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The Department of Radiology, Brooke Army Medical Center and the Office of the Surgeon General sponored this course. The conference was held at the Henry B. Gonzales Convention Center from 24-28 April 1995. The meeting provided attendees with current information in radiology and related areas and offered up to 32 CME credits for all attendees. Lectures covered Diagnostic, MRI, Neuroradiology, Mammography, CT, and Special Procedures. A half day of lectures covered the MDIS system and current issues and lessons learned with implementation. Five residents competed in the resident competition. Over 250 military and civilian radiologists attended the conference.

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Raoul Hagen, M.D. — 16 Oct 95

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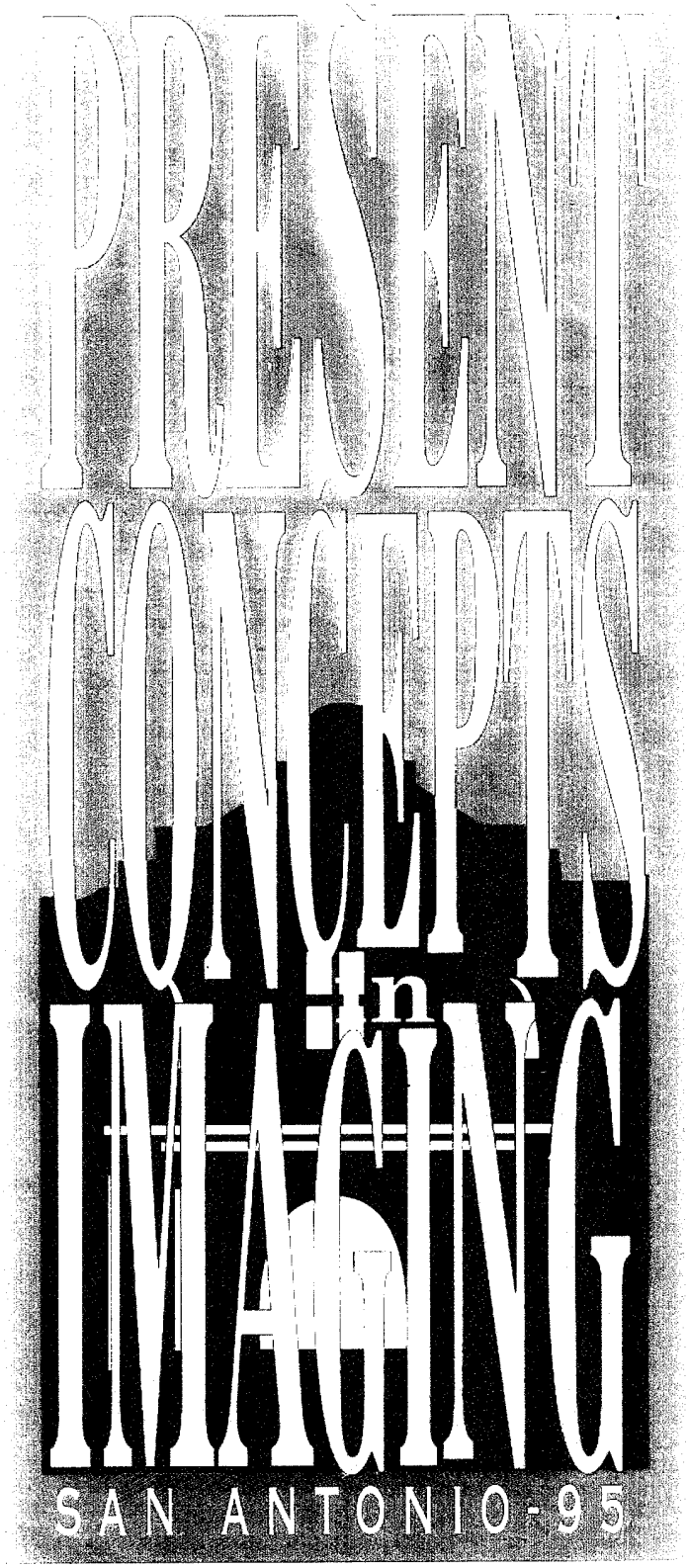

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AGENDA

MONDAY, 24 April 1995

Moderator: Anna K. Chacko, M.D.

0830 Introduction
BG Claypool, COL Chacko

0900 Roentgen Centennial
Peter Riesz

0930 Medical Effects of Nuclear Weapons
Asaf Durakovic

BREAK

1015 MRI of Muscle Trauma
Lynn Steinbach

1045 MRI of Elbow
Lynn Steinbach

1115 Acute Abdomen in Infants and Children, Part I
Fred Johnson

LUNCH

Moderator: Rodrigo Dominguez, M.D.

1315 Congenital Heart Disease - Basic Principles
Len Swischuk

1345 Acute Abdomen in Infants and Children, Part II
Fred Johnson

1415 Congenital Heart Disease - Current Imaging
Len Swischuk

BREAK

Moderator: Rashmikant B. Shah, M.D.

1500 Stereotactic Breast Biopsy
Steve Parker

1530 Screening Mameography Program
E. Glenn

1600 Calcifications on Mammography
Matthew Freedman

1630 Head and Neck Trauma
Angelo DelBalso

TUESDAY, 25 April 1995

Moderator: Nestor L. Nieves, M.D.

0830 Neuro Imaging - Spinal Cord - Neoplastic
John Sherman

0900 Filmless Radiology
Eliot Siegel

0930 Neuro Imaging - Spinal Cord - Non-neoplastic
John Sherman

BREAK

Moderator: Douglas C. Brown, M.D.

1015 Many Faces of Meningioma
Richard Hurwitz

1045 PET Scanning - Brain
Robert Kessler

1115 Potpourri of Neuro MRI
Edward Savolaine

LUNCH

Moderator: Janice C. Stracener, M.D.

1315 Renal Cystic Disease for the Non-Uroradiologist
David Hartman

1345 Prostate Cancer: To Screen or Not To Screen
Robert Finegold

1415 Retroperitoneal Diseases: A Practical Approach
David Hartman

BREAK

Moderator: Brian J. Burke

1500 Ovarian Screening
Alda Cossi

1530 Abdominal Ultrasound
Charles Fagan

1600 OB/GYN Ultrasound
Alda Cossi

1630 Ultrasound & MR Prostate Imaging
Robert Finegold

WEDNESDAY, 26 April 1995

Moderator: Michael A. Cawthon, D.O.

0830 Keynote Address
LTG(Ret) Quinn Becker, M.D.

0900 The Development and Anatomy of a PACS System
COL Fred Goeringer

0930 Three Years of Clinical Experience with PACS/MDIS at MAMC
MAJ Donald Smith

1000 Break

1015 Image Processing and the Future of Image Processing of Projection Radiography
Matthew Freedman

1045 Teleradiology in the Managed Care Environment
MAJ Robert Leckie

1115 Panel Discussion

1130 GI Cases
MAJ Carol Ebersole

1200 Lunch

1315 Resident Presentations
Moderator: Anna K. Chacko, M.D.

High Resolution CT
CPT Tim Cramer

Colonic Perforation During Air Enema in the Porcine Model: Relative Risk of Perforation in the Neonate
CPT Stephen O'Conner

Glenohumeral Labral-Ligamentous Complex: Evaluation Using MR Arthrography
CPT Yong T. Bradley

Cost Effectiveness of Screening Knee MRI: A Prospective Evaluation of 50 Consecutive Patients
CPT Liem Mansfield

Chronic Ankle Instability: Evaluation with Magnetic Resonance Arthrography, Magnetic Resonance Imaging, and Stress Radiography
CPT Michael T. Harper

1445 Break

THURSDAY, 27 April 1995

Moderator: Dennis J. Lee, M.D.

0830 Lower Extremity Trauma
Michael Mulligan

0900 Ischemic Disease of the CNS
Bruce Dean

0930 Bone Potpourri
Maurice Reeder

BREAK

Moderator: Ninh V. Dao, M.D.

1015 MRI of Sports Injuries
Bruce Dean

1045 MRI of Spine, Hip, Knee in Acutely Traumatized
Michael Mulligan

1115 Radiology of Tropical Medicine
Maurice Reeder

LUNCH

Moderator: Carol J. Ebersole, M.D.

1315 Thoracic Lymphomas
Herman Libshitz

1345 CT of Aortic Aneurysms
Michael Federle

1415 Staging Lung Cancer
Virendra Saxena and Patricia Lillis-Hearne

BREAK

Moderator: Frederick Barnett, M.D.

1500 HRCT of the Chest
Chris Meyers

1530 Chest "How in the world did I ever miss that?"
Thomas Harle

1600 Typical & Atypical Facets of TB
Albert Hale

FRIDAY, 28 April 1995

Moderator: Gary Simmons, M.D.

0800 3D Imaging & Reconstruction in the
Evaluaton of Traumа (Head & Neck)
Len Nadalo

0845 Sports Injuries
Lee Rogers

0930 Differential Diagnosis of Posterior Fossa Masses
Blake Johnson

BREAK

1030 Poly-trauma Imaging
Lee Rogers

1115 Differential Diagnosis of Supratentorial Masses
Blake Johnson

LUNCH

Moderator: Steven J. Wegert, M.D.

1300 Plain Film, CT Evaluation of Chest Trauma
Phil Goodman

1345 CT of Abdominal Trauma
Michael Federle

1430 Revascularization Techniques in Peripheral
Vascular Pathology
Anthony Venbrux

BREAK

1530 Angiographic Investigation of Penetrating
Vascular Injury in the Non-renal Extraperitoneum
Yoram Ben-Menachem

1615 Interventional Techniques in GU Trauma
Anthony Venbrux

1700 Angiographic Evaluaton of Chest Trauma
Yorum Ben-Menachem

The Life of Wilhelm Conrad Roentgen
on the Centennial of his Discovery of X-rays on November 8, 1895

This year, 1995, marks the centennial of the discovery of x-ray by Wilhelm Conrad Roentgen on the historical date of that discovery, November 8, 1895. After completing my Radiology residency here at Brooke General Hospital, I was assigned to the U.S. Army 5th General Hospital in Stuttgart-Bad Cannstatt, West Germany. This period of indentured servitude, known as "pay back time," was from October 1966 to August 1969. During these memorable years, the opportunity presented itself for me to make a pilgrimage to every location associated with the life of Roentgen. It will be my pleasure to share that pilgrimage with you.

This is the subject of my lecture -- Roentgen. Actually, he belongs to my partner and son-in-law. This is a bust of the real subject, Roentgen.

He was born on March 27, 1845 in Lennep (now called Remscheid-Lennep) just south of the industrial Ruhr Valley between Düsseldorf and Cologne in what was then the State of Prussia before the unification of Germany. He was born in this comfortable Bergenland style home, still standing, at #287 Poststrasse. His father, Friedrich Conrad was a respected local textile merchant. His mother, Constance Charlotte nee Frowein, was of Dutch heritage. Roentgen was an only child. This plaque was placed on the Roentgen home on March 27, 1920. It reads, "In this house the discoverer of the rays which were named for him Wilhelm Conrad Roentgen was born on March 27, 1845. His home town declared him an honorary citizen in 1896." This monument to Roentgen by Arno Breker was dedicated in his home town on November 30, 1930. This wonderful Deutsches Roentgen Museum was opened on

June 18, 1932. It houses many fine displays and memorabilia of Roentgen and has a fine history of x-ray with many pieces of early original equipment. Roentgen's birth house was acquired by the museum in 1965. It also houses the furniture from his private office in Wurzburg.

On May 23, 1848 when Roentgen was three, his family moved to this town of Apeldoorn, Holland where his mother's family had settled. This move was probably made because of the severe political unrest in Prussia at that time. As a result of this move, they lost their Prussian citizenship and became Dutch citizens. They lived in this home on Dorpsstraat now listed as #171 Hoofdstraat. Later, he returned to this home to be married. It has now been converted to a public health clinic. For fourteen years he attended the primary and secondary school of one Martinus Hermann van Doorn at this site at #8 Regentesselman. This public library now occupies that site. This street was named in his honor.

He left Apeldoorn to obtain more advanced schooling with the encouragement of his parents. On December 27, 1862 he arrived in Utrecht, Holland and enrolled in the Utrecht Technical School. That school was located at this site on Ganzenmarkt. It is now a private health club. He lived on this street, Nieuwegracht, with the Gunning family. Dr. Gunning was a well-known chemist of the time and must have recognized some potential in Roentgen as he encouraged him to pursue further studies. However, an unfortunate chain of events almost permanently ended his academic endeavors. A fellow student drew a caricature of their teacher on the

blackboard and somehow the finger of suspicion was pointed at Willy. He refused to reveal the identity of the true artist and was himself expelled by the authorities. He then decided to take the oral entrance exam to enter the University of Utrecht. Unfortunately, his examiner was none other than the subject of that caricature. Naturally, Roentgen failed the exam. On January 18, 1865, he did register at the University in order to audit some courses. During this period he heard of a new school in Zurich, Switzerland which did not require a technical school diploma, which Roentgen lacked, for admission. After discussion with his parents, they encouraged him to apply for admission. This resulted in his acceptance in the Mechanical Technical Division of the Zurich Polytechnical School.

He left Utrecht for Zurich on November 16, 1865 and was duly enrolled at the Eidgenössischen Technische Hochschule von Zurich (Federal Polytechnical College of Zurich). He lived in this house at #7 Seilergraben where a plaque was mounted in 1922 which reads, "Wilhelm Conrad Roentgen, the discoverer of the rays named after him, lived here 1866 to 1869 when a student at the Federal Technical High School."

He thrived on the technical courses and even took extra hours at the adjacent University of Zurich. He loved the surrounding mountains and went on frequent pleasure hikes. He also enjoyed eating at a local inn, Zum Grünen Glas, run by the innkeeper, Ludwig. One of the waitresses caught his eye. It turned out to be the innkeeper's daughter, Anna Bertha Ludwig, who would later become his wife. She was six years his elder. His grades improved as he progressed through his schooling

and in his last year he received the highest possible marks. On August 6, 1868 he received the degree of Mechanical Engineer and on that happy day he asked for Bertha's hand in marriage. After graduation he stayed on at the University of Zurich. These are the buildings of the Technical High School and University of Zurich. He took courses with the great experimenter and theoretical physicist, Dr. August Kundt. It was Kundt who developed Roentgen's potential as a theoretical scientist and it was under Kundt that he wrote a dissertation "Studies on Gases" for which he received his Doctorate in Philosophy from the university on June 22, 1869. Professor Kundt then invited him to stay on as an assistant in physics. He became fascinated with work on gases and further developed his experimental talents and showed his excellence as an astute scientific observer.

In the spring of 1870, Professor Kundt was called to the chair of physics at the Julius-Maximilians University of Wurzburg, Germany and invited Roentgen to go along with him. By this time Kundt thought so much of Roentgen that he accepted the Wurzburg position only if they would also invite Roentgen which they readily approved. Thus in the fall of 1870, Roentgen moved to the picturesque city of Wurzburg on the Main River where he found comfortable quarters with a Frau Troll who ran the Restaurant Eckertsgarten on Veitshöchheimer Strasse. Meanwhile, his fiance Bertha had gone to Apeldoorn to learn from Wilhelm's mother the German way of being a good wife. After a brief return to Zurich to care for her dying father, she returned to Apeldoorn in the spring of 1871 for further cooking and

housekeeping lessons. Bertha and Wilhelm were married on January 19, 1872 in an elegant ceremony in the Roentgen home in Apeldoorn. Returning to Wurzburg, they settled in a modest home on Heidingsfelder Strasse. Wilhelm returned to experimentation and teaching at the Physics Institute of the Julius-Maximilians University on Neubaustrasse. But the institute was poorly equipped and funded and a disappointment to both Kundt and Roentgen. Therefore, it was good news when both scientists were offered higher positions at the newly reactivated Kaiser-Wilhelms University of Strasbourg.

On April 1, 1872, both men made a move to this beautiful city in Alsace-Lorraine which was once again under German rule. Roentgen continued his scientific investigations in order to gather data for his habilitation, exam, for the academic level of privat dozent or unpaid lecturer. In those years, this was one step on the path that a PhD had to travel to obtain a position as a salaried professor. During this period, Roentgen's mother and father moved to Strasbourg to be near their children, arriving October 3, 1873. The day of the great test arrived on March 13, 1874. After thorough questioning by a panel of distinguished scientists, Roentgen passed with flying colors and was awarded the position of privat dozent. This honor allowed him the privilege of teaching at any university in Germany. It carried no salary but he could now receive fees from the students in his classes. It also qualified him for appointment as assistant professor, a salaried position, which was the next step on the academic ladder. Some waited years for their first call. Roentgen's opportunity

came only one year later.

On April 1, 1875 Roentgen was called by the Agricultural Academy of Stuttgart-Hohenheim in Württemberg and offered a full professorship in physics and mathematics. This position he accepted with reservations as it meant breaking ties with his family and friends and Professor Kundt. However, this new challenge did offer him a salary and also civil service status. The latter also automatically made him a German citizen so Germany could now truly claim him as a native son. The position was less than satisfactory with a small one room department and primitive personal living conditions. Thus, in about one and one-half years, when Professor Kundt created a new chair for theoretical physics and recommended they call Roentgen for the position, he readily accepted.

On October 1, 1876, the Roentgen's gladly returned to Strasbourg. During this period, he worked on perfecting his techniques in physical experimentation and gained teaching experience. He did important work on the specific heat of gases, plane of polarization in gases, conduction of heat in crystals, and the piezoelelectric property of crystals named the Roentgen effect. He published about fifteen important papers during this period and was recognized as a rising star in his profession. As a result, he was recognized for the full professorship then open in Giessen and recommended by the famous German physicists von Helmholtz, Kirchhoff, and Kundt.

On April 1, 1879, the Roentgen's left for that position at the prestigious old

Hessian Ludwigs University recently named the Justus von Liebig University of Giessen. His initial laboratories were in cramped quarters in a private home. He was asked to design a new department and this was built and occupied in the winter of 1880-1881. He continued his special investigations, mostly with the different properties of crystals. His parents followed them to Giessen. During this period they both died, his mother in 1880 and his father in 1884. They were both buried in the Alten Friedhof in Giessen where Wilhelm and his wife were also later buried in a common area. Having no children of their own, they decided to take Bertha's six-year-old niece, Josephine Bertha Donges, into their home in 1887. She was legally adopted by them when she turned twenty-one. His scientific experimentation produced eighteen papers while at Giessen. This monument and inscription in a city park in Giessen illustrates the penetrating power of x-rays. As a result of his recognition as an extraordinary scientist, he was offered the position of professor of physics and director of the new Physical Institute at the University of Wurzburg.

So it was back to Wurzburg on October 1, 1888. This new institute was located on the tree-lined Pleicher Ring, later renamed Roentgen Ring. It was a two-story building with ample space for labs and lecture rooms. The director and his family lived in a spacious apartment on the second floor, directly above the lab where he would make the observations leading to the discovery of x-rays. This is that building. During his Wurzburg experience, he produced seventeen important papers -- the most important of his entire life. In honor of his excellence, he was

elected Rector or President of the Julius-Maximilians University of Wurzburg for the biennium of 1894 and 1895. I wonder if they had a premonition of what was to come.

This brings us to the momentous period at the end of 1895. A great many scientists were experimenting with that they thought were the properties of cathode rays and getting very conflicting results. We know that sometime during October, Roentgen became completely captivated by the investigations of Hittorf, Crookes, Hertz, and Lenard on these strange rays. The intensity of his attention to his own experiments with this area only increased as he progressed with the work, completely occupying his days and frequently captivating him until late into the night. In his laboratory, he was busy accumulating the best equipment available for his cathode ray experiments. This consisted of a large Ruhmkorff induction coil manufactured by Reiniger-Gebbert and Schall in Erlangen. This was attached to a Deprez interrupter which resulted in a high energy discharge. He also acquired several Hittorf-Crookes and some Lenard tubes of different strengths. The Lenard type was a round glass cathode ray tube which had a small window covered by a thin aluminum foil through which the cathode rays could penetrate. The oval Hittorf-Crookes tube had no such window, only a glass target area. There was also a Raps vacuum pump which was essential to evacuate these tubes prior to use for more efficiency. When this equipment was all assembled and in proper working order, he began his observations in earnest.

This brings us to that historic day of Friday, November 8, 1895. Roentgen had been doing experiments confirming earlier works of Lenard. He was using a low output Lenard tube wrapped in cardboard and tinfoil, so that no visible light emanated, and showing the fluorescence of a small cardboard screen coated with barium platinocyanide when it was placed close to the tube and bombarded by cathode rays. He then thought of another approach and wondered if he could observe the same effects from the all glass Hittorf-Crookes tubes of higher strength. He selected a tube, encased it in cardboard, connected it to his Ruhmkorff coil, darkened the room, and activated the coil so as to pass current through the tube. He easily confirmed that there was no visible light leak. He noticed the expected fluorescence of the screen near the tube. He was prepared to turn off the current to the tube to prepare for the next phase of his experiment. But suddenly, from a workbench he knew was about a yard from the tube, the corner of his eye caught the faint flicker of a weak light. This light attracted his attention enough that he continued to energize the tube. He was rewarded by a continued fluorescence of a faint green cloud of flickering light waves moving in unison with the fluctuating discharges of the coil. Highly excited, he lit a match and discovered the source of this dim light was a small barium platinocyanide screen lying on the bench. He continued to apply current to the tube, moving the fluorescent screen further and further away while still fascinated with its continued fluorescence. Cathode rays, he knew, never traveled these distances and he became completely absorbed in

explaining these observations. He hardly noticed the passage of time and could barely be torn away to eat supper in excited silence with his wife before returning to the lab. He started to question his findings as they did not follow the known properties of cathode rays. He held different papers and books in the beam with very little dimming effect on the fluorescence. Metallic objects were seen to be outlined on the screen; and while holding one object, he noticed the shadow of the bones of his fingers! Impossible!! It is perhaps at that time that he made the proper conclusion and thus the discovery; these effects were not due to cathode rays but to a new type of unknown highly penetrating rays he called the x-ray. He continued to devote full time to analyzing the properties of this new kind of ray. He sometimes slept on a couch in the lab over this busy weekend. He examined all kinds of objects: a set of weights, the barrel of his shotgun, a compass, a coil of wire, different types of wood and paper, glass, and different chemical compounds over the next several weeks. The only time he shared his activities occurred when he excitedly invited his wife to visit the laboratory and he performed an exposure of her hand (the fuzzy picture with one ring). She was amazed and this was the first recorded human x-ray, dated December 27, 1895. His major conclusions and observations were finished by the end of the month of December. On December 28, 1895 he delivered his historic paper, "On a New Kind of Rays," to the Wurzburg Physical Medical Society. It outlined seventeen points which he listed as the essential properties of the new rays. It was printed immediately by the society but it was not

due to be read to the society until the next meeting in late January. Rather than risk misinformation to the public, on January 1, 1896 he mailed reprints of that paper to a number of his scientific colleagues and friends. He commented to his beloved Bertha, "Donnerwetter, now all hell will break loose."

