes arteriosclerotic cardiovascular disease (ASCVD), coronary atherosclerosis, hypertensive cardiovascular disease, cardiomyopathy, cardiac hypertrophy, cardiomegaly). Only recorded if findings on autopsy report indicate moderate to severe.
- P=Pulmonary disease (includes chronic obstructive pulmonary disease (COPD), emphysema, asthma).
- N=None. Disease processes detected at autopsy are either "mild" or non-existent.
- U=Unknown. The autopsy report was not available at the time of data collection so the existence or absence of thoracic disease is undocumented.
Specific thoracic disease is specified for each subject in the subject profiles (Appendix B).
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APPENDIX B
SUBJECT PROFILES

Subject 1:
A 21 year old White female with an undocumented history of abuse. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.

Subject 2:
An 18 year old White male with an undocumented history of abuse. The cause of death is cerebral contusions and liver and kidney lacerations and pelvic fracture due to blunt force injury of head and trunk. The manner of death is accident (traffic).

Subject 3:
A 24 year old Black female with an undocumented history of abuse. The cause of death is gunshot wound of the head. The manner of death is homicide.

Subject 4:
Rejected due to damage incurred during the defleshing process.

Subject 5:
A 44 year old White male with a documented history of alcohol abuse. The cause of death is acute subdural hematoma due to blunt force injury of the head. A contributing condition is coagulopathy due to alcoholic cirrhosis. The manner of death is accident (apparent fall).

Subject 6:
A 31 year old White male with a documented 17 year history of intravenous drug (type unspecified) abuse. The cause of death is anoxic encephalopathy and bronchopneumonia due to hanging by ligature about neck. The manner of death is suicide.

Subject 7:
A 27 year old Black male with an undocumented history of abuse. Toxicology findings are significant for cocaine and ethanol on board. The cause of death is basal skull fracture with cerebral lacerations and contusions due to blunt force injury of the head. The manner of death is accident (traffic).

Subject 8:
A 44 year old Black male with an undocumented history of abuse. Autopsy findings are significant for cardiomegaly. The cause of death is acute intoxication due to the combined effects of opiate and cocaine. The manner of death is accident.
Subject 9:
A 22 year old Native American female with an undocumented abuse history. Medical history is significant for conflicting (and therefore undocumented) reports about drug and alcohol abuse. The cause of death is massive hepatic necrosis, etiology uncertain. The manner of death is undetermined.

Subject 10:
A 39 year old White Male with an undocumented history of abuse. The cause of death is asphyxia due to brackish water drowning. The manner of death is accident.

Subject 11:
A 92 year old White male with no known history of alcohol or drug abuse. Autopsy findings are significant for emphysema. The cause of death is acute subdural hematoma and cerebral contusions due to blunt force injury of the head. The manner of death is accident.

Subject 12:
A 55 year old White male with undocumented abuse history. Autopsy findings are significant for atherosclerotic cardiovascular disease. The cause of death is diffuse pulmonary consolidation of adult respiratory distress syndrome (ARDS) as a consequence of thermal injuries involving 80% of body surface area. The manner of death is accident (flash fire while trying to light stove).

Subject 13:
A 64 year old White female with no known history of alcohol or drug abuse. The cause of death is cardiomegaly with left ventricular hypertrophy. The manner of death is natural.

Subject 14:
A 50 year old Hispanic male with a documented history of alcohol, opiate, cocaine and prescription drug abuse. Medical history is significant for chronic obstructive pulmonary disease (COPD). The cause of death is atherosclerotic coronary artery disease with a contributing condition of upper gastrointestinal hemorrhage. The manner of death is natural.

Subject 15:
A 26 year old Native American female with a documented history of heroin abuse. The cause of death is acute intoxication due to the combined affects of opiate and cocaine. The manner of death is accident.

Subject 16:
A 41 year old White male with an undocumented abuse history. The cause of death is hemoperitoneum and hemothorax as a consequence of splenic lacerations and multiple rib fractures due to blunt force injury of the trunk. The manner of death is accident (traffic).
Subject 17:
A 29 year old female of unspecified race with a documented history of heroin abuse. The cause of death is sepsis due to an abscess and necrotizing fascitis of the thigh as a consequence of subcutaneous injection abuse of black tar heroin. The manner of death is natural.

Subject 18:
A 38 year old White male with a documented history of alcohol and heroin abuse. The cause of death is acute intoxication due to the combined effects of opiate, doxepin, nordiazepam, chlordiazepoxide and ethanol. The manner of death is probable accident.