And he was correct. The importance of the discovery was instantly recognized by fellow scientists and the news of Roentgen's discovery was instantly published to the public. He immediately became the focus of international praise, admiration, and curiosity. Letters of congratulation flooded in form all over the world. His work was easily duplicated and verified and its potential, particularly for medicine, was immediately recognized. He was called to Berlin to demonstrate his findings to Emperor Wilhelm II which he did in the presence of the Imperial Court on January 13, 1896. He then returned to Wurzburg to speak publicly to his colleagues which he did on January 23, 1896. He then ended by demonstrating his discovery by taking an x-ray of the hand of Dr. von Kolliker, a distinguished anatomist. This street in Wurzburg is named after von Kolliker. The demonstration was highly successful (sharp picture with two rings) and von Kolliker lead an ovation of three cheers for the discoverer and proposed the rays henceforth be known as Roentgen's Rays. On March 9, 1896 and March 10, 1897 he delivered continued communications "On a New Kind of Rays" to the Wurzburg Physical Medical Society. Mounds of medals and awards followed. This was topped by Roentgen being named the world's first Nobel Laureate in Physics. He accepted the prize in person in Stockholm on

December 10, 1901. This is his actual Nobel Prize certificate which is on display in a small case outside the discovery laboratory. This display case also contains other memorabilia including the gun he examined, the box of weights he x-rayed, some tubes he used, some medals he received, and a letter of congratulations from his scientific colleagues. Notice the signatures of Max Planck and Albert Einstein. In the face of all the adulation, he remained humble and never tried to capitalize his findings or patent them. He moved on to other areas of interest and never did much more with the x-ray. This sign was placed on the discovery building in 1937. It reads, "In this building in the year 1895, Wilhelm Conrad Roentgen discovered the rays that have been named for him." These are my Army X-Ray Technologists outside that building while on a field trip. This is the actual room of the discovery. In 1968, it contained an experimental betatron. Here my daughter stands outside the nursing home where, in 1969, the Roentgen's adopted daughter still resided. We were unable to obtain an interview.

Meanwhile, the Bavarian government had asked Roentgen to take the chair of physics and director of the Physical Science Institute at the Ludwig-Maximilians University of Munich. This was a big promotion and he accepted, moving to Munich on March 23, 1900. Most of his time was devoted to supervising the physics department which he headed and with teaching some classes as he was now a very popular lecturer. He devoted much time to the care of his beloved wife Bertha who had developed a serious kidney disease. Her health steadily deteriorated and she

died on October 31, 1919 in her 80th year. She was buried in the Roentgen plot in Giessen.

Following her passing, Roentgen decided to retire from the University of Munich which he did in the spring of 1920. The post World War I period in Munich was a difficult time, but Roentgen kept himself busy reading and corresponding with old friends. He developed colon trouble which progressed and in early February 1923 was diagnosed with colon cancer. He passed away quietly on February 10, 1923 and was cremated three days later. After his death, he willed some of the equipment he used in his discovery to the Deutsches Museum. These are my Army X-Ray Technologists on a field trip to that institution. This case contains his original Ruhmkorff coil and several Hittorf-Crooke tubes he used. They were willed to the museum at his death. On November 10, 1923 his ashes were buried at this family plot in Giessen along with his wife and parents.

GLOBAL CONCERNS OF THE CURRENT NUCLEAR REALITY

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ABSTRACT

The apparent end of the Cold War did not bring the production and improvement of The Nuclear Arsenal to an end. Instead there is an objective evidence of the development of exceedingly more sophisticated multiple thermonuclear warheads with delivery systems of intercontinental range and shorter speed of response with improved accuracy in both strategic and tactical scenarios of the potential nuclear confrontation. The much advertised reduction of the absolute number of nuclear warheads generally applies to the obsolete and outdated arsenal, whereas the new possibilities of a nuclear confrontation on the tactical scale have been increased by an uncontrolled transfer of weapon grade nuclear materials to an increasing number of the countries either capable of producing or already in the possession of nuclear weapons.

This paper addresses the potential physical , environmental, social, economic, psychological and medical consequences of the current nuclear reality. It also discusses the projected global consequences of the accidents in Nuclear Industry, still unresolved problems of the high level nuclear waste, physical security of military and civilian nuclear facilities and materials, and International concerns of containing the threat of the global dimensions on Nuclear Terrorism.

INTRODUCTION

The strategic nuclear battlefield is a concept related to the war scenario from far behind the conventional confrontational lines of battle by the use of long range delivery systems of nuclear weapons to attack deep into enemy territory. It is distinctly different from tactical nuclear warfare wherein short range delivery systems may be used to deploy the nuclear weapons with the situations of a more conventional battlefield.

This paper addresses several key questions regarding current nuclear reality where many uncertanties prevail after an apparent end of the Cold War. An essential question is the future of the process of democratic changes in the former Soviet Union and the future development of dealing with the Nuclear Arsenal in The Russian Republic and the independent countries that have seceded from The Soviet Union still containing a considerable amounts of nuclear weapons and weapon grade materials, such as Belarus, Ukraine and Kazakhstan. The smuggling activities of Uranium and Plutonium nuclear fuel into the countries with potential regional conflicts and still unresolved issues of the political future of the previous Soviet Bloc necessitates a concern of a question of what kind of Nuclear Weapons will be involved, what are the military strategies, is the civil defense possible, what would be the consequences of a nuclear confrontation on tactical or less likely strategic scale and ultimatelly, what would be its ecological, Social, economical, psychological and medical consequences.

An important aspect of this paper also deals with the issue of the emergency preparedness in the event of the major accidents involving Nuclear Industry, Transportation of Nuclear Materials, Disposal of a High Level radioactive waste and Nuclear Terrorism.

NUCLEAR ARSENAL

Nuclear weapons have evolved from the prototype fission bombs deployed in Japan at the conclusion of The World War II to the awsome arsenal of Thermonuclear warheads with a constantly updated and increasingly sophisticated delivery systems. The combined arsenal of The United States and previous Soviet Union include more than 50.000 nuclear warheads with a total combined yield of approximatelly of 15.000 megatons (explosive power of one million tons of TNT) or the equivalent of one million of Hiroshima bombs (1), The explosive power of today's nuclear weapons range approximatelly between 100 tons to 50 megatons of TNT,as compared with still unresolved yield of 12-15 kilotons of Hiroshima bomb.

The majority of strategic nuclear weapons are deployed by the use of ballistic missiles which are launched into space and can orbit intercontinental distances within the time of minutes. Land based intercontinental ballistic missiles (ICBM) are deployed in underground silos encased in various types of reinforced concrete

The land-based intercontinental missiles are often fitted with multiple warheads independently targetable on sites that are hundreds of miles apart.They comprise the group of Multiple Independently targetable Reentry vehicles (MIRV). The largest partof the nuclear arsenal is deployed in submarine launched ballistic missiles (SLBM). The Trident submarines carry 24 SLMB with a maximum range of close to 5000 miles when equiped with eight 100 kiloton MIRV warheads (2). The advantage of the ICBM systems is mainly contained in their capacity of being highly mobile and non-targetable. The disadvantage of ICBM systems is that they are targetable and as the accuracy of delivery systems improve, the containment silos become more vulnerable to direct hits by the enemy warheads (1).

Strategic war can also be waged by aircraft. Bombers are frequently equipped with a short range Attack Missile Systems (SRAM) with a range of hundreds of miles long range cruise missiles which can be navigated thousands of miles into the enemy territory. Cruise missiles can also be lauched from submarines and battleships (2).

The most recent advent of the nuclear military concerns includes highly sophisticated bombers designed to evade radar detection (e.g. Stealth) and still experimental use of the laser equipped satellites in outer space designed to target and destry missiles in flight (Strategic Defense Initiative) (3).

MILITARY STRATEGY IN CURRENT NUCLEAR TIMES

Although President Harry Truman ordered the first use of the atomic bomb, he justified it as considering it a "Terror Weapon of the last resort" and sought to place the use of this new military technology under international control (4). His immediate successor,President Eisenhower fostered increased and varied research on application of nuclear weapons, making it an integral component of The United States Foreign Policy. His secretary of State John Dulles, developed a program of readiness for massive retaliation, intentended as a deterrence against a Soviet attack and the means of strengthening NATO alliance (4). During the Eisehower's administration the Single Integrated Operational Plan (SIOP) was developed for preparing the world nuclear terget list. This plan identified 1042 possible targets in the Soviet Union and China with over 3500 nuclear weapons ready to be deployed (4) During the Kennedy era, The Secretary of State Robert Mc Namara, developed Nuclear policy to correct the mistakes and nuclear excesses of Eisenhower administration (4). This effort limited the size of resources available to the military and represented a move away from preemptive all-out approach to nuclear strategy (4). McNamara and his staff determined that an effective deterren ce would require 200-400 nuclear warheads with a capacity to destroy about 50% of Soviet Industrial capacity and approximatelly 25% of its population (5),

Mutually assured destruction (MAD) has evolved as a declared United States Policy being a focal point of the strategic relationship between superpoers during the cold war era (6).Military strategists on the other hand have developed the concept of Nuclear Utilization Target Selection (NUTS) which includes plans for using nuclear weapons against specific selected tyargets. Current United States and Russian target selection although recently modified by non-targeting each other, has been derived from a counter-value policy that identifies objects of a national value such as military installations, industrial areas and population centers. The premise for this policy is that a military destruction of the enemy's command and control facilities limits a capability for a retaliatory strike (6). The concept of Mutually assured detsruction has acted as a powerful deterrent to the armed conflict among the superpowers. However, in order for the deterrent to be credible the military must be fully prepared to wage a nuclear war with the aim of destroying the retaliatory capacity of the enemy. Although strategists assume that nuclear war can be waged on a limited scale, there is a high risk of rapid escalation with both sides fearing the other will be first to to deliver a crippling preemptive strike. Unfortunatelly, however, given the diversity, decentralization and mobility of nuclear arsenals, an attack of this nature will almost certainly lead to a massive retaliation with devastating consequences (2).

CIVIL DEFENSE POLICIES

The civil; defense policies propagated in the United States during 1950's and 1960's have gone into a deep decline. This is largely due to the greater technical sophistication, reach and mag-nitude of the current nuclear arsenal, increased awareness of the limited viability of protective measures and a sense of resignation in the face of a total destruction likely to be wrought by a nuclear war (7). Although the Federal Emergency Management Agency (FEMA) has been advocating increased spending for civil defense, planning for protective measures in case of Nuclear War has received rather little support. This is likely due to an overwhelming feeling among the officials that no meaningful social survival will be possible after a nuclear attack.Some have agued that a strong civil defense preparedness policy would invite a nuclear attack because the enemy may assume that the other side is preparing for an offensive and would therefore be more likely to launch a pre-emptive strike. Others contend that sturdy civil defense would only serve as a future deterrent (7).

Many analysts have concluded that the evacuation planning is unrealistic giving the massive traffic congestion which would be an inevitable result. FEMA considers that three days would be the minimum time required for evacuation of Boston and Philadelphia and four days for New York City. However, given that in the event

However, given that in the event of nuclear war there is no safe place for this population to go, the level of cooperation with such plans is likely to be minimal. Furthermore, mass evacuation would be immediatelly apparent to the enemy through the satellite surveillance and intelligence network and would only serve to escalate tensions which might in turn increase the risk of the attack (7).

PHYSICAL IMPACT OF NUCLEAR WEAPONS DEPLOYMENT

An explosion of a one megaton nuclear weapon releases energy equal to one million tons of TNT. This energy release creates the blast, thermal radiation, ionizing radiation and radioactive falout. When a one megaton weapon is detonated within 2000 ft above the surface of the earth, photons are emitted within the time of one millionth of a second. Most of these x-rays are absorbed into the air around them producing an incadescent fireball,

The fireball reached temperature of 100 million degrees centigrade, five times higher that the center of the sun (8).The blaswaves produce a pressure of millions of pounds per a square inch (PSI). Contained within the fireball is the vaporized casing of the bomb, fission particles and other materials such as a dirt and debris which are taken by a negative pressure into it from the surface of the earth. As the surface temperature of the fire ball drops to about 300.000 degrees centigrade, condensation of the vaporized materials ensues which become highly radiactive and result in a radioactive fallout. This mass of energy continits expansion until it reaches approximatelly one mile in diameter and stretches far into the athmosphere (8).

Approximatelly three percent of the energy is released as a prompt nuclear radiation, consisting of X-rays, gamma rays, and neutrons which are lethal to approximatelly 1.7 miles of ground zero (7). Forty to fifty percent of the energy released by a one megaton bomb is in the form of light and heat. This thermal pulse travels at the speed of light and ignites fires as far as ten miles away. Buildings, people, plants and animals will absorb most of the heat within a second of detonation (8).

The blast wave of a nuclear explosion causes the most devastation. It is caused by highly compressed air which creates an overexposure (i.e. pressure above the athmospheric levels) and is followe by winds reaching 150-200 mph. As this blasr wave moves from ground zero it crushes and sweeps everything in its path. When a blast wave reaches about ten miles from ground zero its forces diminish to about one PSI. At five PSI exposure an ordinary framehouse will collapse.With a one megaton explosion this would occur out to 2.8 miles from the epicenter (8).

The radioactive material carried into the atmosphere by the dirt debris taken up by the negative pressure in the fireball is heavier than the fission materials of the bomb and begins falling back to the earth as black rain. The lighter materials are carried higher into the atmosphere and fall down farther downwind of the explosion. Radioactive fallout containing several hundreds of different fission products may integrate into the biosphere causing degenerative, genetic and neoplastic disease entities long after detonation of the nuclear weapon (8).

MEDICAL CONSEQUENCES

Primary blast effects include injury and death from traumatic events as structures collapse causing internal and external traumatic alterations as a consequence of the sudden pressure changes High winds turn building materials, brick, wood, steel, wood and glass into airborne projectiles. Some authors suggest that such objects may have an impact velocity at a range of almost fifteen miles to impact velocity sufficient to have a 50 percent probability of causing a fracture to the human skull (9).

Thermal effects include flash and flame burns. As the probability of massive fires is great, such effects may lead to the various injuries including asphyxia and lung damage from carbon dioxide and toxic fumes, as chemicals, fuels and contents of the buildings continue to burn. Looking at the fireball would produce blidness in some cases and to the mass casualties of heat exhaustion.

Radiation injuries would vary according to the size of the bomb and the victim's proximity. Estimates are difficult bacause of the differences between the ground and surface bursts,the variation in wind patterns, type of radiation received and the age and general health of victims. However, it is probabl;e that at least 30 percent of the population of the Northern Hemisphere would receive doses of at least 2,5 Gy with the entire population of the Hemisphere being exposed to 1 Gy.

Whether the source is the initial burst, fallout exposure or the effects of the low level radiation during the post attack period, the degree of illness depends on whether the exposure is local or a total-body, as well as whether radiation deposited is from external penetrating radiation or internal contamination. The later is determined by the pathways of entry into the internal environment of the organism, by inhalation, ingestion or via the traumatic lesions (10).

Lethal doses are considered to be in the 3-4.5 Gy ranges. Such patients die within the weeks from one of the radiation - induced syndromes. Bone marrow (hematopoietic) syndrome (2-10 Gy) produce loss of coagulation factors leading to various forms of the manifestation of hemorrhagic diathesis with an impairment and loss of specific and non-specific immunological mechanisms due to depletion of the lymphocytes and polymorphonuclear leukocytes.

Gastrointestinal Syndrome (800-3000 cGy) involves a damage to the intestinal mucosa causing severe fluid and ion loss as well as septicemia by the entry of the intestinal microflora into the intravascular compartment, leading to lethal outcome within days to weeks. Neurovascular syndrome (over 5000 cGy) leads to death within hours, dominated by the clinical picture of hypotension, hyperthermia, projectile emesis, severe diarrhea, ataxia, disorientation, circulatory problems, edema, elevated intracranial pressure, cerebral anoxia, oliguria and coma. Additional effects of ionizing radiation include damahge to the skin, lungs, gonads and eyes. In the general scenario of mass exposures to at least 100 cGy there are also delayed effects such as cataracts, vascular damage, genetic changes, and most importantly malignant alterations. One of out of 80 persons would likely develop fatal cancer while two in 80 persons would develop non fatal previously described consequences of ionizing radiation (10).

Considering various injuries as independent entities in isolation from one another may be misleading because of the sychrony of the homeostatic mechanisms and the effects of the combined injuries, having a synergistic effects of the injuries that would lead to the fatal outcome in the case that would not be fatal by the radiation exposure alone, thus reducing LD/50 to the lower values than 300-450 cGy (11).

In the event of a nuclear war medical services would suffer almost total paralysis. The vast majority of the hospitals and physicians tend to be concentrated in large urbane centers, which would constitute targer areas where destruction and fatalities would be highest. Even in the least adverse conditions where only a single major city had been bombed, there would not be enough medical resources in The United States to provide the adequate care for survivors (12). It must also be noted that even in the improbable event that medical services remained intact, professionals would be severely hampered by their total inexperience in handling radiation casualties.

ENVIRONMENTAL REALITY

Regardless of scenario and inherent uncertanties of predictions there is a concesuswithin the international scientific community that even a relatively small scale nuclear war would lead to global climatic and environmental consequences of catastrophic proportions (13, 14).

Multiple nuclear explosions would result in millions of tons of fine dust (a megaton blast carries approximately 200.000 tons of dust) being injected into the upper atmosphere. In addition an estimated 50 to 150 millions tons of smoke would be generated from the fires caused by the explosion (15).If these particles were spread over half of the Northern Hemisphere, only 50 percent of the solar energy and light would pass through to the earth's surface for a period of weeks (16).

This phenomenology has been extensively studied as a scenario that might lead to Nuclear Winter, which would cause the temperatures in the Northern Hemisphere to drop between 5 and 22 degrees centigrade within a few days, causing freezing conditions even in the summer, with a projected decrease of precipitation by as much as 80 percent (16).

Long term effects include an average annual temperature decrease of a few degees and the reduction of light of 5 to 20 percent (17). Nitric oxides generated by the nuclear fireball (a megaton explosion generates 5000 tons of nitric oxide) combined with the very heated smoke residing in the stratosphere. It would reduce the ozone layer by 50 percent causing ultraviolet radiation (UVB) to be increased by 40 to 100 percent for several years (17). The rising smoke could also displace the ozone layer toward the southern hemisphere. The nitrogen oxides combined with the sulfur oxides from the fires would greatly increase the acidity of rains. The release of large amounts of toxic chemicals and gases during the blasts and fires would cause serious local polution of water, air and soil.

Ecological effects arising out of environmental changes induced by nuclear war can not be fully determined because synergistic effects are greater than any of the subcomponents of a nuclear weapon deployment. Global mass starvation of human population would occur due to disturbances of ecosystems together with agricultural production and distribution. The availability of the fresh water supplies would be restricted among the other factors due to freezing. Contamination by the radionuclide products of nuclear fission of the fresh water and oceanic supplies and the introduction of the fission products into the food chain would lead to further death and diseases as a consequence of altered immune system in human and animals (18).

Whether considering effects on ecosystems such as a fresh water, oceanic or terrestrial, as a whole,or their individual components, it is of primary importance to recognize that a disruption of sunlight represents the energy source of photosynthesis, the transfer mechanism through which all life forms derive their energy. The balancing processes of plant respiration and decomposition of organic dead matter would also be affected. Thus, any assault on an ecosystem, as a whole or in part, would compromise its exisstence or its ability to function. Moreover, a disruption of processes or components could result in imbalances such as extiction or reduction of a plant or animal species. This could furtherlead to increased incidents of mutations, pandemias and death (19-23).

In addition to the diminished light, all surviving forms of life after a nuclear attack would be exposed to other physical stressors such as ultraviolet light, ionizing radiation, radionuclide contamination, cold, varied precipitation, acid rain, fires and pollutants. The extent to which these stressors would diminish and qualitatively reduce the life forms would depend to a

large extent upon the combination, duration, timing and length of exposure. The different studies of all of the weather and climate modeling, although containing some uncertanties, agree in general on the overall effects of the consequences of a deployment of a nuclear weapon, including generation of nitogen oxides, reduction of the ozone layer, increase the ultraviolet solar radiation for several years with a long time impairment of the planet's ecological systems, including the marine plankton and the food supplies from the oceans. However, different models including TTAPS, NCAR and Russian theories contain certain differences, with a Russian view that after initial cooling the temperatures may arise above normal. Various models are being integrated in the international Commities such as SCOPE (Scientific Committee on Problems of the Environment) for the ongoing continuos studies (24).

SOCIAL AND ECONOMIC CONCERNS

Without the ability to relocate people from the high-risk areas it has been estimated that less than one half of the population of The United States would survive Russian first strike (25). Since the social systems with which we are familiar would suffer an instant collapse, the survivors would depend on each other and follow the leadership of those demonstrating the most knowledge abour survival, as it was the case in Hiroshima and Nagasaki.

Reconstruction of the society is largely dependent upon resources outside the areas of impact. The magnitude of destruction likely to result from The Strategic Nuclear Confrontation would make an outside assistance improbable. Medical assistance to the victims of the combined injuries would be limited because of disintegration of the community and the post-disaster recovery would be tied with close kin relationships (25). Relocation would depend of the areas remaining intact for placement of evacuees, transportation systems and food supplies and the family unit will be most likely component of the foundation of rebuilding of the societal structure and function. Communication between government and citizens would be compromised due to the mssing chain of command and inoperable equipment. Transportation hindrance on large scale is anticipated because of a non-availability of vehicleas, repair parts and fuel. This would be an additional impediment of distribution of basic commodities such as food, water and medical supplies.

Strategic war's obliteration of the economic infrastructure in an industrial society would be immediate, complete and indiscriminate (26). Organized economiv operations would be annihilated due to the focus of impact on urban areas and strategic centers. Industrial production would cease,since there would be no replacement for essential machinery that performs basic functions relative to economic viability of the society. Transportation from storage facilities (primarily located in remote areas) would be unavailable

for the distribution of basic commodities, contrary to the information provided in the report of The Civil Preparedness Agency of The United States (27). The temperature decrease and radioactive fallout would eliminate economic potential in agriculture.

Society after a strategic nuclear war would undergo extreme fundamental changes with the availability of goods and services reduced to the bare minimum and total disappearance of the activities that are taken for granted in a normally functioning society would totally disappear (27). This would cause an instant change in the standard of living with a crime becoming one of the means of survival. If one were to predict a medium of exchange, it would be a barter system significantly less sophisticated than the medieval methods of commerce. The government and political structures would be dramatically impaired due to the authorities inability to respond to an emergency. Competition for leadership at the local level could impede relief efforts. The strain of a large scale damage and physical deprivation would lead to greater disaffection and hostility toward government and its representatives (16).

PSYCHOLOGICAL CONCERNS

Current research attempts to address the complexity of psychological impact by drawing historical analogies from man-made and natural disasters.

Several common behavioral changes apparent in survivors of catastrophes are that survivors suffer from a loss of meaning, loss of will to live, profound apathy and general depressed motivation state (28-31). At the family level, a small and very independent unit, a varying combination of post disaster stressors (i.e. degree of destruction, disorganization and casualties) introduce multidimensional consequences. Research invetsigating the effects of war upon children since aworld war II suggests that children model parental response to trauma (32, 33). Posttraumatic symptoms include psychosomatic complaints, insomnia, nightmares, chronic fatigue, fear of recurrence, fear of people and regrssive and overt agressive tendencies (32-34).