Subject 19:
Rejected due to damage incurred during the defleshing process.

Subject 20:
A 52 year old White male with a documented history of alcohol abuse. Autopsy findings are significant for atherosclerotic cardiovascular disease. The cause of death is acute subarachnoid hemorrhage due to spontaneous rupture of cerebral aneurysm. The manner of death is natural.

Subject 21:
A 46 year old White male with a documented history of chronic opiate and alcohol abuse. Medical history is significant for emphysema and arteriosclerotic cardiovascular disease. The cause of death is acute intoxication due to the combined effects of opiate and ethanol. The manner of death is accident.

Subject 22:
A 92 year old Asian Pacific Male with no known alcohol or drug abuse history. Autopsy findings include hypertensive and atherosclerotic cardiovascular disease. The cause of death is asphyxia as a consequence of choking on a food bolus. The manner of death is accident.

Subject 23:
A 44 year old White female with a documented history of alcohol and drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of cocaine, sertraline and trazodone. The manner of death is accident.

Subject 24:
A 60 year old White male with no known history of alcohol or drug abuse. Autopsy findings include moderate pulmonary emphysema, and atherosclerotic coronary disease. The cause of death is asphyxia due to hanging by ligature. The manner of death is suicide.
Subject 25:
A 17 year old White female with a known history of limited drug use and no known history of alcohol abuse. The decedent is stated to have experimented only with speed, acid, shrooms and marijuana. Drug use is not chronic and therefore not considered drug abuse. The information on experimental drug use was related by friends of the decedent and was apparently unknown to the legal next of kin (parents). The cause of death is cerebral cortical white matter and midbrain contusions due to blunt force injury to the head. The manner of death is accident (pedestrian struck by motor vehicle).

Subject 26:
A 59 year old Hispanic male with an undocumented abuse history. Autopsy findings include cirrhosis of the liver and atherosclerotic cardiovascular disease. A transient lifestyle and a finding of hepatic cirrhosis suggest probable ethanol abuse. The cause of death is asphyxia as a consequence of choking on a food bolus. The manner of death is accident.

Subject 27:
A 56 year old White male with an undocumented abuse history. The cause of death is hemopericardium due to dissection of ascending aorta with rupture as a consequence of hypertensive cardiovascular disease. The manner of death is natural.

Subject 28:
A 51 year old Native American male with a documented history of chronic alcohol abuse. Previous hospital admits record a blood alcohol level as high as .551. Autopsy findings are significant for fatty liver. The cause of death is acute ethanol intoxication. The manner of death is accident.

Subject 29:
A 49 year old White female with no known history of drug or alcohol abuse. The cause of death is acute intoxication due to the combined effects of carbamazepine, paroxetine, propranolol, amitriptyline, phenobarbital, diazepam and oxycodone. Other significant conditions include atherosclerotic cardiovascular disease. The manner of death is suicide.

Subject 30:
A 27 year old White female with no known history of drug or alcohol abuse. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.

Subject 31:
Rejected due to damage incurred during the defleshing process.
Subject 32:
A 63 year old White male with a documented history of chronic alcohol abuse. The cause of death is asphyxia due to fresh water drowning. Other significant conditions include arteriosclerotic cardiovascular disease and fatty liver due to chronic ethanolism. The manner of death is accident.

Subject 33:
Rejected due to damage incurred during the defleshing process.

Subject 34:
A 66 year old White male with an undocumented abuse history. Autopsy findings are significant for cardiac hypertrophy. The cause of death is arteriosclerotic cardiovascular disease. The manner of death is natural.

Subject 35:
A 48 year old White female with no known history of drug or alcohol abuse. The cause of death is hypertensive cardiovascular disease. The manner of death is natural.

Subject 36:
Rejected due to damage incurred during the defleshing process.

Subject 37:
Rejected due to damage incurred during the defleshing process.

Subject 38:
Rejected due to damage incurred during the defleshing process.

Subject 39:
Rejected due to damage incurred during the defleshing process.

Subject 40:
A 50 year old White male with a documented history of chronic alcohol abuse. Autopsy findings are significant for alcoholic cirrhosis of the liver. The cause of death is gastrointestinal hemorrhage due to ruptured esophageal varices as a consequence of micronodular cirrhosis with fatty change. The manner of death is natural.

Subject 41:
Rejected. The decedent was initially identified and evaluated as a female based on a feminine first name. The fact that the decedent is actually a male was not discovered until records were collected following data collection. The subject is therefore rejected.
Subject 42:
A 42 year old Black female with an undocumented abuse history. Autopsy findings are significant for fatty transformation of the liver and a positive cocaine toxicology screen. The cause of death is marked pulmonary edema as a consequence of acute hepatic necrosis of uncertain etiology. The manner of death is natural.

Subject 43:
A 28 year old White female with a documented history of alcohol, opiate and prescription drug abuse. The cause of death is asphyxia as a consequence of the inhalation of the products of combustion. The manner of death is accident (house fire).

Subject 44:
A 40 year old White female with a documented history of alcohol abuse. The cause of death is not explained by autopsy and is thus listed as no anatomical or toxicological cause of death. The manner of death is natural.

Subject 45:
A 23 year old White male with an undocumented abuse history. The cause of death is hypothermia due to cold water immersion. The manner of death is accident.

Subject 46:
A 39 year old White female with an undocumented history of abuse. Alcohol abuse is suspected and supported by the finding at autopsy of micronodular cirrhosis. The cause of death is anoxic encephalopathy due to near fresh water drowning. Other significant conditions include acute intoxication due to the combined effects of ethanol and benzodiazepine. The manner of death is accident.

Subject 47:
A 46 year old White male who died in an alcohol recovery center. The decedent had a documented history of chronic alcohol abuse. The cause of death is cirrhosis due to chronic ethanolism. The manner of death is natural.

Subject 48:
A 35 year old White male with a documented 15 year history of alcohol and cocaine abuse. The cause of death is arteriosclerotic cardiovascular disease with myocardial scarring. Other significant conditions include acute cocaine intoxication. The manner of death is accident.

Subject 49:
A 52 year old White male with a documented history of alcohol abuse. Autopsy findings are significant for fatty liver. The cause of death is arteriosclerotic cardiovascular disease. The manner of death is natural.
Subject 50:
A 23 year old White female with no known history of alcohol or drug abuse. The cause of death is lacerations of the spleen and liver due to blunt force injury to the trunk. The manner of death is accident (traffic).

Subject 51:
A 35 year old White male with a documented history of prescription drug abuse. The cause of death is acute intoxication due to the combined effects of alcohol and alprazolam. The manner of death is undetermined.

Subject 52:
A 56 year old White male with an undocumented history of abuse. The cause of death is skull fracture and cerebral contusion due to blunt force injury to the head. The manner of death is accident (struck by falling tree).

Subject 53:
A 79 year old White female with no known history of drug or alcohol abuse. The cause of death is spinal contusion due to cervical spine fracture as a consequence of blunt force injury of the head and neck. The manner of death is accident (fall down stairs).

Subject 54:
A 45 year old Black female with a documented history of alcohol, intravenous drugs (unspecified), and cocaine abuse. Medical history is significant for esophageal varices due to chronic ethanolism. The cause of death is bilateral bronchopneumonia with cerebral contusions due to blunt force injury of head, trunk and extremities. Contributing conditions include cirrhosis with hepatic encephalopathy. The manner of death is accident (pedestrian struck by motor vehicle).

Subject 55:
A 46 year old White female with no known history of drug or alcohol abuse. The cause of death is not identified by autopsy and thus stated as no anatomical or toxicological cause of death. The manner of death is natural.

Subject 56:
A 30 year old Black female with an undocumented abuse history. The cause of death is complications of thermal injuries. The manner of death is homicide (arson fire).

Subject 57:
Rejected due to damage incurred during the defleshing process.

Subject 58:
A 59 year old White male with no known history of drug or alcohol abuse. The cause of death is atherosclerotic cardiovascular disease. The manner of death is natural.
Subject 59:
Rejected due to damage incurred during the defleshing process.

Subject 60:
A 42 year old White female with an undocumented history of abuse. The cause of death is acute opiate intoxication. The manner of death is accident.

Subject 61:
Rejected due to damage incurred during the defleshing process.

Subject 62:
A 45 year old Black male with an undocumented abuse history. Autopsy findings are significant for cocaine on board. The cause of death is diabetes mellitus with ketoacidosis. The manner of death is natural.

Subject 63:
A 31 year old White male with a 10 year documented history of opiate, cocaine, methamphetamine and marijuana abuse and a 5 year documented history of alcohol abuse. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.

Subject 64:
A 67 year old White male with a documented history of alcohol, opiate and cocaine abuse. Marijuana was found on his person at the time of death. The cause of death is hypertensive atherosclerotic cardiovascular disease with a contributing condition of bronchogenic carcinoma. The manner of death is natural.