It is generally acknowledged in the literature that psychological disturbances following a nuclear disaster will be asociated with the state of a marked anxiety characterized by apprehensio, fear, confusion and irritability (35).Survivors of Hiroshima and Nagasaki were observed to display characteristics of physical numbness, survivor's guilt, mental decompensation, various psychoneurological disorders, permanent fear and uncertainty,a lifelong identification with the dead and fear of radiation and contamination of future generations (28). It is clear that the psychological reactions of survivors may continue for months or years following a nuclear exchange (28). Long term effects could include demoralization and severe disruption of the social structure .

Fuinally, the issue of assistance available to survivors of a nuclear attack need to be addressed. Several researchers outline the following factors that have to be considered: The number of victims with mental and behavioral disturbances, the number of mental health professionals available to provide treatment following a nuclear confrontation, the amount of time required for treatment, the availability of ytreatment facilities and availability of the pharmaceutical supplies needed for treatment (35). It seems apparent that no adequate treatment would be available for the vast number of psychological casualties.

THE CONCERNS OF THE VIABILITY OF NUCLEAR WAR

Thermonuclear weapons have radically changed the natute of the strategic battlefield. With the development of highly accurate delivery systems, multiple warhead missiles and huge arsenal of weapons, the opposing sides in any nuclear conflict face the real prospect that their countries could be subjected to attack and unprecedented destruction.

Continued theorizing in defense establishments about strategies for fighting and winning a nuclear war seems to be contradicted by the inescapable realities of the nuclear battlefield for some of the following reasons.

1. No effective defense against nuclear weapons has been developed and even with the advent of Strategic Defense Initiative, none is anticipated in the foreseeable future.Current Civil Defense planning does not offer any reasonable prospects for protecting civilian population in the event of a nuclear exchange.
2. Neither side could avoid substantial retaliation in a nuclear war even if it succeeeded in carrying out a relatively succesful first strike. Non-targetable, mobile launchers, such as submarines at sea and aircraft in the air would ensure that even the victorious side in such a scenario will sustain hundreds, if not thousands of strikes on its cities industrial facilities and military bases and installations.
3. It is a matter of considerable doubt whether any nuclear war once initiated, could remain limited or be readily contained or ended. Once either side has sustained a serious attack, the pressure, both politically and militarily to counterattack would likely be irresistible. Moreover, current counter-command strategies (i.e. targeting the other side's leadership for early strikes), might well ensure the elimination of anyone with a sufficient authority to quickly halt an exchange.
4. The consequences of any substantial nuclear exchange would be a massive destruction to both sides, including:

- The obliteration of major urban centers
- Hundreds of millions of direct and indirect fatalities
- Severe injury to surviving population, including radiation syndromes, combined injuries and radionuclide contamination
- Destruction of Medical Facilities and the disruption of health care, resulting in high mortality among the injured and pandemic spread of infectious diseases
- The widespread destruction of agricultural and industrial production, communication networks, transportation systems and economic infrastructure
- Mass psychological damage to survivors

5. Extensive scientific research in recent years has indicated that even a "modest" nuclear exchange is likely to have a severe and lasting impact on the biosphere. It now seems clear that these effects would be so serious and pervasive that even in an unlikely event that one side could attack first and completely escape retaliation, the "winner" will experience disastropus levels of environmental damage and millions of fatalities.

As on-going research expands our understanding of the probable impact of the nuclear war,the viability of thermonuclear weapons as "usable" instruments of war is increasingly thrown into question. In contrast to the myth of the winnable nuclear war, the reality of the strategic nuclear battlefield remains mutually assured destruction.

CURRENT CONCERNS ON NUCLEAR INDUSTRY ACCIDENTS

The most obvious peaceful use of nuclear energy since World War II, has been in the area of the power generation. However it was also through the military use of nuclear power that the technology advanced for the peaceful use of nuclear energy. The US Navy Submarine Nautilus used in January 1955 the first pressurized water power plant. The first commercial power plant in the United States was started in 1957 in Shippingport, Pennsylvania, while a commercial research reactor was started in Calder Hall in Great Britain. The Shippingport Reactor, which resulted directly from the Nautilus design was a PWR, while a Calder Hall reactor was a carbone dioxide cooled, graphite moderated plant, known as a high temperature gas reactor (HTGR). Both plants produced saturated steam of approximatelly 800 psig. There are various commercial reactors designs prevalent today, all of which are based on the process of nuclear fission. The basic reactor uses a geometric array of nuclear fuel in an appropriate container with a device to control a chain reaction and the method of moderating the energy levels for the intended purpose. The fissionable material commonly used is most commonly Uranium or Plutonium in either solid, liquid or gasseous form (36). The reactor fuel is mined, refined and enriched and formed into small pellets which are loaded into cylindrical metal rods arranged in a geometric pattern within a reactor core. The chain reaction occurs and is distinct from the rate of fission in the nuclear weapons by the fact that its rate can be controlled. This is accomplished by control rods which enhance or slow the chain reaction (37). The first control rods were made of cadmium which absorb neutrons and was developed at The University of Chicago. Although the term "control rod" refers to a mechanical device of a specific geometrical shape their use varies in various reactor designs (37). A moderating mechanism is used in a nuclear reactor to slow high energy of fast neutrons to make more of thermal neutrons, because the speed of neutrons, proportional to their energy, determines whether the neutron will cause a nuclear fission. The most common moderators in use today are demineralized or light water, heavy water or graphite. Nuclear fuel in the reactors is most commonly enriched natural uranium (U-235) enriched with U-238 in the ratio of 93.5 to 2.5 percent (38). Nuclear fission products contain several hundred of different radionuclides, highly radioactive, with a photon and or particulate emission. Among the fission products there are at least forty species potentially harmful to humans either by the organotropism, a long half-life, particle emission or all of these factors combined. As of July 1987 over of the 418 reactors in operation around the world PWR's were nearly 60 percent and of the 130 units under construction 76 percent. Current nuclear reactors are almost exclusively of PWR design (39).

The reactor core, placed in a stainless steel (pressurized in the PWR) vessel, cosists of several thousand long, thin and vertically oriented rods or tubes packed with enriched uranium dioxide (UO2) fuel. PWR have a normal operating pressure of 2250 psi. In PWR pressurized water is heated in the container as a result of a heat energy created in the fission process. The heated water passes from the reactor vessel to a separate heat generator which produces the steam. A typical PWR steam generator is about 60 Ft high and contains more than 3000 tubes through which the water from the reactor vessel passes and is returned to the reactor. Feedwater for generating the steam flows at a lower pressure around the outside of the tubes (40). Heat passes through the walls of the tubes from the hot pressurized reactor coolant water to the surrounding feedwater. Since the feedwater is at a lower pressure, it boils and produces steam, typically of about 293 degrees centigrade or 560 degrees F, and atmosphere pressure of about 1100 psig. Excess moisture from the steam is removed by an arrangement in the upper part of the steam generator (41).

The rate of fision is controlled by a neutron absober(boron), in the form of boric acid dissolved ina reactor water. Rods, containing a neutron absorbing material, such as an alloy of silver, indium and cadmium are used for fire control in the reactor.The control rods are arranhged in clusters and are entered into the core of the reactor. They are inserted and removed as required within the guide tubes located in the core assembly. The removal of the control rods causes density of free neutrons to build up, causing increased radioactivity. Radioactivity lasts for a long time after a fuel is removed from the reactor.

Containment in a nuclear installation is defined as a process of restricting to sharply defined volumes the distribution of radioactive materials which are involved in a nuclear fission. In a regular installation there are three concentric containment systems, which all have to be breached before any radioactivity is released to the environment. Containment structures for PWR are typically cylindrical with a domed top and made of reinforced concrete with a steel liner. The containment structure houses the entire primary coolant system, includingnthe reactor vessel, steam generators and pumps. The uranium fuel rods form the first and innermost system of containment. Second barrier includes the pumps, piping, steam generator and in a PWR a pressurizer. The third barrier is the structure of the containment vessel. The PWR vessel is normally dry and depends on volume for pressure limitations while,the BWR containment consisits of a dry well positioned on a suppression chamber. The two structures are connected through a series of parallel pipeline comprising the drywell vent system. The BWR containment carries a water inventory in the pressure suppression chamber.

Industrial accidents involving radioactive sources include medical institutions, research facilities, private industries that employ radioactive isotopes and Nuclear power Plants. The meticulous reference material on all of the types of the industrial accidents is well compiled and recorded (42). Of aa special interest are the Accidents at The University of Tennessee Comparative Animal Research laboratory where a researcher was exposed to 7700 Ci cobalt-60 source with the Total-Body dose of 260 cGy in 1971, a New Jersey exposure to cobalt-60 source of 120.000 Ci of an Industrial source with a Total-Body dose of 410 cGy, Oak Ridge exposure of eight workers at the plant Y-12 with a resulting Total-Body exposure of 235-365 cGy in 1958,Pittsburgh Pennsylvania accident with Van de Graaf linear accelerator with a resulting Total Body doe of one worker of 600 cGy in 1967, and numerous accidents involving plutonium leak in Miamisburg, Ohiao (1974), Americium exposure at DoE Hanford plant with a cumulative dose of 200 cGy (1976). In 1986 a chemical explosion of a storage tank containing urtanium hexafluoride resulted in a mass contamination of 26 residents and local workers, Ohmart Corporation in Cincinnatti accident in 1977, with the industrial radioactive sources, incidents in Tucson, Arizona in mid 1970's, dozens of international incidents with radiography industrial equipment, the wortst being in Algeria with Iridium-192 source in 1978, in Chiba, Japan in 1971 with Iridium-192, in China in 1963 with cobalt-60, Kerr-Mc Gee incident involving the death of Karen Silkwood where a possibility of a conspiracy attracted a wide attention of public media in 1979. Meticulous records of all of these events are kept in the Radiation Accidents epidemiology Registry by The Us DoE.

By far the most significant current global concern of the possibility of Nuclear Accidents of a mass casualties involves the potential disasters at The nuclear Power plants. Between 1970, and 1981. fifty US Nuclear Reactors released into the environment over 40 million Curies of radionuclides equivalent to Chernobyl disaster in 1986. The misleading contention that a Chernobyl-type accident is not possible in The United States, is illustrated by a probability of an explosion following a loss of coolant may lead to contamination of fuel with a water in a reactor vessel. This was testified before the congressional subcommittee in 1986 that in then existing 100 nuclear reactors in The US a probability of an accident would range between 12 and 45 percent.The most significant Nuclear Power plant accidents occured at NRX Reactor in Chalk River, Canada in 1958, Windscale, England in 1957,McKeesport, Ohio in 1960, Idaho Falls in 1961, Detroit Fermi Reactor in 1963, Hanford N Reactor in 1970, Calvert Cliffs emission in 1975, Browns Ferry, Alabama 1975, Rancho Seco, California in 1978, Pilgrim Reactor, Plymouth, Mass. in 1981, Ginna Reactor in Rochester N.Y. in 1982, Shippingport Reactor, Pennsylvania in 1971.

The worst accident involving the Nuclear power Plants in The United States occured at the Three Mile Island Unit 2 Reactor in Pennsylvania, resulting in 40 cGy/hour exposure in the nearby town of Goldsboro, only two miles from the plant. An average estimate of 16 million Curies of Radioactive Noble Gases and only 14 Curies of iodine-131 escaped into the environment. The controversy still exists about the incidence of cancers, leukemia,stillbirths, spontaneous abortions, hair loss thyroid disease and numerous other disease entieties among the humans and farm animals. The clean-up of the plant has been in the multibillion dollar range. Over 150 tons of radioactive was te has been transported to the Idah Falls awaiting its final repository. Over ten thousand temporary workers have been involved in the clean up process with an estimated 13000-46000 man/rem doses to be absorbed with still ongoing scientific debate of the probability of genetic defe cts.

CHERNOBYL UPDATE

The worst radiation accident in the history of nuclear power occured on April 26. 1986. at Chernobyl Nuclear Power Plant in Ukraine, when due to the human error an RBMK type Nuclear Reactor when the emergency core cool ing system was shut off in an experiment. In several seconds reactor power exploded shuttering the fuel rods and turning water to steam, followe by the second explosion probably caused by hydrogen blowing the radioactive material and burning graphite in the biosphere. Over 18 tons of radioactive material escaped into the environment. 30 kilometers exclusion zone was established around the plant, 140.000 people were evacuated within 11 days and approximatelly 17 million people received radioactive contamination including 2.5 million children under 5 years of age. The ongoing research indicates a significant increase of lymphocytic leukemia, multiple myelo and thyroid disease Although it is too early to estimate the ultimate outcome, the most recent BEIR V (Biological Effects of Ionizing Radiation) report, pointed out that the received radiation doses might be higher than initially considered. The current research provides still ongoing analysis of data obtained on th e followup of Chernobyl children in the different centers around the world, one of the most prominent being the medical and epidemiological analysis of the evacuated children in Kfar Chabad Center in Israel. The total amount of radioactive organotropic radionuclides released into the environment has not yet been determined with certainty. The most recent data indicate that Chernobyl accident released total radioactivity of 1-2 EBq excluding noble gases as xenon and krypton (44). Cesium-137 emitte d into the environment represents at least 30 percent of the total cesium-137 inventory in the core of the damag ed reactor (45).

LEGACY OF RADIOACTIVE WASTE

Radioactive waste is created in basically two different ways, one being fragments of nuclear fission from either nuclear power plants or nuclear weapons. The other major category of nuclear waste is spent nuclear fuel. When the fuel rods are irradiated in a nuclear reactor and withdrawn from the use the remaining fuel is still highly radioactive containing over 600.000 Curies per metric ton. The current accumulated radioactivity in The US in the spent fuel alone is over ten billion Curies. Radioactive decay from this fuel generates close to 40 Magawats of heat (46). By the year 2000 it is anticipated that over 73000 metric tons of spent nuclear fuel will be accumulated in The United States. The permanent storage sites for the spent fuel in The US is still an unresolved issue.

High level radioactive waste remains af ter uranium and plutonium are extracted from spent nuclear fuel during reprocessing. Military programs utilize reprocessinhg for recovering plutonium fro the producvtion of nuclear weapons and as a part of the fuel cycle of naval reactors. The current inventory of high-lebvel waste is over 300.000 cubic meters at the end of the year 1990. In The Unitewd States there are four sites of storage of high-level waste, including The Hanford reservation,Washington State, Savannah River Plant, South Carolina, Idaho Falls and The West Valley, New York State.

Another category is transuranic waste, mainly including uranium, plutonium, neptunium and americium from nuclear reactors.Most of transuranic wastes are being created by the military programs. In 1980, 24 million cubic feet of these wastes were buried at 8 of the govern,ment and 4 commercial sites in this country alone.

The last category is a low-level waste,including radioactive materials not created from reprocessing or tailings and having very low transuranic contents, including medical and research wastes, control rods fro,m the reactors, residues from uranium conversion, enrich,ment and fabrication and contaminated items used in the handling of radiation. In The US there are 6 low-level waste sites opreated by DoE and three commercial sites in operation, mostly bried in shallow trenches. In 40 years from now,all of the 111 commercial reactors and 93 military and reseach reactors will be too contaminated to continue operation, not counting 126 reactor-driven naval vessels that will all become part of the discarded low-level waste.

There are three basic strategies of managing radioactive waste The first category is to retain them until decay to harmless state, mainly intended for the short half-life radionuclides. The second category is to dilute them and disperse over a wide area. The third category is to concentrate them and prevent their migration in the environment., mainly intended for the waste of a long

half-life radioactive materials. Concentration of radioactive wastes can be achieved either by the evaporation of excess liquid,by ytheir precipitation as a solid from a large volume of material or by the process of burning the materials, retaining the ashes and filtering the gases.

The radioactive wastes of The United States have been studied for a disposal by the numerous methods. The main approach includes the storage in underground tanks, However it has been found prone to early leakage and contamination of the environment. In addition, they require an elaborate cooling system and constant attendance fro the integrity of the containment. Aboveground storahge has also been found impractical for the long-lived radioactive waste, although their cooling systems have certain advantages over underground tanks. Burial in the ocean floor, capped deep cracks or in the bottom silt was based on the dilution and has encountered considerable environmental controversies owing to concerns of permanent contamination of waters.

Permanent removal of radioactive waste from the biosphere by the rocket launcing into the space, appeared impractical from the viewpoint of a cost of over a billion dollars per launch and the risks of catastrophic consequences for the environment in the event of an explosion of a launch vehicle.

Another suggestion of disposal of the radioactive waste in the ice of Antarctica, although attractive, remains an area of a considerable controversy because of disruption of the ecosystems.

Several other methods have been entertained, including transmutation of the radioactive waste by the neutron bombardment, the creation of the zones as national waste monuments, separation of the high and low activity waste, deep geologic burial six to ten miles below the surface of the earth, or 3000 meters in the hard rocks, horizontal burial in the shafts of the mountains, burial on the lonely islands, solidification in the stable mineral matrix, the burial in canisters in mines and glassification of the radioactive waste, which is currently used in France. Grouting has been proposed as mixing of radioactive waste with cement and burial in shallow pits, a process being developed in Australia to incorporate waste into a mineral matrix of synthetic rock. Calcination is a proposed process of atomizing the wastes and drying them at higjh temperatures. Current approach in this country is The Waste Isolation Pilot Project in Carlsbad, New Mexico, which has been challenged by the possibility of leakage. WIPPhas encountered the resistence from the States of Colorado, Idaho and New Mexico. At present, the problem of radioactive waste remains unresolved. This particullarly refers to not even addressed waste amounts of radioactive waste in the former Soviet Union including ecological disasters of uncontrolled disposal sites in the Ural mountains, Chelyabinsk, Kyshtim and numerous undisclosed sites, including decomissioned military installations. This problem will remain the major global concern for the biosphere viability.

NUCLEAR TERRORISM

The risk of weapon-ready nuclear fuel being illegally acquired and made into actual nuclear weapons is a viable global concern , especially after 1980's which was declared a decade of terrorism. In June 1985 a conference was held in Washinhgton D,C, under the sponsorship of the Nuclear Control Institute and State University of New York Institute for Studies of International Terrorism that addressed The Nuclear Dimension of International Terrorism. The concluding guideliones of the conference included organizing an International Tesk Force for the Prevention of International Terrorism.

Following the demise of The Soviet Union the smuggling of nuclear fuel activities have been significantly enhanced for the reason of lesser control of the borders and easier availability the purchase of a nuclear weapon ready fuel or a theft of fissionable materials or nuclear weapons. An increasing number of intercepted smuggling activities of nuclear fuel are being routinely reported in the press, with a conclusion that plausibility of using nuclear material in terrorist actions increases with the increased accessibility to the theft and transportation of fissionable materials. The Gallup poll opinions consistently indicate that nuclear incidents involving terrorist acts are more plausible than even a tactical nuclear confrontation.It appears greatly enhanced by the State sponsored terrorism where the financial resources, intelligence, transportation and technical expertise add to the possibility of terrorist go nuclear. Recent, much publicized threats of Serbian self-styled government in occupied Bosnia is an example of a declared intent to attack a Nuclear Power Plant in Slovenia in the event of the tactical necessity in their war of agression. Over 260 Nuclear Power Plants througout the world are the prime targets of sabotage, including tjhe facilities of fuel enrichment reprocessing and fabrication plants and transportation routes. In 1978, the CIA emphasized the nuclear arsenals in Western Europe as potential target for the terrorist attacks, since a theft of a nuclear weapon would be an easier task for a terrorist act than the process of buiding the bomb. This report necessitates a physical securiry of the weqapon production and storage facilities as a primary concern for the security of over 50000 nuclear weapons currently present in the world, and all in the possession of the Superpowers, particullarly after the end of The Soviet Union. Two major routes of smuggling of nuclear fuel have been identified as Asian and Europen, mainly through Kazakhstan and Germany, respectively, including highly enriched uranium (HEU) and separated plutonium. Since both of th4ese materials are being used in the world commerce as civilian fuels for the research reactors (HEU) and power reactors (Pu-239), it is quite conceivable that shipment of nuclear fuel may become the target of terrorist hijackings. In October 1984 alone, there was a transportation of

United States originayted plutonium from France to Japan, enough to produce over 30 nuclear weapons. The original shipment plans were so poorly planned that the shipment had to be delayed for two years until sychronized by the US, French and Japanese Military commands at the cost of multimillion dollars security improvements. Regardless of increased awareness of the risk of theft of weapon-grade fissionable material and relative ease of producing a nuclear weapon, in the year 1981 alone there was an unaccounted missing of over 9000 pounds of fissionable material. This issue still remains unresolved, while in Russia and independent republics of the former Soviet Union, there is not even a basic database on the missing weapon grade fuel inventory. Hundreds of smuggling activities have been intercepted in the past several years, allowing a conclusion of much greater number of succesful smuggling of the weapon grade material, since it is not difficult to smuggle nuclear explosives across international borders. The black market value of a kilogram of cocaine is approximatelly the same as a kilogram of uranium. The psychology of treason is not always ideological as well exemplified by the recent cases of the US Navy personnel who betrayed the military secrets not for ideological reasons, but for financial gains. Phylosophical reasons such as a political and religious fundamentalism may play equally important role in nuclear terrorism. In September 1976. it was reported that Yugoslav officials assisted the international master terrorist Carlos escape apprehension with a small nuclear bomb, intended for a terrorist operations (47). The hoaxes of a threat of nuclear terrorism have been effectively used as a psychological weapon and they have been handled by the US NEST team (Nuclear Emergency Search Team), which acts in cooperation with other international agencies, as exemplified by the NEST-Canadian operation in locating and cleanining up debris of tje Soviet Nuclear Satellite in 1978. Since 1978, US NEST team has responded to over 80 nuclear bomb threats. The bombing of the World Trade Center in 1992, resulted in an awareness of a need of better preparedness for detection of nuclear weapon grade materials which are virtually undetectacble by the means of a conventional gamma detectors, being mostly alpha and beta emitters. The emerging times appear more serious than the decade of terrorism in 1980's because of an easier access to fissionable material and higher probability for a state-sponsored terrorism (48)

Nuclear terrorism is not limited to the weapons alone, since it is easier to use radiological weapons such as plutonium dust, which even in the amount of three ounces could provide a lethal effect for a building as a World Trade Center if evenly dispersed through the air conditioning system.

Release of radioactivity in the biosphere by targeting nuclear power plants as a form of nuclear terrorism has already been mentioned as a tactical weapon of renegade terrorist governments, as exemplified by the threat of Bosnian Sertb leadership to the Slovenian Nuclear Power Plant in Videm-Krsko in 1994.

The International task Force on Prevention of Nuclear Terrorism organized by The Nuclear Control Institute in Washington DC, outlines a number of ways by which terrorists can go nuclear, including a theft of nuclear weapons, interception of a shipment of fissionable mayterial, acquisition of the fissionable material in the reactors or waste sites, or using the radiological weapons as dispersal devices (50). The task Force presents a view of an increase in likelihood of nuclear terrorism because of growing incidence of a conventional forms of terrorism, state support of terrorist groups, laxity in the global safeguards at the nuclear materials sites, increasing use of civilian nuclear programs and an increase in a black market availability of fissionable materials. Accrding to the Task Force. there have been at least 155 attacks and violent demonstrations at the sites of civilian nuclear installations in the past 20 years, although none of which caused any accident of a significant impact (50).