Subject 65:
A 34 year old White female with a documented history in excess of 16 years of intravenous heroin abuse. The cause of death is acute intoxication due to the combined effects of opiate, venlafaxine, and phenobarbital. The manner of death is undetermined.

Subject 66:
Rejected due to damage incurred during the defleshing process.

Subject 67:
A 38 year old White male with a documented history of intravenous drug (unspecified) abuse. Autopsy findings are significant for left ventricular hypertrophy. The cause of death is acute intoxication due to the combined effects of opiate, cocaine and diazepam. The manner of death is probable accident.
Subject 68:
A 37 year old White male with an undocumented abuse history. Recent intravenous drug abuse is evidenced by needle puncture wounds in the arms. The cause of death is acute intoxication due to the combined effects of opiate and ethanol. The manner of death is probable accident.

Subject 69:
Rejected due to damage incurred during the defleshing process.

Subject 70:
A 51 year old White male with a documented history of alcohol and drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of opiate, cocaine and alcohol. The manner of death is accident.

Subject 71:
Rejected due to damage incurred during the defleshing process.

Subject 72:
Rejected due to damage incurred during the defleshing process.

Subject 73:
A 13 year old White female with no known history of alcohol or drug abuse. The cause of death is bilateral acute subdural hemorrhage and multiple cerebral contusions due to blunt force injury of the head. The manner of death is accident (traffic).

Subject 74:
A 43 year old White male with an undocumented history of abuse. Decedent ascribed to a transient lifestyle. The cause of death is arteriosclerotic cardiovascular disease with a contributing condition of pulmonary emphysema. The manner of death is natural.

Subject 75:
A 21 year old White male with an undocumented history of abuse. The cause of death is the inhalation of the products of combustion. The manner of death is accident (boat fire).

Subject 76:
A 57 year old White female with a 20 year history of alcohol abuse. The cause of death is multiple rib fractures and splenic laceration due to blunt force injury to the trunk. Other contributing conditions include alcoholic liver disease with cirrhosis. The manner of death is accident (traffic).

Subject 77:
A 32 year old Black female with no known history of drug or alcohol abuse. The cause of death is seizure disorder due to cerebral vascular malformation. The manner of death is natural.
Subject 78:
A 25 year old White male with no known history of drug or alcohol abuse. Medical history is significant for asthma since the age of two. The cause of death is status asthmaticus due to bronchial asthma. The manner of death is natural.

Subject 79:
A 51 year old White male with no known history of drug or alcohol abuse. The cause of death is asphyxia due to fresh water drowning. The manner of death is accident.

Subject 80:
A 38 year old White female with no known history of drug or alcohol abuse. Medical history includes Treacher Collins Syndrome (mandibulofacial dysostosis) and recent cervical (C4-6) discectomy and fusion. The cause of death was not revealed at autopsy and is therefore listed as no anatomical or toxicological cause of death. The manner of death is natural.

Subject 81:
A 33 year old White male with an undocumented abuse history. The decedent is most probably negative for abuse as he was cleared and accepted for organ donation. However, specific comments regarding abuse history are not documented. The cause of death is diffuse subarachnoid hemorrhage due to rupture of a berry aneurysm at the basilar tip. The manner of death is natural.

Subject 82:
A 53 year old White male with a documented history of intravenous drug (not specified) and alcohol abuse. Autopsy findings significant for moderate coronary atherosclerosis. The cause of death is acute opiate intoxication. The manner of death is accident.

Subject 83:
A 28 year old White male with an undocumented abuse history. The cause of death is skull fracture and brain contusions due to blunt force injury to the head. The manner of death is accident (fall).

Subject 84:
A 50 year old White male with a documented history of drug (unspecified) abuse. Autopsy findings are significant for macronodular cirrhosis. The cause of death is acute intoxication due to the combined effects of methamphetamines, methadone, floxetine, promethazine and propoxyphene. The manner of death is probable accident.

Subject 85:
A 42 year old White female with a documented history of alcohol and prescription drug abuse. The cause of death is acute propoxyphene intoxication. The manner of death is accident.
Subject 86:
An 18 year old White male with an undocumented abuse history. The cause of death is lacerations and contusions of the brain due to blunt force injury of the head. The manner of death is accident (traffic).

Subject 87:
A 23 year old Asian female with no known history of alcohol or drug abuse. The cause of death is acute subdural hematoma and cerebral contusions due to blunt force injury of the head. The manner of death is accident.