Antinuclear terrorism preparedness must include bilateral and multilateral cooperation among the nations. The best example of a national program to cope with a nuclear catastrophy has been established in Switzerland where the civil defense program was formed as a part of national defense. Crisis management of the nuclear terrorism must include three essential components: readiness, response and recovery.Public awareness of a nuclear terrorist threat must be sychronized with a realistic and responsible media coverage with more emphasis on prevention than handling an actual crisis. The world governments have to be prepared to take terrorist threats seriously by identifying the sources, analysis of the motives, and a total military-style of their annihilation (51). At present it remains a source of a global nuclear reality in the light of well demontrated incapability of Community of Nations to cope even with the state sponsored non-nuclear terrorism.

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MR Imaging of Muscle Injuries

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Muscle pain related to exertion is common, particularly in athletes, and the severity and significance can be difficult to assess clinically. Before magnetic resonance imaging (MRI) was available, scintigraphy, ultrasound and computed tomography (CT) were used to evaluate muscle trauma (1-7). MRI is now the primary imaging modality for detecting muscle injury and for determining the type of injury and degree of muscle involvement (8-10). MRI is highly sensitive to muscle edema and hemorrhage and can therefore be of aid in evaluating muscle injury. Since MRI is sensitive to tissue alteration that is not apparent clinically, it can also aid in optimal management of patients following trauma, and can be used to identify various sequelae.

Familiarity with normal and abnormal appearances of muscle on MRI is important for evaluation of muscle injury. The primary focus of this chapter will be to discuss the various entities that one sees on MRI in patients following muscle trauma. These include the concepts of laceration, contusion, strains, herniation, delayed onset muscle soreness (DOMS), chronic muscle overuse syndromes, and hematoma. Following trauma, some patients will go on to develop fibrosis, fatty replacement, muscle ossification, as well as various abnormalities associated with compartment syndrome which are also well seen by MRI.

TECHNICAL CONSIDERATIONS

Normal skeletal muscle is characterized by an intermediate to low signal intensity on MRI. This is due to an intermediate to long T1 relaxation time and a relatively short T2 relaxation time. MRI is sensitive to acute and chronic changes in muscle water content (11-14). Various forms of muscle injury including necrosis, strain, and delayed onset muscle soreness (DOMS) are associated with an increase in T2 relaxation time and spin density (12,13,15,16) while T1 relaxation times can vary. Therefore, muscle injuries are more conspicuous on STIR and T2-weighted MR sequences which optimize the contrast between processes such as edema or more acute stages of hemorrhage and intact muscle.

Currently, muscle injuries are best demonstrated on short tau inversion recovery (STIR) pulse sequences. Fat-suppressed T2-weighted sequences show promise but have yet to be assessed. The short TI induces suppression of fat signal and makes the effects of prolonged T1 and T2 relaxation times additive resulting in high signal intensity in regions of muscle injury which provides optimal contrast for detecting muscle injury (17-20). The disadvantages of STIR imaging include relatively decreased signal-to-noise ratios and comparatively long imaging times (20). Other disadvantages of STIR which are more pronounced at high field include increased susceptibility to flow and motion

artifacts and a limited amount of slice locations per unit time. Therefore it is important to obtain T1-weighted and/or proton density sequences in addition to STIR for adequate anatomic detail. A faster STIR [FASTIR] sequence has been designed which uses large pixels, variable TR, and half-Fourier technique to provide 16 contiguous 1-cm sections in less than 1 minute (21). Fast STIR and Fast Spin echo T2-weighted imaging can effectively reduce scan times while maintaining sensitivity to edema. Further experience with this technique will determine if it can be used in place of conventional STIR imaging.

A short TR/TE sequence (T1-weighting) is useful for identifying and characterizing processes that have a short T1 relaxation time such as subacute hematoma (which contains methemoglobin), interstitial hemorrhage, and fatty infiltration. T1-weighting is also utilized for assessment of architectural distortion of muscle. Signal intensity may be slightly increased or decreased (if there is fibrosis) in injured muscle on T1-weighting. At times the lesion is isointense with muscle on T1-weighting and not detected at all (16).

T2-weighted spin-echo sequences can also detect various exertional muscle injuries but they are generally less sensitive than STIR. Unlike STIR, T2-weighted sequences may fail to distinguish edema from fatty infiltration in cases where there is only a small or moderate amount of edema (22). If STIR and T1-weighted images are obtained, time-consuming T2-weighted spin echo sequences are frequently unnecessary (18,19) .

The body coil is used for examination of larger structures such as the upper thigh and when comparison with the opposite side is indicated. Comparison views aid in detecting both alterations in muscle size and signal intensity. For localized muscle injury in the extremities, an extremity coil is used. The added signal to noise allows for higher spatial resolution which demonstrates fine detail in subtle lesions. If there is a palpable lesion or a painful area, the site of abnormality is delineated with oil- or water-containing skin markers which are placed above and below the region of clinical abnormality, allowing for precise localization of the lesion.

Images are obtained in the axial plane which allows for simultaneous visualization of all the muscles in cross-section. The transverse extent of the lesion and its relationship to surrounding muscles and the neurovascular bundle is determined. Coronal and/or sagittal images are also employed to demonstrate the longitudinal extent of muscle injury as well as the relationship of muscle lesions to osseous structures. The longitudinal planes are also useful for demonstrating tendon avulsions.

NORMAL MUSCLE PHYSIOLOGY

Exercise-enhanced MRI has shown dynamic muscle physiology and pathophysiology. With exercise, increases in muscle water normally occur in the extracellular space with low-intensity exercise, and also in the intracellular space at maximal intensity exercise (23). The T2 relaxation time increases with an

increase in water, resulting in high signal intensity on T2-weighted or STIR images. This effect has been seen when the work is performed during muscle shortening (concentric contractions), and to a lesser extent when the same work is performed during muscle lengthening (eccentric contractions). It is believed that the relative hyperosmolarity of exercised muscle, produced by products of glycogenolysis cause water to diffuse from the vascular space into the muscle (LJ). Blood flow is not required for this effect to occur (24), and the changes correlate with high rates of glycogenolysis and lactate production (11,25-28). This is further supported by the observation that an increase in signal intensity in muscle does not occur in patients with McArdle's disease, in whom glycogenolysis and lactate production are blocked due to myophosphorylase deficiency (29). The signal intensity changes visualized on MRI are dependent upon the intensity of exercise and the imaging parameters used.

Muscle fibers can be simplistically categorized into two major types- type I and type II (30-32). Type I fibers have a slower contraction time, low amounts of adenosine triphosphatase (ATPase) activity, low force per unit, and are more resistant to fatigue. These muscles are used for prolonged activity. Type II fibers have a longer contraction time, a high ATPase activity, undergo high peaks of tension in short periods of time, are optimal for short energy bursts, and usually are seen in muscles that cross joints. Most human muscle is mixed with variations in proportion of type I and II fibers. The Type II fibers are common in the biceps, triceps, quadriceps, and hamstring muscles.

MRI may also aid in the study of normal histological variations of composition in muscle fiber types, although findings in animals are reportedly different than those in humans. In the rat, T1 and T2 relaxation times are longer in muscles with type I fibers which are slow twitch and oxidative than those with type II fibers which are fast twitch and glycolytic (33). Kuno found the opposite to be true in humans (34). Type I fibers were associated with longer relaxation times than type II fibers. More work is needed to determine if MRI will be able to distinguish these different fiber types.

ACUTE MUSCLE INJURY

Laceration

A laceration is a direct muscle insult which results from a penetrating injury. The wound rapidly resolves with a residual scar and little muscle regeneration in the site. The portion of the muscle distal to the site is poorly innervated associated with chronic loss of muscle tension and shortening ability, resulting in muscle weakening (30,35). On MRI, one visualizes a transverse defect surrounded by a mass composed of blood and edema. The laceration is inhomogeneous on T1- and T2-weighted images (36). Laceration in the region of a tendon may result in its disruption. Denervated skeletal muscle resulting from laceration may not demonstrate signal intensity changes acutely, however

muscles of patients with subacute denervation have prolonged T1 and T2 relaxation times, which produce hyperintense signal on STIR or T2-weighted images (37). Chronically denervated muscle will be atrophic with high signal intensity fatty infiltration identified on T1-weighted images (37).

Muscle Contusion

A muscle contusion is produced by direct trauma to a muscle. Muscle pain is immediate and often prolonged. The direct trauma produces disruption of vessels and interstitial hemorrhage as well as edema and inflammation in the muscle. The muscle remains functional, associated with sequelae of spasm, pain, swelling, stiffness, and disability. Contusions are graded according to the severity of restriction in range of motion of the adjacent joint (38).

On MRI, the signal intensity reflects the inflammatory and edematous changes in the muscle. It is intermediate signal intensity on T1-weighted images, and therefore may be isointense with muscle. The signal intensity increases on STIR and T2-weighting, allowing for identification of the lesion (36,39). Contusions have a feathery, infiltrative appearance without significant architectural distortion or fiber discontinuity. The girth of the muscle often increases (36).

The goal of treatment is reduction of inflammation and early controlled movement. Contusions usually resolve on their own. They are usually not immobilized but the affected limb should be protected to avoid reinjury and myositis ossificans (40).

Muscle Strain

A muscle strain is produced by an indirect injury to muscle caused by excessive stretch or tension (35). A strain usually occurs at a point of critical tension that is proportional to the stretch in the muscle (30). It has been shown that certain muscles within a group of synergists are more prone to strain injury than others. Muscle strains are most frequent in muscles with the highest proportion of type II fibers, those that perform eccentric contractions which control or regulate movement and in those muscles that cross two joints. Commonly strained muscles include the hamstrings, rectus femoris, hip adductors and flexors, and medial gastrocnemius (10,35,41,42). Paraspinal musculature is also subject to strain, particularly in the lumbar (43) and cervical regions, and the workup of low back pain and "whiplash" injuries is often done using expensive and nondiagnostic tests. Strains due to excessive force are also common in muscles that are eccentrically exercised (stretched during contraction) (35,42.44-46). The musculotendinous junction is the site of most strains since it is the weakest link in the musculoskeletal unit (35,42,47). Myofascial strains are also visible on MRI.

Muscle strains can be difficult to evaluate clinically (48-51), and MRI can be used as an aid in diagnosis and treatment. It may be difficult to separate a

subcutaneous injury from a muscular one. Strains, like other muscle injuries, have MR imaging features similar to a variety of pathologic processes in muscle. Pathologically, the strained muscle demonstrates a combination of torn fibers, inflammation, edema, and some hemorrhage with protein degradation, and regeneration. Fiber disruption and hemorrhage occurs immediately following the injury, with infiltration of inflammatory cells and fibroblastic activity during the first 24 to 48 hours. Fibrosis increases thereafter with resolution of the inflammation. Tissue abnormalities appear focally in specifically affected muscle groups. Measurement of relaxation times of affected muscles in patients with acute strains reveal that both T1- and T2-relaxation times are prolonged producing an edema-like pattern of signal intensity manifest as intermediate signal intensity on T1-weighted images and high signal intensity on STIR and T2-weighted images. These signal intensity changes reflect underlying muscle edema, inflammation, and hemorrhage. MRI can demonstrate acute muscle strains and can also be used to guide clinical management and follow the injury to assess for tissue healing, which may occur well after complete clinical resolution (16). When a fascial or tendinous tear is small, the injury can be conservatively managed. On the other hand, when an associated fibrous tear is partial or complete, prompt surgery may be indicated to avoid retraction and fibrosis which would result in diminished function (35). Muscle herniation can also result (see next section).

A clinical grading system categorizes acute strains as first degree (mild), second degree (moderate), or third degree (severe) (52). This grading system is somewhat simplistic as it is often difficult to clinically categorize the strain. There are no precise correlations of MRI with this clinical grading system, however some guidelines can be followed.

The first degree strain produces minimal disruption of the tissue and there is no loss of muscle strength on clinical exam. There is less than 5% loss of motion distal to the injury. Pathologically, there is mild inflammatory cell infiltration, edema and swelling without significant muscle fiber disruption. The MR appearance is similar to a contusion. A diffuse, infiltrative pattern of edema and hemorrhage is seen, which can involve the musculotendinous junction and the muscle periphery if there is myofascial involvement. There is no evidence of architectural distortion. There may be an associated band of perifascial fluid, typically seen approximately three days following injury. These strains are usually treated conservatively with rest, ice, compression and elevation.

A muscle with a clinically apparent second-degree strain by definition has loss of strength associated with a variable degree of separation of muscle from tendon or fascia (this type of injury is basically a partial tear). On MRI, second degree strains demonstrate focal areas of muscle fiber disruption. Management of secondary strain depends upon the severity of the injury. Patients with less extensive involvement are conservatively managed while larger tears are repaired surgically. In addition to providing symptomatic relief, conservative treatment should be aimed at protecting the area with splinting or taping of the

injured muscle for several weeks in order to avoid a situation where the tear could become complete. Most second-degree strains resolve clinically within 10-14 days, however the MRI may remain abnormal for a much longer time (16,36).

In the third-degree strain there is muscle paresis with complete disruption of the myotendinous junction or tendo-osseous avulsion- a complete muscle tear (35,42,50). This is usually the result of a violent contraction of a muscle against firm resistance. On MRI, there is usually complete discontinuity of the muscle, usually at the musculotendinous junction, presenting as high signal intensity disruption on STIR and T2-weighted images. The muscle retracts and the torn ends may have a wavy or lobulated appearance. Focal fluid or "ganglionlike" collections may also be seen in the gap produced by the muscle tear, particularly with recurrent injury (20,53). Early surgical intervention is often indicated to avoid sequelae (spasm, retraction, more extensive tear, atrophy, scar, and muscle shortening) that can complicate repair.

MRI also detects edema and hemorrhage around the muscle and neurovascular structures as well as in the subcutaneous tissue.These fluid collections may persist and are composed of a combination of edema, muscle debris, and blood. In some cases, they may benefit from drainage (49).

HERNIATION (FASCIAL TEARS)

Focal herniation of muscle through a defect in the overlying fascia is most common in the lower leg, especially in the anterior tibial compartment (54). The usual etiology is localized blunt trauma that causes an interruption in the overlying fascia which allows the muscle to protrude through the fascia into the subcutaneous tissue. Muscle hypertrophy can also produce abnormal stretching of vascular fenestrations which allows the muscle to herniate (55).

Clinically, muscle herniation produces a palpable soft tissue mass. The mass is usually located along the anterolateral and middle to lower aspect of the leg (54,55). The lesion may be painless, particularly in chronic cases. The herniated muscle may be difficult to distinguish from other causes of soft tissue mass.

MRI is useful for characterizing the mass in suspected cases of muscle herniation, since muscle tissue has a distinct MR appearance (15,56). MRI is more useful for evaluating muscle herniation than CT because unlike CT, on MRI, muscle and fascia are of different signal intensity and the herniation can be seen in multiple planes. MRI is also of use preoperatively in delineating the extent of fascial discontinuity however, the fascial defect may not always be demonstrated (56). The scans are easier to evaluate if the mass has been delineated with skin markers and if the contralateral side has been included for comparison. Kinematic studies can be employed during muscle contraction and relaxation to bring out the herniation. Contusion and edema may be seen in the muscle in acute herniations. Muscle strangulation can also occur if the fascial tear forms a tight band around the herniated muscle.

Once the diagnosis of muscle herniation is made, asymptomatic patients are usually left alone or treated with conservative measures such as support stockings. Symptomatic patients who do not respond to conservative measures are treated with fasciotomy. Acute muscle herniation is generally not repaired by fascial closure because of the potential for a compartment syndrome (57).

DELAYED-ONSET MUSCLE SORENESS (DOMS)

Fatiguing exercise may be followed by a variable amount of muscle pain, soreness, swelling, and joint stiffness. When it occurs in otherwise healthy people several hours to days following exercise, it is termed delayed-onset muscle soreness (DOMS) (44,46). DOMS most commonly follows eccentric contractions (58). Eccentric contractions require less oxygen and energy, produce less lactate, and use smaller numbers of muscle fibers than concentric contractions.

In DOMS, the soreness increases progressively over the first 24-48 hours following exercise, peaks at two to three days, and usually resolves by seven days (58,59).The cause of these symptoms is not known but is thought to be related to increased intramuscular fluid pressure, inflammation, and/or damage to the corresponding connective tissues (44-46,60-62). The pain is most commonly localized to the musculotendinous junction. Pain receptors are prevalent in regions of the tendons and related connective tissues (63-65). Pain can sometimes be alleviated by massage or when the motor task that caused the DOMS is performed, but the soreness returns following this exercise. Pain and soreness during DOMS may be partially caused by the tension that muscle swelling produces on the muscle fibers (59,61). However, the symptoms may disappear before the swelling has resolved (61).There is a marked decrease in the ability of the patient to perform exercise using the affected muscle due to a reduced voluntary effort related to the pain as well as a decreased ability of the muscle to produce force (44,58). The process is usually self-limited, and there does not appear to be any treatment. Occasionally, the muscle goes on to necrosis (rhabdomyolysis), especially in sedentary muscular men (22).

The appearance of DOMS on MRI is similar to a first-degree strain and, as defined above, clinical history is essential for differentiation. Muscles that are painful following exertion frequently demonstrate abnormal signal intensity on MRI, but the distribution of the signal abnormality may not occur in all muscles that were used during the exertion (16).The area of abnormality is often seen near tendinous or fascial attachments. Muscle T1 and T2 relaxation times increase, consistent with the presence of edema, with a possible minor contribution from hemorrhage that is produced in response to muscle injury (16,28). Perifascial fluid-like collections that can represent a combination of edema from muscle necrosis as well as hemorrhage are sometimes seen in the early phase of the injury. These disappear with resolution of symptoms and return of creatine kinase levels to normal (16). Abnormal signal intensity may remain for up to 80 days following injury (58,66).

In a recent study of mild DOMS by Nurenberg et al, good correlation was found between the grade of MR signal intensity on T1-weighted and spin-density images and the degree of ultrastructural injury in the form of disrupted proteins on muscle biopsy 48 hours after exercise (67). However, there was no correlation between areas of muscle soreness (DOMS) and areas of signal intensity increase. This interesting finding requires more investigation.

There is often a disparity between symptoms and MR imaging abnormalities. Symptoms and abnormal creatine kinase levels may disappear before resolution of the abnormal changes observed on serial MR images (16,58). Therefore, clinical assessment of muscle injuries may be insufficient to demonstrate the total recovery of injured muscles in exertion-induced damage, and a serial evaluation of the muscles on MRI may be of use for accurate determination of recovery.

With severe DOMS, postexercise necrosis (also called exertional rhabdomyolysis) can be seen. This usually occurs following intense physical activity in individuals who suddenly engage in sports activity to which they are not accustomed (the "weekend jock"). Histologically, there is myocyte swelling, inflammation and hyaline degeneration (68). MRI is highly sensitive to muscle necrosis (27,69). On MRI, myonecrosis produces high signal intensity on spin-density, T2-weighted, and STIR images with minimal changes in signal intensity on T1-weighted images unless it is very severe (16,68).

CHRONIC MUSCLE OVERUSE SYNDROMES

MRI alterations can be seen near the myotendinous junction in some occupational (musicians) and recreational (tennis elbow, little league elbow) overuse syndromes resulting from chronic or repetitive muscular stress (22). Recurrent muscle strains can devastate athletes (70) and cause absence among workers, including any whose occupations involve repetitive movements. Muscle overuse syndromes are poorly documented with a lack of objective tests for confirmation of tissue abnormality (71-79). In this regard, MRI can provide objective evidence of a patient's complaint and can demonstrate the location and degree of involvement at the musculotendinous junction, allowing for proper treatment. It can also play a role in legal judgements and workers' compensation. Findings on MRI may include persistent hyperintense signal intensity in the region of the injury on T2-weighted or STIR images. The area of abnormality may also increase in size or surround a previously formed fibrous scar (20). MRI alteration in strained muscles can persist longer than clinical symptoms, suggesting ongoing repair (16). Confirmatory studies are clearly needed, but it is tempting to speculate that restraint from full-level exercise may be necessary until the muscle returns to normal on MRI.

HEMATOMA

Hematomas are usually well-defined, loculated masses that are frequently located in a single muscle. The MR signal intensity of the hematoma is variable and is dependent upon the field strength of the MR imaging system as well as the chronicity of the lesion (15,39). Evaluation of hematoma is best accomplished with T1- and T2-weighted sequences, where relative oxidative states of hemoglobin can be more specifically characterized (20). The relaxation time of hemorrhage is affected by several factors: 1) concentration of protein; 2) methemoglobin content; 3) T2 proton relaxation enhancement by local heterogeneity in magnetic susceptibility at high field strength; and 4) tissue clearance. There is often considerable inhomogeneity within the hematoma, reflecting different stages of hemorrhage. Acute hematomas can have signal characteristics similar to fluid with both long T1 and T2 relaxation times. They may appear low signal intensity or isointense with muscle on T1-weighted images and isointense with muscle or high signal intensity on T2-weighted or STIR images. With aging, the protein content increases and the water content is reduced resulting in lower T1 and T2 relaxation times. Subacute hematomas are frequently higher signal intensity than surrounding muscle on T1- and T2-weighted MR images partly due to the presence of oxidative denaturation of hemoglobin, resulting in the paramagnetic substance, methemoglobin, which shortens T1 relaxation time (8,80,81).A central low signal intensity area may be seen within a hematoma on high field strength images which due to T2 shortening produced by deoxyhemoglobin within intact red cells. A rim of low signal intensity may be seen surrounding the chronic hematoma due to the presence of hemosiderin-laden macrophages (8). Chronic hematomas have a large amount of hemosiderin which produces low signal intensity on T2-weighted images. Importantly, it may be difficult to distinguish hematomas from other masses including sarcomas and abscesses, emphasizing the critical need for accurate clinical information before concluding a benign diagnosis. (82,83).

MESENCHYMAL COMPLICATIONS OF MUSCLE INJURY

Occasional sequelae of muscle injury include fibrosis, atrophy, and muscle ossification (10). Calcific myonecrosis, compensatory hypertrophy and compartment syndrome may also be seen following muscle injury. Persistent MRI abnormalities such as hematoma or scar may predict injury recurrence (20,53).

Fibrosis

Recurrent or severe injury, regardless of the cause, may result in fibroblast proliferation and subsequent fibrosis (20,84,85). When there is a delay in treatment, surgery can be difficult if the muscle is fibrosed (35). Post-traumatic scar is also associated with recurrent muscle strains. On MRI, chronic fibrosis presents as an area of low signal intensity on all imaging sequences (20,86).