Subject 88:
A 44 year old White male with a documented history of alcohol, marijuana and intravenous drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of opiate and ethanol. The manner of death is probable accident.

Subject 89:
A 46 year old White male with an undocumented abuse history. Autopsy findings are significant for cardiomegaly. The cause of death is acute ethyl chloride intoxication. The manner of death is accident.

Subject 90:
A 43 year old Hispanic male with a documented history of cocaine abuse. Autopsy findings are significant for hypertensive cardiovascular disease and left ventricular cardiac hypertrophy. The cause of death is acute cocaine intoxication. The manner of death is accident.

Subject 91:
A 46 year old Hispanic male with an undocumented abuse history. The cause of death is multiple gunshot wounds of the trunk and leg. The manner of death is homicide.

Subject 92:
A 46 year old White male with a documented history of alcohol, marijuana and drug (unspecified) abuse. Autopsy findings are significant for bullous emphysema and fatty liver. The cause of death is acute intoxication due to the combined effects of opiate and ethanol. The manner of death is probable accident.

Subject 93:
A 31 year old White female with no known history of alcohol or drug abuse. The cause of death is not explained by autopsy and is thus listed as no anatomical or toxicologic cause of death. The manner of death is natural.
Subject 94:
A 39 year old White male with a documented history of drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of opiate and alcohol. The manner of death is accident.

Subject 95:
A 38 year old White male with no known history of drug or alcohol abuse. The cause of death is moderately to focally severe arteriosclerotic cardiovascular disease. The manner of death is natural.

Subject 96:
A 42 year old Black male with no known history of drug or alcohol abuse. Family and friends are adamant that decedent has no illicit drug history and there is no mention of such in the medical records. The decedent does, however, have an extensive medical history including many years of pain medication usage. The cause of death is arteriosclerotic and hypertensive cardiovascular disease with a contributing factor of acute intoxication due to the combined effects of amitriptyline and cocaine. The manner of death is accident.

Subject 97:
A 37 year old White Male with no known history of drug or alcohol abuse. The cause of death is brain contusions and basilar skull fracture due to blunt force injury to the head. The manner of death is accident (traffic)

Subject 98:
Rejected. The decedent was initially identified and evaluated as a female based on a feminine first name. The fact that the decedent is actually a male was not discovered until records were collected following data collection. The subject is therefore rejected.

Subject 99:
A 30 year old White male with an undocumented abuse history. The cause of death is transection of aorta, basilar skull fractures and pelvic and rib fractures due to blunt force injuries to the head and trunk. The manner of death is accident (traffic).

Subject 100:
A 30 year old White male with a 17 year history of drug abuse including alcohol, marijuana, LSD, cocaine, and intravenous drugs (unspecified). The cause of death is anoxic encephalopathy due to the acute combination of opiate and cocaine intoxication. The manner of death is probable accident.

Subject 101:
A 34 year old White male with a documented history of alcohol abuse. Autopsy findings are significant for fatty liver. The cause of death is acute intoxication due to the combined effects of alcohol and opiate. The manner of death is accident.
Subject 102:
A 42 year old White female with no known history of drug or alcohol abuse. Autopsy findings are significant for cardiomegaly. The cause of death is pulmonary thromboembolism due to deep vein thrombosis of the lower extremity. A contributing condition is morbid obesity. The manner of death is natural.

Subject 103:
A 42 year old White male with no known history of drug or alcohol abuse. The cause of death is thoracicvertebral fracture with probable spinal cord contusion and left renal artery laceration with hemoperitoneum due to blunt force injury of the trunk. The manner of death is accident (traffic).

Subject 104:
A 35 year old Native American male with a documented history of alcohol abuse. The cause of death is basal skull fracture with cerebral contusions due to blunt force injury of the head. The manner of death is undetermined.

Subject 105:
A 43 year old Black female with a documented history of alcohol abuse and drug (unspecified) use. The cause of death is idiopathic seizure disorder. The manner of death is natural.

Subject 106:
Rejected due to damage incurred during the defleshing process.

Subject 107:
A 40 year old White female with a documented history of alcohol and opiate abuse. The cause of death is acute intoxication due to the combined effects of alcohol and methadone. The manner of death is accident.

Subject 108:
A 23 year old White male with no known history of alcohol or drug abuse. The cause of death is contact handgun wound of the head. The manner of death is suicide.

Subject 109:
A 25 year old White male with a documented 8 year history of oral methadone abuse. The cause of death is acute intoxication due to the combined effects of methadone and doxepin. The manner of death is accident.