Atrophy and Fatty Change

Muscle atrophy (loss of bulk) and fatty change (mesenchymal infiltration with fat) are rare following trauma in otherwise healthy individuals, and must be distinguished from other causes of myopathy. These changes are frequently secondary to immobilization and can result in rapid loss of muscle strength. Muscle atrophy can be extremely rapid, developing 5 to 10 days after immobilization, and can be irreversible by 4 months (87). Atrophy is reflected as a decrease in muscle size, which is best determined in more subtle cases with comparison to the opposite side. MRI can be used to differentiate fat from most other substances because of the characteristically short T1 of fat. Fatty change is reflected on MRI as diffuse areas of increased signal intensity within the muscle on T1-weighting (88).

Myositis Ossificans

Myositis ossificans circumscripta is a sequela of muscle trauma that typically presents as an area of soft tissue swelling that eventually undergoes ossification. It can be seen following muscle trauma, burns, and immobilization due to paralysis or coma. The exact etiology of myositis ossificans is unknown but is thought to include connective tissue metaplasia and/or ossification of hematoma (89).

Clinically, the area is at first painful and swollen with loss of muscle function disproportional to the severity of the muscle trauma. Symptoms tend to increase with time rather than resolve, as in an uncomplicated contusion. Histologically, in the first 3-4 weeks there is a central fibroblastic reaction which organizes peripherally. The lesion can be confused histologically with a sarcoma. Peripheral new bone formation is seen at 6-8 weeks in areas of interstitial hemorrhage (52,89). By 6 months, the bone matures into compact bone in the periphery with a central core of lamellar bone (89,90). Joint motion is often regained and osseous resorption follows.

MRI can detect edema within muscles adjacent to areas of ossification, prior to the detection of ossification on conventional radiographs, allowing for early identification, localization, and intervention (22). MR features of myositis ossificans are variable depending upon the degree of maturity, and are not pathognomonic (90-92). Imaging characteristics can be similar to other types of muscle injury and masses. In the early phase, prior to ossification, the lesions are ill-defined and are usually isointense to muscle on T1-weighted images and higher signal intensity than fat on T2-weighted images. In some lesions there is central high signal intensity on T1-weighting. Fluid-fluid levels may also be seen (90,91). Edema may also be seen in the marrow adjacent to the area of myositis ossificans. A low signal intensity rim may be present. In subacute lesions with early peripheral ossification, the central portion of the mass may be isointense or slightly increased in signal intensity relative to muscle on T1-weighted images and high signal intensity T2-weighted images. A low signal intensity rim is seen

surrounding the lesion in areas of ossification. Peripheral soft tissue edema is still present. Bone marrow edema may also be present. More mature lesions often demonstrate inhomogeneous areas of signal intensity that are isointense with fat and represent fatty marrow within trabeculae as well as low signal intensity cortical bone within and surrounding the lesion (90,91). Occasionally the chronic lesion may present with low signal intensity on all imaging sequences, corresponding to dense ossification and fibrosis (91).

Myositis ossificans can sometimes be mistaken for other soft tissue masses- both benign and malignant (91). Since MRI is used for detection and characterization of soft tissue masses, it is important to consider myositis ossificans. If a rim of low signal intensity is seen, conventional radiographs and/or CT should be scrutinized for characteristic features of peripheral ossification.

Calcific Myonecrosis

Another uncommon and late complication of muscle trauma is calcific myonecrosis, which can be evaluated with CT or MRI (93). Calcific myonecrosis usually involves a single muscle, which can distinguish it from dermatomyositis and polymyositis, which have more diffuse involvement. This sequela appears to be associated with peripheral nerve injury and has been described in the calf muscles following involvement of the common peroneal nerve. One sees a fusiform mass with plaquelike peripheral calcifications replacing the muscle. Calcific myonecrosis lacks central calcifications and has fluid centrally. On MRI one may see central homogeneous signal intensity consistent with fluid surrounded by a calcified rim of low signal intensity cartilage. This can be distinguished from myositis ossificans which typically demonstrates osseous tissue in the periphery with progresses centrally, with extensive ossification usually present by 6 weeks (94).

Compensatory muscle hypertophy

Occasionally, compensatory hypertrophy can result in muscles surrounding an area of chronic muscle tear (15). MRI is useful for characterizing the hypertrophy, and if a mass is present, to distinguish muscle hypertrophy from other abnormalities including tumor.

Compartment Syndrome

Compartment syndrome is another complication of hemorrhage, direct trauma, and exertional muscle injuries as well as fracture that is characterized by muscle edema and/or hemorrhage within confined fascial boundaries. The result is increased intracompartmental pressure measured between 15 to 20 mm Hg at rest (normal =0-4), and increasing to approximately 75 mm Hg during exercise (normal=50) (2). This rise in pressure can impair capillary perfusion and oxygen supply. The syndrome is characterized by pain, intact pulses, and sensory deficits. Compartment syndrome can be confused with deep venous thrombosis

or a ruptured popliteal cyst and can be difficult to diagnose clinically. The most common locations are the anterior and lateral compartments of the leg and the anterior compartment of the arm.

In patients clinically diagnosed with compartment syndrome, MRI can be of use in the evaluation of the muscle, vessels, and subcutaneous tissues. It can also be used to noninvasively define the compartments of involvement for the surgeon (9,26,95). Findings on MRI include unilateral increase in muscle size and increased signal intensity within the muscle on STIR and T2-weighted images. Qualitative assessment of blood flow to the muscle can be obtained with intravenous contrast material such as gadolinium (9,22). It is important to remember that mass-like changes in muscle may be the result of a benign or malignant tumor, rather than compartment syndrome, even in patients with clinical symptoms of compartment syndrome, and it may be difficult to distinguish these entities on MRI.

Compartment syndrome is a surgical emergency, usually treated by fasciotomy and debridement of necrotic tissue to avoid muscle necrosis and fibrous contracture. MRI can be utilized postoperatively as an aid for evaluating the extent of necrotic and viable muscle. Exercise-enhanced MRI purportedly improves assessment of chronic compartment syndromes as well (26,95).

Denervation

Trauma to peripheral nerves frequently results in denervation and abnormal signal intensity in the affected muscles on MRI. Because of this it has been speculated that MRI may represent a monitoring tool that can replace the more invasive, inexact, and time-consuming electromyogram. MRI is the only imaging technique that can demonstrate subacute denervation of muscles prior to fatty deposition.

In a study of denervation in rats, Polak et al demonstrated T1 and T2 prolongation 15 days after transection of the sciatic nerve (96). This was caused by fiber atrophy (shrinking of the myoplasm) and reduction in intracellular water with compensatory increase in extracellular water, producing an edema-like pattern rather than true edema. This results in a prolongation of proton T1 and T2 relaxation times on MRI (97,98). At this acute stage, changes in muscle from denervation are reversible. With chronic denervation, T1-shortening results from fatty infiltration, which may be irreversable. The signal intensity may remain normal during the first few weeks of denervation. Further studies are needed to identify the etiology of the signal changes in denervation. Changes may occur at different rates in muscles with the same denervation. This may be related to collateral nerve supply or variable rates of fiber atrophy.

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MRI OF THE ELBOW

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Many abnormalities seen in the elbow are a result of trauma, often from sports such as baseball and tennis. Elbow abnormalities seen on MRI include distal biceps tendon ruptures, tears of other tendons and ligaments, tendinitis, loose bodies, osteochondritis dissecans, fractures, nerve entrapment, arthritides, and masses, including bursae (1-8).

BICEPS TENDON

Rupture of the distal biceps tendon is infrequent, accounting for 3-10% of all biceps tendon ruptures (9). It is usually caused by a single event, often 40 kg or more against resistance with the elbow in approximately 90 degrees of flexion. Tears occur at or near the distal insertion site on the radial tuberosity and can sometimes be difficult to clinically diagnose, especially in situations where the bicipital aponeurosis remains intact with minimal proximal retraction of the biceps. The muscle contracts proximally with elbow flexion with marked weakness of flexion and supination. A soft tissue mass may be palpated in the antecubital fossa. It is important to document rupture of the biceps early so that a repair still can be done. If the tendon is left torn, it usually retracts into the substance of the biceps muscle, making retrieval and reattachment difficult, if not impossible.

MRI is useful for confirming distal biceps tendon ruptures, distinguishing partial from complete tears, and excluding other space-occupying lesions as a cause for the mass (5,6). The tendon should be followed to its insertion on the radial tuberosity on both axial and sagittal MR images. With distal rupture, the tendon is not visualized near its insertion on the proximal radius. There may be fluid in the empty tendon sheath. As with other tendinous and ligamentous tears, MR images demonstrate a gap in the low signal tendon at the site of a complete tear (10-12). There is proximal retraction of the biceps muscle. In acute tendon injury T1- and T2-weighted spin-echo, STIR, or gradient echo images show increased signal intensity in the region of the tear due to hemorrhage and inflammatory response. Partial rupture of the tendon may produce enlargement or attenuation in the region of abnormality.

TRICEPS TENDON

Rupture of the triceps tendon is rare. It usually occurs from a direct blow or a deceleration force to the arm while the triceps is contracted, usually after a fall. Sports that rely on repetitive elbow extension such as bowling, pitching and weight-lifting can also produce triceps tendon disruption as can systemic

disease or steroids. Axial and sagittal MR images are useful for evaluating tears of the triceps tendon (13). The triceps tendon can also sublux over the medial humeral epicondyle. To demonstrate this, one can do axial images in pronation and supination in varying degrees of elbow flexion.

EPICONDYLITIS

Epicondylitis is characterized by abnormalities of the tendons that attach on the medial and lateral epicondyles of the distal humerus. It can be caused by repetitive movements of the wrist with pronation or supination of the forearm. Lateral epicondylitis or "tennis elbow" occurs at the origin of the extensor carpi radialis brevis. It is commonly associated with varus stress to the elbow. Medially, abnormalities are seen at the origins of the flexors and pronator teres ("golfer's elbow"). These tendons are often injured from excessive valgus stress to the elbow, which can occur in baseball pitchers (4), javelin throwers, football quarterbacks, golfers, tennis players, and violinists.

Many elbow problems are related to the medial tension-lateral compression phenomenon where repeated valgus stress produces flexor-pronator strain, ulnar collateral ligament sprain, ulnar traction spurring, and ulnar neuropathy. The lateral compression causes osteochondritis dissecans of the capitellum and radial head, degenerative arthritis, and loose bodies.

MRI can be used to evaluate the pathologic processes associated with excessive varus and valgus stress to the elbow. Coronal and axial sequences are useful for assessing the degree of tendon injury. One may see partial or complete tendon rupture, tenosynovitis, tendinitis, or tendinosis. Histologic studies have demonstrated that many of the affected tendons have angiofibroblastic hyperplasia with lack of inflammation suggesting that some of the abnormal signal intensity demonstrated within the tendons on MRI is caused by tendon degeneration and repair rather than tendinitis (13). This phenomenon is termed tendinosis or tendinopathy and is manifest as normal to increased tendon thickness with increased signal intensity on T1-weighted images, which does not further increase in signal intensity on T2-weighted images. On MRI there is usually high signal intensity in this region on T2-weighting as well as STIR imaging if there is a complete or partial tear. Partial tears are characterized by thinning of the tendon, which is usually outlined by adjacent fluid on the T2-weighted images. Complete tears are diagnosed on MRI when there is a fluid-filled gap separating the tendon from its adjacent epicondylar attachment site. MRI is useful for identifying high-grade partial tears and complete tears, which are unlikely to improve with rest and repeated steroid injections. Surrounding muscles, may demonstrate increased signal intensity due to edema, granulation tissue and inflammation, as has been reported in the anconeus muscle in patients with lateral epicondylitis (7). One word of caution is that abnormal signal intensity within a tendon or neighboring muscle can also

be produced by therapeutic injection for epicondylitis. It is important to inquire about this in the initial patient questionnaire.

COLLATERAL LIGAMENTS

The ulnar and radial collateral ligaments can degenerate and tear with or without injury to the overlying flexor or extensor tendons. These ligaments are well evaluated with MRI. Arthrography is not useful for detecting tears except in the early stages following acute rupture (within 24 hours) (2,11). Stress radiography (14) can be used to diagnose tears of the ulnar collateral ligaments, however additional abnormalities that are so frequently seen in association with these tears will not be completely assessed using that method.

The radial collateral ligaments are weaker and thinner than the ulnar. They offer varus stability and are rarely stressed in the athlete. The anconeus muscle also contributes to joint stability. The radial collateral ligament complex is variable and has three components, the radial collateral ligament proper, which extends from the lateral epicondyle of the humerus to the annular ligament, the accessory collateral ligament and the lateral collateral ligament. The lateral collateral ligament is present in 90% of people and provides the primary restraint to varus stress. The radial collateral ligament proper and the lateral collateral ligament should be evaluated separately. Disruption of the lateral collateral ligament results in the recently described pivot shift phenomenon of the elbow. Unsupected ruptures of the radial collateral ligaments may occur with tears of the common extensor tendon.

The ulnar collateral ligament originates from the medial epicondyle and attaches medial to the coronoid process of the ulna. It consists of three parts. The major ligament is the anterior oblique bundle, which is taut with extension, and inserts on the ulna along the medial aspect of the coronoid process- the sublime tubercle. The other components are the posterior oblique, which is fan-like, smaller, and taut with flexion, and the transverse segment which bridges the ulnar attachments of the anterior and posterior bands. The posterior oblique and transverse segments are often difficult to define on MRI and may be absent. These latter two ligaments do not have a definite functional role in terms of maintaining elbow stability. Rupture of the ulnar collateral ligament usually occurs in the flexed elbow with valgus stress. This ligament is injured in throwing sports. In contrast to medial tendinitis, injury to the ulnar collateral ligament in the throwing athlete can be devastating, since athletic performance is hindered due to pain and altered biomechanics. On MRI, one looks for increased signal intensity within and adjacent to the ligament, which can represent degeneration, hemorrhage and edema due to microtears resulting from repetitive injury (4). Most tears occur in the mid proximal fibers of the anterior bundle. The injured ligament can also display thickening and irregularity, ligamentous laxity, and poor definition. Recovery is generally slow, and surgery is often required. Primary repair of the frayed ligament is usually

not possible, and instead, insertion of a tendon graft is more common using either the palmaris longus, plantaris, or tensor fascia lata. Postoperative recovery requires 12-18 months. Ulnar traction spurs are frequent with a thickened collateral ligament (75% baseball pitchers) and can extend into the region of the cubital tunnel and irritate the ulnar nerve. Laxity of the ulnar collateral ligament may also lead to incongruity between the medial olecranon and the medial aspect of the olecranon fossa, resulting in loose body formation (15,16).

LOOSE BODIES

Intraarticular loose bodies are frequent in the elbow and may result from destruction of bone or cartilage following excessive elbow stresses, osteochondritis dissecans, osteonecrosis, or osteoarthritis. They may also be produced in an uncommon disorder known as primary synovial osteochondromatosis. Loose bodies can produce mechanical symptoms such as locking and limitation of motion and they can lead to premature degenerative arthritis. They are usually removed arthroscopically. MRI has advantages over imaging modalities such as arthrography in the search for loose bodies because it provides a non-invasive multiplanar assessment of complex joints such as the elbow. Loose bodies vary in signal intensity on spin-echo images, depending on the marrow content of the fragments. They are usually surrounded by intraarticular fluid and are well seen on T2-weighted or small flip angle gradient echo images. Small losse bodies may be difficult to exclude with MRI, however, in the absence of a joint effusion. An accessory ossicle, the os supratrochlea dorsale, can lie in the olecranon fossa and simulate a loose body.

OSTEOCHONDRITIS DISSECANS

Osteochondritis dissecans is a frequent source of joint pain and loose bodies in the elbow, particularly in adolescents. The incidence in the elbow is 25% that of the knee (17,18). The etiology is unknown, but is thought to be post-traumatic, secondary to chronic lateral impaction from exaggerated valgus stress. It is often seen in the dominant upper extremity in children between the ages of 9-15 years. When it occurs in the elbow, it is seen in the capitellum. Stable osteochondral lesions are usually treated with rest and splinting, whereas unstable lesions are either removed or pinned (19,20). MRI can be utilized to identify the lesion, assess the condition of the overlying articular cartilage, and determine if there are any signs of loosening, which can be suggested when there is fluid between the fragment and the parent bone or by identifying a cystlike lesion beneath the osteochondral fragment (21). MR arthrography may also play a role in the evaluation of loosening. MRI can also aid in identification of loose bodies, which can result from this disorder.

FRACTURES

MRI is generally not indicated for the evaluation of a known fracture in the elbow, however, as in the knee, unsuspected fracture or contusion may be demonstrated in a patient with recent trauma and elbow pain. Supracondylar fractures of the humerus represent 50 to 60% of fractures in children, while radial head fractures are more common in adults (15). Other areas prone to fracture in the elbow include the olecranon and proximal ulna. MRI is useful for demonstrating occult stress fractures of the olecranon, seen in throwing athletes due to overload by the triceps mechanism (22). If untreated these fractures can go on to true fractures which must be treated surgically. The anterior coronoid process may be fractured or contused following posterior elbow dislocation and this may be difficult to see on conventional radiographs since the coronoid process is often covered by the radial head. Surrounding soft tissue damage associated with fracture or dislocation of the elbow can also be assessed with MRI. MRI is useful for evaluating the extent of trauma in the child's elbow, since the articular cartilage is well demonstrated. Dislocation at articular surfaces as well as extension of fractures into the cartilaginous epiphysis is well seen with MRI (23). The medial epicondylar apophysis may avulse in skeletally immature baseball players and MRI may detect these injuries before complete avulsion and displacement (13).

NERVE ENTRAPMENT

Nerve entrapment syndromes are frequent in the elbow, and MRI can be used to assist in their detection (8). The three major nerves that traverse the elbow are the ulnar, median and radial nerves. Each of them are subjected to entrapment and compression from a variety of causes. Abnormalities of the nerves may manifest on MRI in one or more of the following ways: increased signal intensity on T2-weighted spin-echo images, focal changes in girth, and deviation, including subluxation or displacement by a space-occupying mass (8). Keep in mind that such displacement may or may not be associated with symptoms.

The ulnar nerve is the most frequently injured nerve due to its location in the cubital tunnel, which is a tight compartment posteromedial to the humerus. The floor of the tunnel is formed by the capsule of the elbow and the posterior and transverse portions of the ulnar collateral ligament. The roof of the tunnel is formed proximally by the cubital tunnel retinaculum and distally by the flexor carpi ulnaris aponeurosis. Anatomic variations of the retinaculum may affect the ulnar nerve. The retinaculum may be absent in 10% of cases, allowing the nerve to dislocate anteriorly over the medial epicondyle during flexion, producing a friction neuritis. The retinaculum may also be thickened resulting in dynamic compression of the ulnar nerve during elbow flexion. An anomalous muscle, called the anconeus epitrochlearis may replace the retinaculum in 11%

of cases, causing a static compression of the ulnar nerve. In addition, there are many other causes of ulnar neuritis including thickening of the overlying ulnar collateral ligament, medial osteophytes projecting into the cubital tunnel, adhesions, muscle hypertrophy, direct trauma, and callus from a fracture of the medial epicondyle. MRI can be used to identify these abnormalities and to assess the ulnar nerve itself. When compressed, the nerve may become enlarged and edematous. If conservative treatment fails, the nerve can be transposed anteriorly, deep to the flexor muscle group, or more superficially, in the subcutaneous tissue. One can follow these patients with MRI postoperatively if they become symptomatic to determine if symptoms are secondary to scarring or infection around the area of nerve transposition.

The radial nerve can become entrapped following direct trauma, mechanical compression by a cast or overlying space-occupying mass, or a dynamic compression as a result of repeated pronation, forearm extension, and wrist flexion as is seen in violinists and swimmers (15,24,25). Compression of the median nerve may be seen with osseous or muscular variants and anomalies, soft tissue masses and dynamic forces.

TUMORS AND SYNOVIAL PROCESSES

MRI is superior for delineating the extent of benign and malignant masses in and around the elbow (26). Occasionally, the lesions can be further characterized with MRI, particularly in benign conditions such as lipoma, hemangioma, bursae, or ganglia.

There are a number of bursae around the elbow. The most commonly distended bursa is the subcutaneous olecranon bursa. Olecranon bursitis may be seen in patients with gout and rheumatoid arthritis. Bursae can form along the medial and lateral epicondyles, and should not be confused with epicondylitis. A medial epicondylar bursa may compress the ulnar nerve in the cubital tunnel.

MRI can also be utilized to evaluate the progression of arthritides in the elbow. The degree of intraarticular effusion, as well as the condition of the hyaline cartilage and subchondral bone can be assessed. Response to therapy can be monitored using MRI.

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CONGENITAL HEART DISEASE - BASIC PRINCIPLES

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An orderly approach to the analysis of the plain film in congenital heart disease is presented. It begins with an assessment of the pulmonary vascularity and thereafter an assessment of individual chambers, and finally an assessment of the aorta and pulmonary artery. The significance of findings such as increased blood flow, decreased blood flow, large or small aorta or pulmonary artery and chamber enlargement is presented. Thereafter, the physiology of diastolic and systolic overloading of the ventricles and their resultant configurations is addressed. All of these aspects of analyzing the plain film in congenital heart disease are important for they allow one to initially categorize the cardiac problem, and then in some cases arrive at a specific diagnosis.

In summary, the various plain film changes associated with congenital heart disease are addressed with the primary objective being to pathophysiologically explain why they occur.

THE ACUTE ABDOMINAL SERIES IN INFANT AND CHILDREN

J. Fred Johnson III, MD

OUTLINE

Introduction

Objectives of the acute abdominal series

Etiologies of acute abdomen in infants and children

Discrimination of large from small bowel in infants

Acute abdomen in the nursery

Intestinal obstruction during the first three months of life

Ileocolic intussusception

General recommendations for acute abdominal series in infants

Appendicitis

THE ACUTE ABDOMINAL SERIES IN INFANT AND CHILDREN

Introduction

Recently we have noticed that the proliferation of imaging modalities has produced a decreased reliance on plain films and, along with this, decreased skill in interpreting plain films. Yet plain films precede other imaging studies. Therefore, ideally other imaging studies should be regarded as a direct extension of a careful performance and analysis of the plain films.

In a baby with a medical versus surgical abdomen, clinicians frequently assume that the plain films are only valuable for detecting free air or free fluid. We believe that the plain films can also be utilized to prevent inappropriate diagnostic and therapeutic measures, and to help direct additional imaging procedures. In fact, when appropriate views are taken, the plain films are often diagnostic. With this objective in mind, this article will illustrate some tricks that we have learned which maximize the information available from the acute abdominal series.

THE ACUTE ABDOMINAL SERIES IN INFANT AND CHILDREN

Objectives of the Acute Abdominal Series

The primary objective of the acute abdominal series is to distinguish a medical from a surgical abdomen. Traditionally, in this situation, supine and upright films of the abdomen are performed. However, in infants and young children, these views can be insensitive and are sometimes frankly misleading.

As a result of this problem, we have recommended an alternative approach at our hospital over the past several years. This approach involves the addition of a prone film in an effort to distribute gas into the ascending and descending colon [1]. This redistribution of gas helps to accomplish four objectives: (1) it improves discrimination of small from large bowel; (2) it depicts bowel obstruction more accurately; and, in the newborn, (3) it evaluates the extent and cause of intestinal mottling, and (4) the significance of focally distended bowel.

A free air view, if one is necessary, can be a supine crosstable lateral view or a left lateral decubitus film, depending upon which of these views is also most useful for evaluating the diagnosis under consideration. Unless the patient is thought to have a midgut volvulus, the supine crosstable lateral view usually provides the most additional diagnostic information during infancy. After age three, a left lateral decubitus view improves detection of appendiceal perforation.

Etiologies of Acute Abdomen in Infants and Children

The purpose of this chapter is to show the application of this technique to the various causes of acute abdomen at different ages (Table I). For example, the newborn nursery produces a variety of conditions that may lead to surgery. This approach is designed to differentiate this group of conditions from conditions that are always managed medically and to direct attention to a specific etiology of acute abdomen when the clinical findings are unclear.

After discharge from the nursery, but during the first three months of life, the differential diagnosis of bowel obstruction by and large narrows to inguinal hernia, Hirschsprung disease, and midgut volvulus. Simple inspection of the baby narrows the differential diagnosis even more. Inguinal hernia is ordinarily visible. Babies with Hirschsprung disease are usually distended. Conversely, an uncomplicated midgut volvulus associated with a high small bowel obstruction produces a normal abdominal girth or even a scaphoid abdomen. There are, however, some exceptions to these observations and pitfalls in diagnosis that can be minimized by using this radiographic approach.

At about three months of age, the proliferation and enlargement of lymphoid tissue in the terminal ileum begins to provoke ileocolic intussusception, and between six months and two years of age, ileocolic intussusception becomes the most common cause of small bowel obstruction. Therefore, unless there is information to the contrary, the acute abdominal series should evaluate that possibility in that age group.

After the age of two, appendicitis increases in frequency and usually by age three appendiceal perforation replaces ileocolic intussusception as the most common cause of acute small bowel obstruction in a child who has not had previous surgery.

TABLE I

Age	Common Causes of Acute Abdomen In Infants and Children
Nursery	Intestinal atresia, Hirschsprung disease, meconium plug/small left colon, meconium ileus, necrotizing enterocolitis, midgut volvulus
First 3 mo.	Inguinal hernia, Hirschsprung disease, midgut volvulus
6 mo. - 2 yrs.	Ileocolic intussusception
Childhood	Appendicitis

Discrimination of Large from Small Bowel in Infants

SMALL BOWEL OBSTRUCTION VS. DISTENDED TRANSVERSE COLON

By delaying consideration of the correct diagnosis, an erroneous plain film diagnosis of bowel obstruction during infancy can produce unnecessary morbidity, anxiety and expense, and, when the clinical findings are also misleading, may contribute to provoking an unnecessary laparotomy. Therefore, in an infant, one of the main functions of supplementing a supine with a prone film is to avoid overcalling the diagnosis of small bowel obstruction when the clinical findings are confusing enough to require an acute abdominal series [2,3].

On supine and upright films during infancy, the gas-filled transverse colon becomes tortuous and occupies a position that is also appropriate for the small bowel. Since the transverse colon is also elevated in these positions, it tends to trap and accumulate air and may selectively distend to the point that it simulates the appearance of acute small bowel obstruction [3]. This erroneous impression of small bowel obstruction is particularly misleading when the plain film identifies a predisposition for obstruction and the distended transverse colon simulates the morphology as well as the position of small bowel.

Prone filming is ideally suited for eliminating this erroneous impression of small bowel obstruction. By compressing the transverse colon against the spine and elevating the ascending and descending colon, prone filming tends to express gas into the right and left colon, so that the colonic position of the gas is more clearly depicted. [1-3].

INTESTINAL ATRESIA

The diagnosis of bowel atresia usually becomes apparent shortly after birth. However, a supplemental prone film may eliminate alternative considerations immediately and also produce additional information.

The dilated loop of bowel just proximal to a high grade longstanding obstruction is known as a bulbous bowel segment sign [6]. This sign is produced by prolonged impaction of intestinal contents just proximal to the obstruction. This segment has considerable clinical importance. Since this segment has been acclimated to high intraluminal pressures, it will often not peristalse properly after the pressure is reduced to normal. Therefore, in order to assure appropriate bowel function after surgery, this segment has to either be completely resected [4] or reconstructed [5] at surgery.

Notably, however, the bulbous bowel segment is oriented in the abdomen so that it does not distend equally with air in the supine and the prone positions. Therefore, in order to maximize the odds of demonstrating the presence and extent of this segment, both views are necessary.

Once the bulbous bowel segment is correctly identified, it can be traced distally to identify the site, cause, and level of obstruction. A curvilinear termination of the bulbous bowel segment depicts the site of obstruction and indicates a diagnosis of bowel atresia. The level of atresia is estimated from the number of distended loops proximal to the bulbous bowel segment.

NECROTIZING ENTEROCOLITIS
Intramural Air vs. Collapsed Bowel

In the premature baby, the two most common causes of pathological intestinal distention include necrotizing enterocolitis (NEC) and the meconium plug and/or small left colon syndromes [6]. Usually the distinction between these two possibilities is clinically obvious. For example, if the history is carefully examined, a baby with meconium plug or small left colon syndrome is almost invariably symptomatic within the first 24 hours of life. On the other hand, premature babies with the sporadic form of necrotizing enterocolitis rarely present before day three of life. However, sometimes the clinical distinction between these two categories is not obvious. In this situation it is very important for the plain film to discriminate between these two possibilities before anything else is done. A meconium plug is often diagnosed and removed with a contrast enema. Conversely, the therapy of NEC includes bowel decompression. Distention of marginally viable bowel with contrast material is potentially counterproductive.

Sometimes the supine film may not distinguish between these conditions. Both NEC and meconium plug tend to present with bowel distention. Furthermore, babies with meconium plug may have "bowel mottling" indistinguishable from the appearance produced by submucosal air. Remember that in the supine and upright positions, the elevated tortuous transverse colon tends to trap and accumulate air and may distend to the point that it occupies the entire upper part of the abdomen. conversely, the ascending and descending colon tend to collapse in these positions. Consequently, collapsed ascending or descending colon applied to the lateral surfaces of distended transverse colon can produce "mottling" that is indistinguishable from submucosal air.

If such a baby really has intramural air surrounding the right or left colon, distention of these areas by prone positioning should make the position of the intramural air even more obvious on the basis of increased interfaces with air in the lumen [6]. Conversely, if distention of the bowel

lumen eliminates the appearance of intramural air in the flanks, the impression of intramural air on the supine film is artifactually produced by application of collapsed descending (or ascending) colon against the margins of distended transverse colon [6].

When the descending colon contains intraluminal material, collapse of bowel wall around the intraluminal material in the supine or upright position can produce a "pseudobezoar effect" which simulates the appearance of subserosal air [6]. Distention of the descending colon by prone filming identifies the intraluminal material and eliminates the erroneous impression of subserosal air.

FOCALLY DISTENDED BOWEL
Fixed Bowel vs. Positional Air Trapping

In a baby known to have necrotizing enterocolitis, the supplemental prone film helps to establish the cause of focally distended bowel [3]. In the supine or upright position, focally distended bowel can represent nothing more than positional air trapping in the transverse colon. Similar positional air trapping in the rectosigmoid colon can produce focally distended bowel in the central and lower abdomen in the supine and upright positions [3].

On the other hand, focally distended bowel in a baby with necrotizing enterocolitis can also represent "fixed" bowel [7,8] that has been devitalized to the point that it no longer peristalses. The significance of fixed bowel is much more substantial than positional air trapping in that fixed bowel is a precursor of perforation [7,8]. In fact, at least four out of seven babies with fixed bowel will perforate within 24 to 48 hours [8]. Since perforation can be silent clinically, such a baby has to be followed with plain films every four to six ours until the bowel either perforates or regains compliance. Therefore distinguishing between fixed bowel and positional air trapping is very important clinically [3].

Positional air trapping in the transverse or rectosigmoid colon is characterized by tightly folded loops [3]. Conversely, fixed loops are so

stiff that they unwind and develop arcuate parallel walls. As the involvement becomes extensive, fixed loops tend to arrange themselves into rigid concentric coils which are strikingly different from the tightly folded appearance of bowel loops that are distended by elevation. Furthermore, in contrast to bowel loops that are distended because of elevation, fixed loops are so uncompliant that they do not empty in the prone position [3].

NECROTIZING ENTEROCOLITIS VS. MECONIUM ILEUS
Value of the Prone Abdominal Radiograph

Mottling in the right flank of a newborn can represent either necrotizing enterocolitis or impacted meconium. Usually the distinction between these two possibilities is clinically obvious. For example, meconium ileus does not selectively involve premature babies and, when the history is carefully reviewed, usually presents with signs of intestinal obstruction within the first 24 hours of life. On the other hand, the sporadic form of necrotizing enterocolitis usually affects premature babies and normally does not present before the third day of life [6].

Sometimes, however, the clinical findings and supine films do not distinguish between these two conditions. Since a contrast enema is indicated for meconium ileus but is potentially counterproductive in necrotizing enterocolitis, the distinction between these conditions by plain film becomes important when clinical findings are inconclusive.

In contrast to babies with NEC, babies with meconium ileus almost always have a microcolon. Therefore, distention of the descending and rectosigmoid colon by prone positioning supports a diagnosis of NEC. Furthermore, distention of this area as well as the ascending colon by prone positioning makes the position of the surrounding intramural air more obvious by increasing interfaces with air in the lumen. In the supine or upright positions, it may not be clear whether crevices of gas in the flanks represent intramural gas or collapsed lumen applied to the surfaces o distended transverse colon.

SUMMARY:
Three Indications for a Prone Abdominal Radiography in Babies with Possible NEC

In patients filmed for necrotizing enterocolitis, the supplemental prone film reduces the false negatives as well as the false positives for intramural air [6]. Furthermore, the prone film helps to distinguish between positional air trapping in the transverse or rectosigmoid colon and fixed bowel. Finally, the supplemental prone film is also very helpful for distinguishing between necrotizing enterocolitis and meconium plug or meconium ileus when the clinical findings and supine film are inconclusive [6].

NECROTIZING ENTEROCOLITIS VS. MECONIUM ILEUS
Value of the Supine Crosstable Lateral Radiograph

Sometimes the intestinal mottling associated with necrotizing enterocolitis or meconium ileus is obscured on supine or prone films by superimposed soft tissue of the abdomen and back. In newborn babies who may have one of these conditions, using the supine crosstable lateral for the free air view [9] may also detect the existence, extent, and cause of intestinal mottling not seen on supine or prone films.

In patients with meconium ileus, supine crosstable lateral positioning causes bowel gas to converge at the anterior part of the abdomen so that a window is provided for visualization of abnormal meconium obscured by superimposed soft tissue in other positions. Unlike the diffuse poorly marginated mottling that is seen in patients with necrotizing enterocolitis, the abnormal meconium seen in patients with meconium ileus has margins and often produces an aggregate mass or masses on the supine crosstable lateral film.

The contrast enema explains the utility of the supine crosstable lateral plain film for diagnosing meconium ileus. The dilated terminal ileum, which contains the abnormal meconium, occupies the anterior

portion of the abdomen. Therefore, the meconium is best profiled by gas on the supine crosstable lateral plain film.

The supine crosstable lateral film also helps to diagnose NEC as a cause of mottling. By displaying more bowel loops in cross-section, the supine crosstable lateral is the most sensitive view for distinguishing the "target sign" produced by intramural gas from intraluminal mottling. Furthermore, this position improves depiction of the left lobe of the liver, which is the most common location of the portal venous gas associated with necrotizing enterocolitis [10].

SUMMARY:
Recommendations for Distinguishing NEC
from Meconium Ileus

Prone positioning may help to distinguish necrotizing enterocolitis from meconium ileus by distending the ascending, descending, and rectosigmoid colon so that a microcolon is excluded and intramural air in those areas becomes more obvious on the basis of increased interfaces with intraluminal gas.

The supine crosstable lateral view should be used as a free air view when the existence of mottling or the issue of meconium ileus vs. NEC remains unresolved. This is the most sensitive view for defining the existence and the extent of intestinal mottling and for distinguishing the marginated masses produced by meconium ileus from the diffuse poorly marginated mottling, "target sign", and portal venous gas associated with necrotizing enterocolitis.

Intestinal Obstruction During the First Three Months of Life

During the first three months following discharge from the nursery, the three major causes of intestinal obstruction (below the pyloric canal) include inguinal hernia, Hirschsprung disease and midgut volvulus. Simple inspection of the baby narrows the differential diagnosis. Inguinal hernia

is usually visible. Babies with Hirschprung disease are ordinarily distended. Conversely, an uncomplicated midgut volvulus associated with a high small bowel obstruction usually produces a normal abdominal girth or even a scaphoid abdomen. There are, however, some pitfalls in the clinical diagnosis and exceptions to these observations which can be minimized by utilizing the appropriate plain films.

MIDGUT VOLVULUS

During the first three months of life, bilious vomiting associated with a normal inguinal canal and a normal or decreased abdominal girth usually represents either midgut volvulus or medical disease. Radiographically, findings can be similar on flat and upright films. For example, in the supine position, both conditions can present with distended bowel in the left upper quadrant relative to a deficiency or absence of bowel gas elsewhere. However, small bowel obstruction can be differentiated from positional air trapping in the transverse colon by a failure of the left upper quadrant gas to establish continuity with the descending colon in the prone position. (Also see sections on Intussusception and Appendicitis.)

A left lateral decubitus film as a free air view in such babies may also suggest the correct diagnosis. Babies with midgut volvulus and/or bands will sometimes demonstrate an acute obstruction of the duodenum in this position. By elevating and isolating the duodenal C-loop so that it distends with gas, the left lateral decubitus view may reveal the selective distention of the duodenum associated with obstructing bands and/or midgut volvulus.

MIDGUT VOLVULUS VS. PYLORIC STENOSIS

Since the peak incidence of midgut volvulus and pyloric stenosis are in the same age group and pyloric stenosis is more common, midgut volvulus is occasionally mistaken for pyloric stenosis by clinicians. Furthermore, a supine plain film of the abdomen in a patient who really has a midgut volvulus may support an erroneous impression of gastric

outlet obstruction secondary to pyloric stenosis. This mistake occurs when the obstructed duodenum is filled with fluid.

Since these pitfalls interfere with the need to diagnose and treat a midgut volvulus quickly, whenever possible, we have the policy of asking the parents whether such a baby has bilious vomiting. The baby without bilious vomiting continuous to be handled as a possible pyloric stenosis. That is, usually the baby is returned to the ward or emergency room for gastric decompression (so that the pyloric olive is more easily palpated) and parenteral hydration; and then, if clinical findings remain inconclusive, the baby is returned to the radiology department for confirmation of pyloric stenosis with ultrasound or a positive contrast upper GI series. Since this type of delay in a baby who really has midgut volvulus can lead to bowel gangrene, if there is any question about the possibility of bilious vomiting, we obtain a left lateral decubitus film (sometimes after injecting 5 to 7 cc/kilo of air into the stomach) in order to determine whether the obstruction is really at the level of the duodenum. If the left lateral decubitus view demonstrates that the obstruction is actually at the level of the duodenum and does not show any evidence of free air, the baby is retained in the department for a positive contrast study of the upper gastrointestinal tract to exclude midgut volvulus.

THE GAS FILLED VOLVULUS

Distention of a volvulus with gas is often a reflection of the fact that the mesenteric veins are obstructed to the point that they can no longer absorb the intraluminal gas. Therefore, although such a patient will often require a positive contrast study to define the precise anatomy, it is helpful to depict the volvulus on the plain films so that preparations can be underway for surgery.

The prone and/or left lateral decubitus films may show that a collection of bowel loops distal to the obstructed duodenum remains isolated and entrapped. These characteristics of isolation and entrapment are features of closed loop obstruction. Therefore, in a baby with acute

obstruction of the duodenum, these additional radiographic findings should suggest a gas filled volvulus.

Sometimes a gas filled volvulus may produce a round dilated loop of bowel in the central portion of the abdomen on a supine film [3]. Using an upright lateral view to evaluate for free air may also prove that a round dilated loop is a midgut volvulus. By profiling the gas filled bowel against a fluid filled stomach, an upright lateral view may show the pedicle of the twisted bowel when the round loop on the supine radiograph represents a volvulus.

THE FLUID FILLED VOLVULUS

When mesenteric veins are patent, gas is absorbed from the round dilated loop of bowel. In this setting, the retained fluid may produce a round water density pseudotumor. The combination of mass effect and bowel distention should suggest intussusception or volvulus. With intussusception, the patient is usually over three months of age, and the mass is intraluminal. With volvulus, the patient is usually under one month of age and the mass is extraluminal.

SUMMARY
Recommended Plain Films for Possible Midgut Volvulus

In summary, when the clinical findings suggest midgut volvulus, a supine film is usually evaluated initially. If the supine film demonstrates dilated bowel in the left upper quadrant, prone filming prevents overcalling the diagnosis of obstruction in a baby who really has positional air trapping in the transverse colon. On the other hand, when the gas pattern is altered but nonspecific in the supine position, the improved discrimination of the colon and small bowel in the prone position may also facilitate detection of a subtle small bowel obstruction.

The left lateral decubitus view is usually the ideal free air view in this situation because of the ability of this view to also detect a subtle

obstruction of the duodenum in some patients with midgut volvulus. However, if a baby with a provisional diagnosis of midgut volvulus has a round dilated loop of bowel on the supine film, an upright lateral view as a free air view has the potential of showing the pedicle where the bowel is twisted.

Significantly, although plain films may help to rule in midgut volvulus, it is not possible to rule out midgut volvulus by plain film alone. Sometimes, babies with midgut volvulus have a normal gas pattern. A midgut volvulus complicated by gangrene, a distal small bowel volvulus, or a midgut volvulus provoked by a second more distal bowel obstruction (such as Hirschsprung disease) may suggest a distal bowel obstruction on plain films. Clinical findings, positive contrast studies, and occasionally other imaging modalities help to conclusively rule out midgut volvulus in such a patient.

HIRSCHSPRUNG DISEASE

Hirschsprung disease generally presents at two points in time. Some patients with Hirschsprung disease present late in infancy or in early childhood. Usually the diagnosis of the later presentations of Hirschsprung disease is not difficult. These patients generally have an unrelenting history of constipation. Furthermore, unless the patient has ultrashort segment Hirschsprung disease (a controversial diagnosis), obvious bowel tapering (the transition zone) is apparent on the barium enema at the level of the aganglionosis.

On the other hand, most patients with Hirschsprung disease present during the neonatal period. If these babies are not detected on the basis of a failure to pass meconium at birth, the diagnosis can be very elusive. The major obstacle to a correct diagnosis relates to the fact that there are often conflicts between the clinical findings, the plain films and the barium enema. Furthermore, in this situation there is a tendency to subordinate the information provided by the plain films to these other two parameters [11].

It is important to recognize that the clinical findings in babies with Hirschsprung disease are often not as unrelenting as they are in older patients [11]. The baby with Hirschsprung disease can unpredictably go into a remission. Furthermore, the barium enema in this age group is normal on inconclusive about 20% of the time. Therefore, in early infancy, the plain films may produce the most useful information.

The axiom to remember is that recurrent or persistent, unexplained colonic distention should be regarded as presumptive evidence of Hirschsprung disease. Furthermore, since colonic distention is the factor that is most closely associated with the development of a potentially fatal Hirschsprung enterocolitis, it is very important to diagnose or exclude Hirschsprung disease quickly by biopsy in this setting [11]. Hirschsprung enterocolitis, even under the best of circumstances, has as high as a 50% mortality rate. Once that condition develops, the patient can deteriorate so suddenly and quickly that it is impossible to salvage the situation.

Although unexplained colonic distention is an important sign of possible Hirschsprung disease, it is equally important to remember the converse axiom that the bowel distention proximal to a high grade obstruction is not always striking. In fact, the bowel gas pattern proximal to an obstruction may be relatively deficient because of vomiting (see the section on Intussusception). In such cases, however, the bowel occasionally distends focally just proximal to the obstruction. Although this focal distention may be acute, it often represents the bulbous bowel segment sign [4]. In babies with Hirschsprung disease, the natural tortuosity of the transverse colon predisposes to the development of this sign when the transition zone is at the splenic flexure.

SUMMARY
Recommended Plain Films for Possible Hirschsprung Disease

In patients with distal aganglionosis, the supine film will often show a dilated transverse colon with unusual clarity. The prone film is helpful for localizing distended bowel and establishing the existence and position of a transition zone. When marked intestinal distention obscures the rectum in the supine and prone positions or when the clinical and radiographic findings are otherwise inconclusive, a prone crosstable lateral view of the rectum to profile the caliber and configuration of the rectum relative to the proximal bowel may show a transition zone that is not apparent in other views.

DIFFERENTIAL DIAGNOSIS OF FOCALLY DISTENDED BOWEL

In the newborn and young infant, there are four causes of focally distended bowel on the supine film which can often be differentiated by prone positioning. When the supine position produces focal distention of the transverse or sigmoid colon, prone positioning expresses the gas proximally and distally so that the colonic position and normal compliance of the dilated loops are depicted more clearly.

Focally distended bowel can also be produced by uncomplicated obstruction. If the distended gas collection is colon, prone positioning will express the gas into a more proximal position so that the colonic position of the gas is more clearly depicted. However, gas will either not extend distal to the obstruction at all or it will extend distally in reduced amounts.

Finally, focally distended bowel can be produced by intestinal ischemia (the so-called "fixed bowel sign") [7,8] or closed loop obstruction. Under those circumstances, the bowel gas will remain collected in the same loops in the prone position. When bowel is "fixed" because of ischemia, it also stiffens into a tubular configuration, develops arcuate parallel walls, and as involvement extends, the loops tend to arrange themselves into rigid concentric coils.

INGUINAL HERNIA

Although inguinal hernia is the most common cause of small bowel obstruction in the first three months of life, it is usually a clinical diagnosis. However, occasionally the hernia is not apparent clinically. Therefore, it is very important to carefully scrutinize the inguinal region for soft tissue thickening or a loculus of air [12].

If a baby with an inguinal hernia is shielded, the inguinal canal may not be visible. Under these circumstances the plain film may provide another sign of inguinal hernia. Sometimes a baby with inguinal hernia will have focally distended bowel just above the hernia. This focally distended bowel may deviate in orientation from the rest of the bowel, and then point toward the inguinal canal. When the proximal bowel becomes distended because of increasing obstruction, the selective distention of the segment of bowel just proximal to the hernia is no longer apparent. However, it is still possible, in some patients, to detect a loop of bowel in the lower abdomen or pelvis that deviates in orientation from the rest of the bowel and then turns to point toward the inguinal canal [3].

Ileocolic Intussusception

INTRODUCTION

Ileocolic intussusception is the most common cause of acute small bowel obstruction between the ages of six months and two years. Unfortunately, gastrointestinal infection often simulates ileocolic intussusception clinically. Therefore in this age group, the acute abdominal series should evaluate the relative likelihood of infectious enterocolitis versus ileocolic intussusception. The addition of the supine crosstable lateral and prone films helps to distinguish intussusception from infection as a cause of similar symptoms.