Subject 110:
A 40 year old White male with no known history of alcohol or drug abuse. The cause of death is asphyxia due to hanging by ligature about neck. The manner of death is suicide.
Subject 111:
A 47 year old White male with an undocumented abuse history. Autopsy findings are significant for fatty liver and cardiomegaly. The cause of death is arteriosclerotic cardiovascular disease. The manner of death is natural.

Subject 112:
A 51 year old White male with no known history of alcohol or drug abuse. The cause of death is atherosclerotic coronary artery disease. The manner of death is natural.

Subject 113:
A 45 year old White male with a documented history of alcohol, intravenous drug (unspecified) and drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of opiate and codeine. The manner of death is accident.

Subject 114:
A 40 year old White male with no known history of drug or alcohol abuse. The cause of death is cardiomegaly with left ventricular hypertrophy. The manner of death is natural.

Subject 115:
A 41 year old White male with a documented history of alcohol abuse and drug (unspecified) use. The autopsy report was not available at the time of data collection so thoracic disease is undocumented. The cause of death is lobar pneumonia with a contributing condition of chronic ethanolism. The manner of death is natural.

Subject 116:
A 77 year old White male with no known history of alcohol or drug abuse. Autopsy findings are significant for moderate coronary atherosclerosis. The cause of death is cerebral contusions and skull fracture due to blunt force injury of the head. The manner of death is accident (pedestrian struck by motor vehicle).

Subject 117:
A 42 year old Black female with an undocumented abuse history. Autopsy findings are significant for fatty liver. The cause of death is acute intoxication due to the combined effects of methadone, imipramine, diphenhydramine, diazepam and oxycodone. The manner of death is probable accident.

Subject 118:
A 50 year old White male with a documented history of opiate abuse. Autopsy findings are significant for severe coronary atherosclerosis and fatty liver. The cause of death is acute intoxication due to the combined effects of opiate and trazodone. The manner of death is probable accident.
Subject 119:
A 78 year old White male with no known history of alcohol or drug abuse. Autopsy findings are significant for atherosclerotic cardiovascular disease and centriacinar emphysema. The cause of death is contact perforating centerfire rifle wound of the head. The manner of death is suicide.

Subject 120:
A 36 year old White male with a documented history of intravenous drug (unspecified) abuse and alcohol use. The cause of death is arteriosclerotic cardiovascular disease. The manner of death is natural.

Subject 121:
A 49 year old Black male with a documented history of alcohol and intravenous drug (unspecified) abuse. Autopsy findings are significant for arteriosclerotic cardiovascular disease. The cause of death is acute intoxication due to the combined effects of opiate, cocaine, and ethanol. The manner of death is accident.

Subject 122:
A 44 year old White female with a documented history of alcohol and prescription drug abuse. The cause of death is hypertensive and atherosclerotic cardiovascular disease with a contributing condition of diabetes mellitus. The manner of death is natural.

Subject 123:
A 48 year old White male with a documented history of alcohol and prescription drug abuse. Autopsy findings are significant for coronary arteriosclerosis. The cause of death is acute meperidine intoxication. The manner of death is accident.

Subject 124:
A 25 year old White Male with no known history of alcohol or drug abuse. The cause of death is contact perforating handgun wound of the head. The manner of death is suicide.

Subject 125:
A 34 year old White male with no known history of alcohol or drug abuse. Medical history is significant for asthma since his teen years. Cause of death is idiopathic seizure disorder. The manner of death is natural.

Subject 126:
A 39 year old White female with no known history of alcohol or drug abuse. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.
Subject 127:
A 19 year old Asian male with no known history of alcohol or drug abuse. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.

Subject 128:
A 47 year old White female with a documented history of intravenous drug (unspecified) abuse which is substantiated by autopsy findings of healed track marks on both arms. The cause of death is acute opiate intoxication. The manner of death is probable accident.

Subject 129:
A 65 year old White male with a documented history of alcohol abuse. Autopsy findings are significant for atherosclerotic cardiovascular disease. The cause of death is gastrointestinal hemorrhage due to ruptured esophageal varices as a consequence of alcoholic cirrhosis.

Subject 130:
A 42 year old Black male with no known history of alcohol or drug abuse. The cause of death is hypertensive cardiovascular disease with a contributing condition of diabetes mellitus with chronic renal failure. The manner of death is natural.

Subject 131:
A 15 year old white female with no known history of alcohol or drug abuse. The decedent is stated to have experimented only with alcohol and drugs (unspecified). The cause of death is stab wound of the neck with left carotid artery transection. The manner of death is homicide.