UTILIZATION OF PLAIN FILMS TO "RULE OUT" INTUSSUSCEPTION

By localizing bowel distention to large bowel, the prone film may help to rule out small bowel obstruction when the supine and upright films seem to actually suggest that diagnosis. Remember, in the supine infant, distended loops of transverse colon occupying central abdomen can simulate small bowel obstruction. Adding to the confusion, centralization of these loops and development of differential air fluid levels on upright films may seem to support this erroneous impression of small bowel obstruction in a baby who really has a distended colon secondary to infection.

The prone film helps to avoid these pitfalls by improving discrimination of distended colon from small bowel in infants. Furthermore, the prone film helps to distinguish between the three causes of right lower quadrant gas collections - cecal pole, rectosigmoid colon and terminal ileum - by segregating these areas when they are superimposed in other positions.

These advantages of the prone film facilitate identification of three landmarks that exclude ileocolic intussusception: a normal cecal pole, gas in the terminal ileum, and ileocecal valve. Since an advancing ileocolic intussusception displaces gas ahead of it and out of these areas [13], clear cut identification of one or more of these landmarks excludes ileocolic intussusception.

In normal infants, these landmarks are not well seen, even on prone films. However, early bowel infection mimicking intussusception usually distends the colon so that one or more of these landmarks can be detected in the prone position.

One word of caution, however. In patients with ileocolic intussusception, the small bowel may dislocate into the right lower quadrant and occupy a position appropriate for the cecal pole and ascending colon [13]. Therefore, a loop of bowel occupying this position

does not necessarily represent ascending colon unless it can be clearly traced beyond the hepatic flexture without any areas of discontinuity produced by soft tissues or unless there is a continuous column of feces between these areas.

UTILIZATION OF PLAIN FILMS TO "RULE IN" INTUSSUSCEPTION

Another function of the acute abdominal series in patients with possible intussusception is to demonstrate the intussusception or the associated small bowel obstruction. Some might say that this role is not critical, because the baby will undergo a diagnostic enema if the clinical findings suggest one is needed. However, the fact of the matter is that the symptoms associated with intussusception are often episodic. The baby may have classical findings at home which disappear in the pediatrician's office, so that the pediatrician is merely "going through the motions" when ordering the films and would not necessarily be motivated to order a contrast enema if the films are inconclusive. Therefore, if there is any question from the history about the possibility of intussusception, the acute abdominal series should be designed to pick up the intussusception or the associated small bowel obstruction.

DETECTION OF ASSOCIATED SMALL BOWEL OBSTRUCTION

By improving discrimination of small and large bowel, prone positioning improves detection of a subtle small bowel obstruction in the child with intussusception. With early small bowel obstruction, the supine film may demonstrate several poorly localized normal caliber loops of bowel distributed throughout an otherwise gasless abdomen. In the prone position, small bowel loops are recognized by a reorientation into a position and configuration more typical of small bowel and a failure to establish contact with the colon [15]. Distention of such loops out of proportion to the cecum and ascending colon on the prone film is an indication of an early small bowel obstruction.

Prone filming is also helpful when conventional flat and upright films fail to determine the source and significance of an isolated left upper quadrant bowel loop [15]. In these views, it may not be clear whether a left upper quadrant bowel loop is transverse colon or dilated small bowel. In the prone position, a left upper quadrant gas collection is identified as small bowel on the basis of the fact that it moves medially instead of establishing continuity with the descending colon. The distention of such a loop proximal to a gasless cecal pole on the prone film favors a diagnosis of obstruction secondary to ileocolic intussusception over gastroenteritis.

DIRECT VISUALIZATION OF THE INTUSSUSCEPTION

It has been known for some time that the supine, prone, and upright films are valuable for depicting an intussusception, particularly when it is in the transverse colon [13,14]. The intussusception tends to become snared as it negotiates the turn at the hepatic flexture so that it produces an intraluminal soft tissue mass in these views which sometimes obscures the fat under the liver shadow. In fact, sometimes the mesenteric fat trapped between the telescoped loops produces curvilinear lucencies within the mass when the intussusception turns into a position perpendicular to the radiographs [15,16].

Once the intussusception enters an air filled transverse colon, a "crescent sign" is produced by protrusion of the soft tissue mass into the intraluminal gas [15,16]. Further progression of the intussusception through the transverse colon creates a soft tissue mass which separates distended small bowel from the greater curvature of the stomach.

We have noticed, however, that the supine crosstable lateral film is the most effective view for diagnosing an intussusception [17]. Whereas the right and left lateral decubitus views profile an intussusception in the left and right colon respectively, and the supine and upright positions are designed for depicting the intussusception in the transverse colon, the supine crosstable lateral view tends to surround the intussusception with

gas no matter where it is so that it is clearly seen approximately 75% of the time [17].

The exact appearance of the intussusception depends on its location. As ascending colon intussusception may produce a homogenous water density mass which protrudes into the bowel gas converging in the anterior aspect of the abdomen. The sharp anteriorly convex margin of the mass represents the advancing margin of the intussusceptum. The mechanism of this effect is analogous to the depiction of meconium ileus. The supine crosstable lateral position removes the intussusception from the relative obscurity produced by superimposition on the soft tissues of the abdomen and back and profiles it with gas.

The more distal intussusceptions may produce a different appearance. Normally on a supine crosstable lateral film there is no soft tissue in the anterior abdomen caudal to the liver shadow because bowel loops that are distended with gas rise into this area and directly approximate adjacent loops. In patients with intussusceptions at or distal to the hepatic flexture, these anterior bowel loops may be separated in the craniocaudal direction by soft tissue [17]. This soft tissue represents the gasless telescoped bowel (intussusception), interposed between the site of initiation of the intussusception and the advancing edge of the intussusceptum. In other words, by virtue of its position and mass effect, the portion of the intussusception within the transverse colon tends to block convergence and direct approximation of gas filled bowel loops in the anterior abdomen.

In our experience, when bowel loops are adequately distended with gas, it is extremely unusual to see either of these signs unless the patient has an intussusception. In fact, occasionally, both signs are seen simultaneously on the supine crosstable lateral film.

SUMMARY
Recommended Plain Films for Possible Intussusception

In a baby with a provisional diagnosis of ileocolic intussusception, the supine and prone films are very helpful for detecting an intussusception, particularly when it is trapped at the hepatic flexture or in the transverse colon. The prone film serves two additional purposes. The potential of the prone film to discriminate large and small bowel is very helpful for detecting the subtle small bowel obstruction associated with an ileocolic intussusception. Conversely, the prone film is also helpful for segregating the distended cecum, terminal ileum, and rectosigmoid colon in patients with infection mimicking ileocolic intussusception. Clear cut depiction of the cecal pole, ileocecal valve or terminal ileum excludes an ileocolic intussusception. The supine crosstable lateral film is an ideal free air view in this situation because of the potential of this view to also detect the intussusception itself.

General Recommendations for Acute Abdominal Series in Infants
SUPINE, PRONE, AND SUPINE CROSSTABLE LATERAL OR LEFT LATERAL DECUBITUS FILMS

The material shown in this section demonstrates that the supine film provides diagnostic information in many infants with the differential diagnosis of medical versus surgical abdomen. The prone film also has several important functions in a baby with medical versus surgical abdomen. Since prone filming improves discrimination of large and small bowel, a prone film reduces the false positives and negatives for small bowel obstruction. Additionally, by distributing bowel gas more uniformly throughout all portions of the large and small bowel, sequential supine and prone films have the potential of reducing the false positives and negatives for intramural air. Furthermore, sequential supine and prone films can help determine whether focally distended bowel is produced by obstruction, ischemia, or positional air trapping in the transverse of rectosigmoid colon.

We would suggest that the upright film should be discarded as the free air view in infants and children. Although this view detects free air, it can produce very misleading bowel gas patterns. Generally, under the age of two, a supine crosstable lateral film is a very sensitive free air view which also has the potential of providing additional positive diagnostic information. In the newborn, the supine crosstable lateral film is the most sensitive view for determining the existence, extent, and cause of intestinal mottling (impacted meconium versus intramural air). In the six month to two year age group, it is the most sensitive view for depicting an ileocolic intussusception.

In babies with a possible midgut volvulus, a left lateral decubitus film is usually preferable to a supine crosstable lateral film since it may demonstrate an acute obstruction of the duodenum as well as free air. A chest x-ray is also often included as a part of an acute abdominal series because of the potential for acute pneumonia to mimic many of the conditions discussed here clinically.

Appendicitis

RELATIVE VALUE OF CLINICAL FINDINGS VS. PLAIN FILMS

After the age of two, appendicitis begins to increase in frequency and by age three appendicitis becomes the most common cause of acute abdomen in children. The diagnosis of appendicitis is simplified by recognizing the reciprocal roles of clinical findings and plane films. The relative value of clinical findings and plain films for making this diagnosis depends on whether the appendix has perforated. The most diagnostic finding for unperforated appendicitis is McBurney sign or pain and tenderness over the inflamed appendix [18-24]. This sign is seen up to 90% of the time and is an indication for surgery. Unperforated appendicitis also provokes varying degrees of anorexia, nausea, vomiting and diarrhea [18]. These clinical findings produce a gasless abdomen which usually does not change the position of appendicitis in the differential diagnosis [18,20,25]. The only plain film finding of diagnostic value prior to perforation is a calcified appendicolith which, at the most, is

seen about 20% of the time [19,21,22]. Therefore, before perforation, the clinical findings are usually more diagnostic than the plain films in children with appendicitis [20-25].

After perforation, this relationship between clinical findings and plain films often reverses. McBurney sign tends to disappear after the pressure within the appendiceal lumen is relieved by perforation. In fact, the clinical findings may subside or may simulate a number of alternative diagnostic possibilities depending on where the pus spreads [21,22,26].

However, the plain film findings become more distinctive after perforation. Plain films taken in a variety of positions are about 80% sensitive and 94% specific for detecting perforation in children with appendicitis [21,22].

THERAPEUTIC IMPLICATIONS OF CLINICAL FINDINGS AND PLAIN FILMS

Knowledge of the reciprocal role of clinical findings and plain films is also therapeutically important [27,28]. Unperforated appendicitis (usually unremarkable radiographs, but positive clinical findings) is almost always managed with an appendectomy. Evidence of appendiceal perforation (usually nonspecific clinical findings, but positive radiographs) [18-22,29-33], diversifies the management possibilities to three options - antibiotic therapy with delayed appendectomy, percutaneous abscess drainage under CT guidance, and immediate laporatomy - depending on the nature and extent of periappendiceal involvement on a contrast enhanced CT [27,28]. The conservative approaches are more of an option if perforation is recognized quickly.

PLAIN FILM SIGNS OF APPENDICEAL PERFORATION

Small Bowel Obstruction Pattern

The most common finding after appendiceal perforation is a small bowel obstruction pattern. In a child over the age of three who has not had surgery, a small bowel obstruction pattern is most commonly caused by an appendicitis with perforation [20,32]. Notably this finding can

develop within a short period of time without any discernible changes in the clinical findings. In fact, when a patient under observation for appendicitis turns out to have that diagnosis, the nonspecific clinical findings that require observation usually mean that the appendix has perforated so that the radiographic findings become distinctive within a short period of time. For this reason, we would suggest that a repeat set of plain films should precede more elaborate methods for diagnosing or excluding appendicitis in a child under observation for that possibility [22].

Colon Cut-Off Sign

The colon cut-off sign is virtually diagnostic of appendiceal perforation in children. When fully developed this sign has three components: 1) an absence of gas and feces in the right lower quadrant because of spasm in the ascending colon; 2) reflex dilatation of the transverse colon; and 3) an amputation of gas at the hepatic flexure [21,22,30]. The fully developed colon cut-off sign is easily distinguishable from the normal effects of supine and upright positioning. Although flat and upright positions normally induce gas migration from the ascending and descending colon into the transverse colon, the distention of the transverse colon is much less striking than the reflex dilatation seen with the colon cut-off sign. Furthermore, flat and upright positioning cannot produce an absence of feces as well as gas in the right lower quadrant.

The striking reflex dilatation of the transverse colon in some patients with the colon cut-off sign should not distract attention away from an associated small bowel obstruction pattern [19-22,30]. This pitfall can be avoided if the diagnosis of small bowel obstruction is based on a comparison of the caliber of the small bowel to the caliber of the cecum and ascending colon.

When the colon cut-off sign was first described, it was assumed that reflex dilatation of the transverse colon always coexisted with the spasm of the ascending colon [30]. In our experience, reflex dilatation of the transverse colon only occurs about 50% of the time. In this setting,

particularly when the colon is totally devoid of feces, it may be difficult to distinguish spasm of the ascending colon from the gravitational collapse that occurs in the flat and upright positions. For this reason, we have occasionally made a diagnosis of the colon cut-off on barium enema examination simply because it is obscured on the flat and upright films [31].

However, a barium enema is not an ideal method for diagnosing the colon cut-of sign. The barium enema may obliterate this finding when it is obvious on the plain film. Furthermore, it is sometimes difficult to tell whether a small gas collection in the right lower quadrant is in a spastic lumen or is extra luminal. Inability to make this distinction risks introduction of barium into the peritoneum [21,22].

It is therefore best to diagnose the colon cut-off sign as well as other signs of appendiceal perforation on plain films before doing anything else. Unfortunately, the flat and upright films will often obscure these signs when they are clearly seen on the prone and/or left lateral decubitus films [21,22]. For this reason, our acute abdominal series n a patient with the possibility of appendicitis usually consists of supine, prone and left lateral decubitus films.

EXTRALUMINAL GAS

Free air is uncommon in children with appendicitis, but extraluminal air does occur fairly frequently. In our experience, extraluminal gas tends to occur in one of three forms: 1) gas loculations; 2) bacteriogenic gas [20]; and 3) the triangle with the absent wall.

Gas Loculations

Gas loculations may be large or small and may or may not contain air fluid levels. When they are in the right flank, they tend to occupy a water density mass that displaces ascending colon from the properitoneal fat line. These loculations are usually apparent on conventional flat and upright films.

Bacteriogenic Gas

Bacteriogenic gas has a mottled appearance that resembles feces. Distention of the ascending colon by prone positioning may help to show that the mottling is clearly separate from the bowel.

The Triangle With an Absent Wall

The triangle with the absent wall represents gas that is trapped in the paracolic gutter [21,22]. On the supine film, the triangle occupies the medial portion of a water density mass that displaces distended bowel from the properitoneal fat line. The triangle differs in appearance from adjacent distended bowel in that the margin of the gas not interfacing with bowel wall is unsharp in comparison to similarly distended loops of bowel. That, of course, is because the bowel wall is absent.

In the upright position the gas does not rise to the high point of the abdomen but instead maintains the same position in relationship to he water density mass and adjacent distended bowel. However, the absent wall sign disappears in the upright position. Therefore, the supine or the prone film may be more diagnostic of this form of extraluminal gas than the upright film. Furthermore, because of the tendency for extraluminal gas to migrate laterally and form a broad base on the peritoneum in the prone position [1], the prone film may show this sign more clearly than the supine film.

Notably, when the adjacent ascending colon is poorly distended because of supine positioning, adherent fecal material may contribute to the appearance of an absent wall sign when the gas is actually intraluminal. Distention of the ascending colon and centrifugal migration of intraluminal gas on the prone film establishes a clear cut bowel margin in contrast to the tendency for extraluminal gas to form a broad base on the peritoneum in the prone position [22]. When the ascending colon is distended on the supine film or by prone positioning, we have not seen

any triangles with absent walls that are falsely positive for extraluminal gas [22].

Water Density Mass

The definition of a water density mass is somewhat arbitrary. Retraction of undistended ascending colon from the properitoneal fat line in the supine position may create the appearance of a water density mass that disappears when the ascending colon is distended by prone positioning. In fact, even when the adjacent bowel is distended, there may be some separation of bowel from properitoneal fat line on the supine film that disappears when the patient is placed prone. Therefore, the prone position may reduce the false positives for water density mass. The prone film may also reduce the false negatives for water density mass because of drainage of material from the pelvis into the more dependent paracolic gutters in the prone position.

Because of these pitfalls, it is important not to suggest a water density mass on the supine film unless it is obvious, or unless it is verified by prone positioning. Even with these qualifications, however, a water density mass is a common sign of appendiceal perforation. Furthermore, this sign is almost always seen in association with other signs of perforation [21,22].

Obliteration of Pelvic Fat Planes

Sato et al [33] have suggested that obliteration of the fat around the bladder or the right obturator fat pad may occur prior to appendiceal perforation. In our experience, this sign almost always means that the appendix has perforated. Precise supine positioning is necessary for representing this sign accurately since slight degrees of relation may obscure these fat plains.

SUMMARY

In summary, before perforation, the clinical findings are generally more diagnostic than the plain films in children with appendicitis. After perforation this relationship often reverses. However, the flat and upright films may obscure signs of appendiceal perforation. The prone film reduces the false positives and negatives for small bowel obstruction, the colon cut-off sign, extraluminal air and the fluid density mass. The left lateral decubitus view reduces the false positives and negatives for small bowel obstruction and the colon cut-off sign. Therefore, we would recommend that the acute abdominal series in children with the possibility of appendicitis should consist of the supine, prone and left lateral decubitus films.

TABLE 2

PLAIN FILM SIGNS ASSOCIATED WITH APPENDICEAL PERFORATION

Radiographic Sign	Number & Percentage of Patients with Sign	Frequency of Association With Other Signs of Perforation
Small bowel obstruction	9/21 (43%)	7/9 (78%)
Extraluminal gas	7/21 (33%)	4/7 (60%)
Right lower quadrant pain	5/21 (24%)	5/5 (100%)
Colon cut-off sign	4/21 (20%)	4/4 (100%)
Calcified appendicolith	4/21 20%)	3/4 (75%)
Loss of pelvic fat pads	3/21 (14%)	2/3 (67%)

(Reprinted with permission from: Johnson JF, Coughlin WF, Stark P. The sensitivity of plain films for detecting appendiceal perforation in children. Fortschr Rontgenstr 1988; 149:619-623, and Johnson JF, Coughlin WF. Plain film diagnosis of appendiceal perforation in children. Sem Ultrasound CT MR 1989; 10:306-313.)

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CONGENITAL HEART DISEASE - CURRENT IMAGING

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Current imaging of congenital heart disease still begins with the plain film although seldom are all four views obtained. There is no longer a serious effort to diagnose every condition from the plain films, for almost invariably these patients now are examined with ultrasound. For the most part, this examination is not performed by radiologists. Nonetheless it has become the major screening modality for congenital heart disease. Thereafter cardioangiography is utilized in most cases. It provides both anatomic and physiologic data and has been difficult to replace with fast CT or magnetic resonance imaging. Magnetic resonance imaging is used more for anatomic definition, especially when the patient's condition renders cardioangiography too high a risk or when only anatomic information is required.

In summary, while plain films still provide some information, ultrasonography is utilized in almost all cases and in some conditions no further imaging is required. However, the majority of cardiac lesions still require cardioangiography and a few require magnetic resonance imaging. Examples of some more common conditions demonstrated with the various imaging modalities are presented.

SPINAL CORD TUMORS

Approximately 15 to 20% of central nervous system tumors involve the spinal cord or canal. Primary tumors of the spinal cord and canal are relatively rare, and the true incidence of spine neoplasms is uncertain. Regional population studies suggest that the average annual incidence is probably 1 or 2 per 100,000, with a prevalence rate of approximately 13 per 100,000. These studies do not include primary bone tumors of the spine nor metastatic involvement Large series of spine tumors may be significantly skewed by population variables such as referral patterns, surgical interest, or preferential selection for autopsy series. To further complicate matters, metastatic involvement of the spine is often excluded.

Primary spine tumors occur primarily in young or middle-aged adults and are less frequently encountered in children and the elderly . The sex incidence is roughly equal, although there is a definite female predilection for meningiomas. In general, spine tumors are most frequently found in the thoracic spine, but this is explained largely by the longer length of the thoracic spine relative to the remaining spine . Metastatic involvement of the spine is seen primarily in patients between the ages of 50 and 70 years and is only rarely encountered in either younger or older age groups . The thoracic spine is most frequently involved.

The differential diagnosis of spine neoplasms is primarily based on location of the lesion relative to the spinal cord and the age, sex, and clinical presentation . Tumors within the cord are referred to as intramedullary and account for 11 to 25% of spine tumors . Intramedullary neoplasms are usually associated with fusiform enlargement of the cord with narrowing of the adjacent subarachnoid space.

Tumors between the cord and the dura are referred to as intradural extramedullary and account for nearly 60% of primary spine tumors. The great majority of these are neurofibromas and meningiomas. These tumors are associated with displacement of the spinal cord away from the tumor, with widening of the ipsilateral subarachnoid space and narrowing of the contralateral space.

Extradural tumors account for less than 25% of primary spine neoplasms . However, when metastatic disease is included, extradural tumors may account for over half of all spine neoplasms. When these involve the spinal canal, there may be displacement of the cord with narrowing of the subarachnoid space both ipsilateral and contralateral to the lesion.

As a general rule, intramedullary tumors are more common in children and extramedullary tumors are more common in adults . Mass lesions of the spinal canal usually produce symptoms by involving long tracts of the spinal cord or by involving nerve roots . Pain is a common presentation and may help with localization. Sensory disturbances predominate with intramedullary tumors, motor symptoms with meningiomas, and root symptoms with nerve sheath tumors. Slowly growing tumors

such as meningiomas and neurofibromas may produce a spastic paresis. Rapidly growing tumors such as metastases may result in acute transection syndromes with flaccid paresis and loss of reflexes.

This presentation will review the most common tumors encountered in the spine with a few additional comments about the some of the uncommon tumors. The outline at the end of this section shows the scope of this subject.

Glioma. The great majority of gliomas are either ependymomas or astrocytomas . Imaging characteristics of ependymoma and astrocytoma can be quite. The differential diagnosis between ependymomas and astrocytomas is primarily based on size, location, and patient age.

Ependymomas are the most common intramedullary tumor and account for roughly 13% of all spinal cord tumors. These are much more common in the distal segment of the cord. Nearly 50% involve the cauda equina. Cervical or thoracic ependymomas are generally fusiform shaped, but lesions involving the cauda equina or filum terminale may appear as bulky, extramedullary masses. These may occasionally fill the entire lumbar canal . The classic ependymoma is 4 to 8 cm in length and rarely extends greater than five vertebral segments in length. Unlike intracranial ependymomas, cord ependymomas are extremely rare in children under age 4. Ependymoma may present with subarachnoid hemorrhage and frequently there is evidence of hemorrhage in the tumor. A hemosiderin "cap" is much more likely to be seen in ependymoma than in an astrocytoma.

Astrocytomas are less frequent than ependymomas, accounting for 30% of intramedullary tumors . However, in the pediatric population, astrocytomas are more common than ependymomas . Astrocytomas tend to involve a longer segment of cord than ependymomas and may extend over greater than five vertebral segments . In the pediatric population, a cervical location is more common . The tumor may be associated with holocord enlargement . Although generally solitary, multiple lesions may be present in central neurofibromatosis . There is a small male predominance with the more benign histologic grades (grades 1 and 2), and the sex incidence is equal for the malignant variety.