Subject 132:
A 49 year old White male with a documented 20 year history of alcohol and drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of cocaine, opiate and amitriptyline. A contributing condition is atherosclerotic coronary artery disease. The manner of death is accident.

Subject 133:
A 45 year old White female with a documented history of alcohol abuse. The cause of death is diffuse pulmonary consolidation of adult respiratory distress syndrome (ARDS) due to acute hepatic necrosis as a consequence of acetaminophen intoxication. A contributing condition is chronic ethanolism with fatty liver. The manner of death is accident.

Subject 134:
A 23 year old White female with no known history of alcohol or drug abuse. The cause of death is multiple rib fractures, lacerations of spleen and kidney and lung contusions due to blunt force injury to the trunk. The manner of death is accident (traffic).
Subject 135:
A 31 year old White male with a documented history of alcohol and drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of opiate, cocaine and ethanol. The manner of death is probable accident.

Subject 136:
A 20 year old White male with no known history of drug or alcohol abuse. The cause of death is multiple rib fractures with aortic and multiple visceral lacerations due to blunt force injury of the trunk. The manner of death is accident (traffic).

Subject 137:
A 32 year old White female with a documented history of alcohol and intravenous drug (unspecified) abuse. The cause of death is acute intoxication due to the combined effects of opiate, cocaine and alcohol. The manner of death is accident.

Subject 138:
A 43 year old Black female with a documented history of opiate and cocaine abuse. The cause of death is subarachnoid hemorrhage, etiology undetermined. The manner of death is accident.

Subject 139:
A 41 year old Native American female with a documented history of alcohol and opiate abuse. Autopsy findings are significant for needle track scarring of the arms and cardiomegaly. The cause of death is acute intoxication due to the combined effects of cocaine, methadone, diphenhydramine, benztropine and fluoxetine. The manner of death is probable accident.

Subject 140:
A 38 year old Black male with no known history of alcohol or drug abuse. Autopsy findings are significant for cardiomegaly. The cause of death is hypertensive cardiovascular disease. The manner of death is natural.

Subject 141:
A 19 year old White female with no known history of alcohol or drug abuse. The cause of death is atlanto-occipital dissociation, fractures of the cervicothoracic vertebral column and multiple visceral lacerations due to blunt force injury of the head and trunk. The manner of death is suicide (jump from bridge).

Subject 142:
A 40 year old White male with a documented history of chronic opiate and cocaine abuse. Medical history is significant for one previous overdose. Autopsy findings are significant for moderate coronary atherosclerosis. Cause of death is acute intoxication due to the combined effects of opiate and cocaine. The manner of death is probable accident.
Subject 143:
A 39 year old White male with a documented history of methamphetamine abuse. The cause of death is Pneumocystis pneumonia due to Acquired Immune Deficiency Syndrome.

Subject 144:
A 43 year old White male with a documented history of alcohol abuse. Autopsy findings are significant for evidence of past intravenous drug use. Intravenous drug paraphernalia was present at the scene. The cause of death is cirrhosis due to chronic ethanolism. The manner of death is natural.

Subject 145:
A 48 year old White male with no known history of alcohol or drug abuse. The cause of death is arteriosclerotic cardiovascular disease. The manner of death is natural.

Subject 146:
A 29 year old White male with a documented history of alcohol and intravenous drug (unspecified) abuse. Autopsy findings are significant for dilated cardiomyopathy. The cause of death is acute intoxication due to the combined effects of alcohol, opiates and cocaine. The manner of death is accident.

Subject 147:
A 49 year old White female with a documented 25 year history of alcohol abuse. The cause of death is chronic ethanolism with fatty liver. The manner of death is natural.

Subject 148:
A 55 year old White female with no known history of alcohol or drug abuse. The cause of death is arteriosclerotic cardiovascular disease. The manner of death is natural.

Subject 149:
A 57 year old White female with no known history of alcohol or drug abuse. The cause of death is atherosclerotic cardiovascular disease with a contributing condition of cardiomegaly. The manner of death is natural.

Subject 150:
A 52 year old White male with no known history of alcohol or drug abuse. The cause of death is moderate to severe arteriosclerotic cardiovascular disease with coronary occlusion. The manner of death is natural.

Subject 151:
A 49 year old White male with no known history of alcohol or drug abuse. Autopsy findings are significant for fatty liver although next of kin (wife) stated no alcohol abuse history. The cause of death is pulmonary thromboembolism due to a deep vein thrombosis of the lower extremity. The manner of death is natural.
Subject 152:
A 48 year old White male with no known history of alcohol or drug abuse. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.

Subject 153:
A 24 year old White female with no known history of alcohol or drug abuse. The cause of death is not revealed by autopsy and thus stated as no anatomical or toxicologic cause of death. The manner of death is natural.

Subject 154:
A 43 year old White male with no known history of alcohol or drug abuse. Autopsy findings are significant for moderate coronary atherosclerosis. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.

Subject 155:
An 82 year old White male with no known history of alcohol or drug abuse. Autopsy findings are significant for arteriosclerotic cardiovascular disease and emphysema. The cause of death is subdural hematoma due to blunt force injury to the head. The manner of death is accident (fall).

Subject 156:
A 25 year old White female with no known history of alcohol or drug abuse. The decedent has a possible past use of drugs (type not specified) however use is known not to be chronic and thus not considered abuse. The cause of death is asphyxia due to hanging by ligature about the neck. The manner of death is suicide.

Subject 157:
A 39 year old White male with a documented history of drug (type unspecified) abuse. Autopsy findings are significant for atherosclerotic cardiovascular disease. The cause of death is acute intoxication due to the combined effects of cocaine, methamphetamine, diphenhydramine and methocarbamol. The manner of death is accident.

Subject 158:
A 33 year old White male with an undocumented abuse history. Autopsy findings are significant for cardiomegaly and fatty liver. The cause of death is arteriosclerotic and hypertensive cardiovascular disease. The manner of death is natural.

Subject 159:
A 44 year old White male with a documented history of alcohol, cocaine and heroin abuse. The cause of death is anoxic encephalopathy due to left ventricular cardiac hypertrophy as a consequence of probable hypertensive cardiovascular disease. A contributing condition is chronic intravenous drug abuse. The manner of death is natural.
Subject 160:
A 30 year old White female with a documented history of heroin abuse. The cause of death is laceration of the aorta and pelvic fractures due to blunt force injury of the trunk. The manner of death is accident (pedestrian struck by motor vehicle).

Subject 161:
A 40 year old White male with an undocumented history of abuse. The cause of death is acute opiate intoxication. The manner of death is probable accident.

Subject 162:
A 17 year old White male with no known history of alcohol or drug abuse. Medical history is significant for asthma. The cause of death is acute intoxication due to the combined effects of diphenhydramine and doxylamine. The manner of death is suicide.

Subject 163:
A 67 year old White male with a documented history of alcohol abuse. Autopsy findings are significant for atherosclerotic cardiovascular disease. The cause of death is penetrating near to contact handgun wound of the head. The manner of death is suicide.

Subject 164:
A 48 year old White male with no known history of alcohol or drug abuse. The cause of death is arteriosclerotic cardiovascular disease. The manner of death is natural.

Subject 165:
A 79 year old White male with no known history of alcohol or drug abuse. The autopsy report was not available at the time of data collection and thus thoracic disease is undocumented. The cause of death is metastatic carcinoma of the lung. The manner of death is natural.

Subject 166:
A 43 year old White male with a documented history of alcohol and drug (unspecified) abuse. The autopsy report was not available at the time of data collection and thus thoracic disease is undocumented. The cause of death is alcoholic liver disease with fatty metamorphosis. The manner of death is natural.

Subject 167:
A 28 year old White male with a documented history of alcohol and drug (unspecified) abuse. The cause of death is anoxic encephalopathy due to acute opiate intoxication. The manner of death is probable accident.

Subject 168:
A 37 year old Hispanic male with an undocumented abuse history. The cause of death is cerebral contusions and lacerations due to blunt force injury to the head. The manner of death is undetermined (unwitnessed fall).
Subject 169:
A 47 year old White Male with no known history of drug or alcohol abuse. The cause of death is positional asphyxia with a contributing condition of Multiple Sclerosis. The manner of death is accident.

Subject 170:
A 57 year old male of unspecified race and an undocumented history of abuse. The cause of death is diabetic ketoacidosis. The manner of death is natural.

Subject 171:
A 59 year old White female with no known history of alcohol or drug abuse. The cause of death is contact perforating gunshot wound of the head. The manner of death is suicide.

Subject 172:
A 32 year old White male with an undocumented abuse history. The cause of death is basal skull fractures with pontomedullary transection due to blunt force injury of the head. The manner of death is accident (traffic).

Subject 173:
A 43 year old White male with a documented history of chronic alcohol abuse. The cause of death is left bronchopneumonia. The manner of death is natural.
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