MRI has largely replaced myelography in the evaluation of intramedullary lesions. MRI demonstrates the contour abnormality of the cord and allows direct visualization of associated cysts and tumor nodules. Intramedullary lesions typically have prolongation of both T1 and T2 values; that is, tumors are hypointense to cord on T1-weighted images and hyperintense on T2-weighted images. However, the T1 values are not always significantly different The signal intensity of the tumor may be relatively isointense to cord on the T1-weighted images.

The signal changes in ependymoma tend to be more inhomogeneous than with astrocytomas. Signal changes associated with some ependymomas may be relatively minimal, and the nodule may only be recognized due to the surrounding cystic fluid which has prolonged T1 and T2 values.

No specific morphologic or signal characteristics reliably distinguish ependymoma from astrocytoma. Both can be solid, cystic with one or more nodules, or of variable length . However, some generalizations can be made. A solid tumor involving almost all the spinal cord with a prolonged T2 relaxation time in a young patient is more likely to be an astrocytoma A less extensive tumor that is inhomogeneous as far as T1 and T2 relaxation times is most likely to be an ependymoma. Paramagnetic contrast material improves the sensitivity and specificity in the evaluation of spinal neoplasms. The majority of intramedullary neoplasms demonstrate enhancement. Enhancement allows more accurate assessment of lesion size and configuration and precise localization of the lesion. Contrast enhancement also may help identify additional lesions and is useful in distinguishing tumor from edema There is a well-known association between intramedullary tumors and spinal cord cavitation. About 1/3 of intramedullary tumors are associated with syringomyelia.. The cavitation associated with intramedullary lesions such as ependymoma may be relatively small, located within the tumor or involving only one or two segments above or below the tumor. These cavities or intratumoral cysts result from transudation from the tumor. However, larger cavities (syringomyelia) may extend well away from the tumor and involve most of the cord. Distinguishing between the intratumoral cysts and associated nonmalignant syringomyelia is important for surgical planning, since the associated syrinx requires only decompression. In patients with an expanded cord, the combination of distinct margins, uniform signal intensity, and isointensity of cyst fluid with cerebrospinal fluid (CSF) is characteristic of hydromyelia or syringohydromyelia . Since intramedullary tumors tend to be infiltrative and associated

Intramedullary Neoplasms

Ependymomas
- Most common intramedullary tumor
- Cauda and distal cord
- 4 to 8 cm length
- Often associated with syringomyelia
- CT: iso- to hypodense, enhance
- MRI: iso- to hypointense on T1, hyperintense on T2

Astrocytomas
- Most common intramedullary tumor in children
- Cervical > thoracic location
- Longer segment involved than ependymomas, may involve whole cord
- Often associated with syringomyelia
- CT and MRI: similar to ependymoma, may be more inhomogeneous

Hemangioblastoma
- 80% solitary
- One-third associated with von Hippel-Lindau disease
- Cervical involvement more common
- One-half associated with syrinx—look for tumor nodule
- Cord enlargement out of proportion to tumor size
- Meningeal varicosities, especially dorsal surface
- CT: marked homogeneous enhancement of hypodense mass
- MRI: hypointense on T1, hyperintense on T2—look for adjacent "flow voids" due to AV shunts

with edema, margins tend to be poorly defined. In addition, findings associated with malignancy, such as necrosis, cystic degeneration, or hemorrhage, lead to an inhomogeneous MRI appearance. Highly proteinaceous fluid within zones of cystic degeneration may produce hyperintensity or heterogeneous signal intensity on MRI. The combination of poorly defined margins, signal inhomogeneity, and lack of isointensity Subarachnoid seeding with intracranial metastases have been reported following surgical resection of malignant cord astrocytomas. Radiation therapy seems to improve prognosis and functional recovery only in the category of patients who have had incomplete surgical resections.

Oligodendrogliomas account for 3 to 4% of intramedullary tumors and tend to present later in life , usually in the fourth or fifth decade. They are clinically indistinguishable from other gliomas. There is considerable variability in vascularity, and loops of vessels may be seen around necrotic areas . Rupture of degenerated vessels may lead to hemorrhage, and areas of calcification are not infrequent Calcification and hemorrhage are best demonstrated on unenhanced CT . On Tl-weighted MRI sequences, hemorrhage in the methemoglobin stage has high signal, whereas older areas of hemorrhage may show marked hypointensity on T2-weighted images due to hemosiderin deposition. MRI is not useful in differentiating these neoplasms from other intraspinal gliomas . Metastases from oligodendroglioma usually

Hemangioblastomas account for 1 to 3% of intramedullary tumors, although the actual incidence may be higher . Symptoms often begin in the fourth decade, but neurologic findings may be minimal and the lesion may never be diagnosed. About 80% are solitary, but tumors may be present at multiple sites when associated with von Hippel— Lindau disease . Although cerebellar hemangioblastomas are common in von Hippel—Lindau disease, the reported incidence of spinal cord involvement can range from 5 to 33%. Cervical involvement is more common than thoracic, but even the cauda equina may be involved. About 60% are intramedullary, but a signiScant number may be both intramedullary and intradural extramedullary. Most are located dorsally in the spinal cana. , Lesions may be cystic, and approximately 50% are associated with syringomyelia 121]. When cystic, a tumor nodule can often be identified. Since these are slowly growing neoplasms, plain films may demonstrate evidence of spinal canal enlargement. Over half the cases will show meningeal varicosities on myelography or angiography. These serpentine vascular structures are usually seen over the dorsal surface of the cord. Contrast enhancement may allow distinction between the vascular tumor nodule and adjacent syrinx. MRI is well suited in the evaluation of hemangioblastomas and may eliminate the need for time-consuming angiography. Multiple spinal lesions or concurrent involvement of the posterior fossa can be detected. These tumors are associated with prolongation of both the T1 and T2 relaxation times (hypointense on T1- and hyperintense on T2weighted images) . Cystic components have higher signal intensity than CSF on the intermediate or T2weighted image. The high-flow arteriovenous shunts are associated with dilated afferent and efferent vessels which may be seen as adjacent

tubular areas of "flow void". Cord enlargement out of proportion to the actual size of the hemangioblastoma is an unexplained phenomenon that appears to be unique to this type of tumor . The cord enlargement extends beyond the associated cyst. Theories include diffuse cord edema secondary to marked venous congestion associated with the prominent arteriovenous shunting, as well as possible production of some edemaproducing factor by the tumor . Since these tumors are highly vascular, intense enhancement of the tumor nodule with paramagnetic contrast material is common. Contrast-enhanced MRI is useful in identifying both the vascular nodule of the tumor and the presence of other lesions along the entire extent of the cord or posterior fossa In the absence of a cystic component with nodular enhancement, differentiation from a high-flow type of spinal vascular anomaly may be difficult.

Metastases To Spinal Cord. Intramedullary spinal cord metastases are rare, with less than 5% of central nervous system (CNS) metastases involving the cord. Intramedullary metastases are usually associated with epidural metastases. Solitary intramedullary involvement is quite rare. Intramedullary metastases usually result from leptomeningeal spread of primary CNS lesions or hematogenous spread of other primaries. Fifty percent of intramedullary cord metastases are from bronchogenic carcinoma. Breast cancer is the next most frequent primary neoplasm, accounting for 15%. Others include lymphoma, colorectal, head and neck, renal cell, and melanoma Pain and weakness were the most frequent presenting complaints, and neurologic examination revealed weakness or paralysis in all cases. In 34% of cases, neurologic symptoms were the initial manifestation of systemic malignancy . Plain films are usually normal due to the rapid growth of these lesions. Since clinically significant cord lesions may be quite small with limited edema, nearly half the myelograms may be normal . Nonspecific fusiform cord enlargement may be seen.

CT reports are limited, but hypo- or hyperdense enhancing masses have been reported. Postsmyelography CT may show cord enlargement but is of limited value in smaller lesions with little enhancement. Diffusion of intrathecal contrast material into the lesion may result in enhancement either acutely or delayed up to 20 hours. As with the other cord lesions, MRI is the imaging modality of choice. The entire cord can be evaluated and small areas of signal abnormality identified. As with other intramedullary lesions, the metastatic lesions are generally hypointense on T1- and hyperintense on T2-weighted images. Contrast enhancement aids in detection of small lesions. The prognosis for patients with intramedullary metastases is grave, and the role of screening MRI in detection of clinically occult spinal metastases is still not clear. Over half such patients die within 1 month of the initial medical intervention for the neurologic dysfunction. Fewer than 20% are alive at 3 months.

Some Non-Neoplastic Diseases Of The Spinal Cord

Multiple sclerosis. MRI is the most effective technique for demonstration of MS plaques. Spinal cord MRI is positive in 75% to 86% of patients in whom MS of the cord is clinically suspected. The ability of MRI to demonstrate multiple lesions in the brain, as well as in the cord, helps to establish the diagnosis by showing dissemination in space and by revealing "silent" lesions that may reflect prior involvement. The occurrence of a normal brain MRI and positive spinal cord MRI occurs in 16% to 50% of patients. Ormerod et al and Miller et showed that 74% of patients with isolated brainstem signs, 82% of patients with isolated chronic myelopathy, and 56% of patients with acute myelopathy had brain MRI lesions of the type seen in MS. Miller et al additionally showed that brain MRI was superior to evoked response testing in detecting abnormalities in acute and chronic non-compressive spinal cord syndromes. They found that 72% of 18 patients with acute myelopathy with baseline brain MRI abnormalities of the type seen in MS developed clinically definite MS during the subsequent 14.1 (mean) months. In contrast, only 7% of 15 patients with acute myelopathy without baseline brain MRI abnormalities developed clinically definite MS. Ford et al reported on 15 patients with isolated partial transverse myelopathy for a mean of 38.5 months and found that 93% of those patients with baseline brain MRI lesions of the MS type were subsequently confirmed to have clinically definite MS. The criterion requiring multiplicity in time and space for clinically definite MS could be satisfied by a gadolinium-enhanced MRI study if it showed chronic non-enhancing lesions as well as acute enhancing lesions.

MS plaques in the spinal cord can rarely undergo cavitation. Cavitation is presumed to result from severe inflammatory changes occurring in some acute plaques. Large acute lesions may be associated with severe edema and cord swelling leading to vascular compression, ischemia and acute necrotic myelopathy.

Multiple sclerosis (MS) is the most common demyelinating disease of the central nervous system

* Possible autoimmune-mediated
* Female predonderance, young adults
* Cervical cord most often involved
* Posterior lateral cord most affected
* One or more poorly marginated elongated plaques
* Hyperintense plaque on T2w images
* Brain also positive in over 75%
* Acute plaque may swell, enhance
* Myelin and oligodendrocyte destruction

Chronic lesions cannot be differentiated from acute or subacute lesions on non-contrast images unless there is a change in cord size. Focal or diffuse spinal cord atrophy may occur in chronic MS. Short segment focal atrophic areas result from involution of a localized plaque. These areas often have a persistent high signal abnormality on T2-weighted MR images. Diffuse cord atrophy is a well known end result of chronic MS. and frequently there is no signal abnormality.

MRI Technique

Spin-echo T1-weighted images often fail to show spinal cord MS plaques. Long-TR, long -TE spin-echo T2-weighted images show the MS plaques as areas of increased signal intensity compared with the adjacent normal matter.

The hyperintensity may be quite subtle and frequently is only recognized as a loss of differentiation between the gray matter and the normal low signal intensity of the dorsal white funiculi. T2* low flip -angle gradient-echo images an effectively demonstrate spinal cord MS, particularly when images are acquired in the axial plane. They are considered as sensitive as conventional spin-echo images for detection of cord pathology. Gradient-echo images often have superior resolution, are more robust, and are easier to obtain than their conventional spin-echo counterparts. However, optimized spin-echo T2-weighted images are generally considered superior to optimized gradient-echo T2* images. Because inactive lesions are not seen on T1-weighted enhanced or non-enhanced images a T2-weighted image should always be obtained. Fast-spin-echo (FSE) T2-weighted images are increasingly being used in the spine. Because of time saving these sequences can combine T2-weighting with higher resolution matrices e.g. 512 X 512 acquisition. When these images are obtained without artifact they offer increased sensitivity to spinal cord lesions. There is some concern that FSE images are less sensitive than conventional SE images for plaque detection but the higher resolution FSE images may offset this disadvantage compared to lower resolution SE images.

Contrast Enhanced MRI In MS

Gadolinium contrast enhancement occurs in acute lesions but the duration of enhancement is variable, correlating with the variable course of disease in patients. Miller et al studied 10 patients with serial scans and made four important observations including; (1) 8 of 10 patients showed new enhancing lesions; (2) most new lesions were asymptomatic [brain lesions]; (3) enhancement usually lasted less than 1 month, never longer than 6 months, and there were persistent lesion on T2w images after that; (4) pre-existing lesions accounted for only 10% of the new enhancing activity. Capra et al monitored 10 relapsing remitting patients with MS with serial Gd-DTPA enhanced MRI scans of the brain and spinal cord every 14 days for 3 months and noted that only 50% of the enhancing lesions persisted for more than one scan.. Enhancement may occasionally show a small peripheral lesion that is obscured on T2w-imaging due to its small size and proximity to high signal intensity CSF.

MS plaque enhancement is not likely in patients with clinically stable disease. High dose steroids are used in the treatment of acute MS. Barkoff et all studied the relationship between steroid and gadolinium enhancement and concluded that (1) improvement in blood-brain-barrier integrity correlated with clinical improvement and (2) serial enhanced MRI scans were more likely to detect

disease-related activity than were serial non-contrast MRI scans. Burnham et al suggested that lack of resolution of enhancing lesions or increasing size of lesions on T2w images during the steroid taper might predict relapse or continued deterioration (this has not been confirmed). Enhancement of acute brain and spinal cord lesions reflect disruption in the blood-brain-barrier due to inflammation with associated myelin breakdown. The clinical effect of high dose steroid treatment might be accompanied not only by stabilization of the BBB and improvement in the volume of edema but also with a reduction of myelin breakdown.

Devic Disease. The simultaneous or consecutive involvement of the optic nerves and spinal cord was remarked upon by Clifford Albutt in 1870 and expanded upon Gault (studying under Devic.) in 1894 The principal features of this disease are acute to subacute onset of blindness of one or both eyes preceded or followed within days or weeks by a transverse or ascending myelits. The lesions are more necrotizing than demyelinating and the clinical effects are more likely to be permanent. Unlike MS there is no evidence of cognitive dysfunction.

Devics's Disease
(neuromyelitis optica)
* Acute necrotizing myelopathy and optic deymyelination
* Primarily seen in females
* Some cases appear unique and differentiated from M.S.
* Normal brain MRI with enlargment and cavitation on spinal cord MRI. Hyperintense optic nerves.
* Decreased serum/CSF albumin ratio usually without CSF oligoclonal bands

Acute Transverse myelitis (ATM) is an inflammatory intramedullary disorder of the spinal cord. The term is non-speficic in that a number of conditions and etiologic agents are associated with the clinical syndrome of transverse myelitis including parainfectious and postvaccinial events, multiple sclerosis, paraneoplastic myelopathy, vascular insuffienciecy and autoimmune disorders. Some of these disease processes are included in other areas of this book.

Acute Transverse Myelitis (ATM)
* Inflammatory disease of the spinal cord
* Five strict clinical
* Etiology is very variable
* Autoimmune etiology favored
* Postinfectious ATM most common under age 40 years
* Possible ischemic etiology in older
* Recovery in most patients between 4 weeks and 3 months after onset
*MRI shows increased T2w-signal, variable cord area involved, possible cord swelling and gadolinium enhancement

-CLINICAL-
In the clinical setting, it often difficult to distinguish amonth the different causes of TM. Multiple sclerosis is often considered a likely diagnosis but there may be little colinical or laboratory evidence ot support that diagnosis.

The diagnostic criteria [Berman; Jeffery]of the disease included the following
1)acute or subacute development of sensory , motor and sphincteric dysfunction
2)spinal segmetal level of sensory disturbance with a well-defined upper limit
3) no clinical or laboratory evidence of spinal cored comnpression
4) absence of other known neurologic disease
5) lack of clincial progression over 4 weeks such that the period for onset of neurologic symptoms to peak deficit was no greater than 4 weeks.

Subacute Encephalomyelitis . This disease may be a variant of acute transverse myelitis (ATM). It is distinguished from ATM by the gradual progressive development of spinal cord and brainstem dysfunction. MRI before treatment with high dose steroids reveals spinal cord swelling, increased intensity on T2-weighted images and enhancement following gadolinium-DTPA injection. The etiology of the disease is unknown but histologic analysis reveals inflammatory changes without infection similar to findings in patients with ATM [14][**McCombe 1989**].

AIDS-related spinal cord disorders include a range of infectious, neoplastic and idiopathic etiologies. AIDS patients presenting with chronic myelopathy may have normal MR of the cord or cord atrophy. Pathologically these patients have often have vacuolar myelopathy primarily involving the lateral and posterior columns, resembling that of subacute combined degeneration of vitamin B-12 deficiency. The myelopathy may be due to a remote or indirect effect of HIV or other infectious agent[15]. It is most common in patients with opportunistic infections. Myelitis due to mycobacteria[16][**Woolsey 1988**], toxoplasma[17] and viral infection with varicella-zoster, herpes simplex, CMV and HTLV III viruses has been reported[18][**Britton 1984**] [19][**Tucker 1985**] [20] [**Ho 1985**]. Increased intensity with or without cord swelling has been seen on T2-weighted MR images[21][**Harris 1990**] [22]**Mehren 1988**] [23][**Sze 1987**]. The MR appearance of AIDS myelitis is indistinguishable from acute transvere myelitis [24][**Barakos 1990**].Myelopathy due to viruses in immunocompromised patients may be caused by primary or secondary mechanisms. Secondary mechanisms include immune-mediated demyelination of the spinal cord and parainfectious vasculitis with subsequent cord ischemia [25][**Britton 1984**].

Collagen vascular disease may occasionally involve the spinal cord. Systemic lupus erythematosis (SLE) is the most likely disease of this category to be recognized clinically. Vasculitis and the associated complications of this disease and its treatment may affect the spinal cord including infarction and transverse myelitis. SLE patients may develop vacuolar myelopathy similar to that seen in subacute combined degeneration[40][**Devinsky 1988**].

MISCELLANEOUS MYELITIS/MYELOPATHY

The differential diagnosis of myelopathies poses a clinical problem. Multiple sclerosis is often suspected but accounts for only about 10% - 32% of idiopathic myelopathy[42] **[Ropper 1978]** [43] **[Marti-Fabregas 1989]**. Myelitis can be caused by a wide variety of infectious and toxic agents. Although uncommon, spinal cord infections may be caused by bacteria, viruses, fungi or parasites, particularly in patients who are intravenous drug abusers or immunocompromised[44] **[Johnson 1982]** [45] **[Dupuis 1990]** [46] **[Gero 1990]** [47] **[Koppel 1990].** MR with gadolinium-DTPA MR may be helpful by indicating that the primary disease site is either intramedullary or meningeal.

Cystercercosis may present with single or multiple cord cysts [48] **[Castillo 1988]**.

Lyme disease (spirochete Borrelia burgdorferi) diversely affects the CNS and may therefore simulate multiple sclerosis clinically and on MR studies.

Radiation Myelopathy. The diagnosis of radiation myelopathy is based on the following criteria: spinal cord within the irradiation field, late onset of the clinical disorder (36 months), concordance between the level of clinical signs and that of the spinal cord radiation port, absence of clinical and imaging evidence of metastasis [52] **[Palmer 1976]**.Chronic radiation myelopathy has a variable appearance on MR [53] **[Sze 1989]** [54] **[deToffol 1989]**. The MR may be normal, reveal diffuse atrophy or abnormal areas of increased intensity in the cord may be seen on T2-weighted images. The areas of hyperintensity correspond to radiation ports and may be asssociated with either a normal-sized cord or with cord enlargement. Gadolinium enhancement may be seen in these patients.

VASCULAR DISEASE OF THE SPINAL CORD

Vascular diseases of the spinal cord include AVM, AVF, ischemia, aneurysm, angiitis and hemorrhage.

Arteriovenous Malformations and Fistulas

Spinal AVMs have been broadly divided into two groups; intramedullary and extramedullary. Intramedullary AVMs are usually seen in young patients and are characterized by acute hemorrhage and primarily derived from the anterior spinal cord blood supply. Most spinal AVMs are extramedullary and often seen in elderly men. These lesions are characterized by progressive neurologic deficits and a posterior blood supply.

Vascular malformations of the spinal cord have been classified into four different types.

CLASSIFICATION

Type I spinal vascular malformations are characterized by a single tortuous arterialized vein that trellises over the pia of the spinal cord.. Some of these lesions may have additional supply from one or more adjacent levels. These cases can be subdivided as type I-A (single feeder) and type I-B(multiple feeders). Type I-B lesions have small vessels running within or beneath the dura from the adjacent feeders to the site of the primary dural fistula. The differentiation of these subtypes is important for optimal therapeutic management. The lesion will persist or recanalize with supply from the additional feeders if only the main feeder of type I-B AVMs is obliterated.

* Arteriovenous malformation (AVM)
  true nidus of abnormal vessels
* Arteriovenous fistula (AVF)
  direct arterial to venous connection
* Classification includes AVM & AVF
  Type I dural AVF
  Type II intramedullary (glomus) AVM
  Type III (juvenile) intramedullary, dural and extraspinal
  Type IV intradural perimedullary AVF
* Dural AVM, AVF most common type
  patients usually > 40 years
  progressive myelopathy
* Intradural AVM, AVF uncommon
  patients usually < 40 years (avg. 25)
  abrupt onset more common
  hemorrhage

Type II, or glomus, spinal vascular malformation contain an identifiable focal intramedullary nidus of AVM supplied by multiple feeders from the anterior or posterior spinal arteries. Type II AVMs drain into a tortuous , arterialized venous plexus that surrounds the spinal cord.

Type III, or juvenile, spinal AVM's are also called juvenile AVM's and are large, complex, diffuse lesions that involve the cord and paraspinal (even extraspinal) structures. Multiple arterial feeders from several different vertebral levels are common.

Type IV. Intradural, or perimedullary, arteriovenous fistula. These lesions like completely outside the spinal cord and pia mater. There is no intervening small vessel network, and the fistula drains directly into an enlarged venous outflow tract of variable size. These lesions can range from small arteriovenous fistulas between the anterior spinal artery and an adjacent vein to giant fistulas with high flow and marked dilation of the artery and draining venous channels. Most type IV AVMs are anterior to the spinal cord and are fed by the anterior spinal artery. Most occur near the conus medullaris. Type IV AVMs occur in patients between their third an sixth decades. Endovascular occlusion of the fistula , as well as surgery should be considered, particularly for the giant fistulas.

-EPIDEMIOLOGY-

Spinal AVMs represent 3% to 4% of spinal cord masses. Spinal AVMs occur more commonly in males and are usually manifested between the fifth and

seventh decade Symon noted a 9:1 male predominance with an age range of 29 to 75 years. Only 13% of all spinal AVMs occur in patients under the age of 20[Morgan] The lower thoracic spinal cord and conus are usually the most severely affected, despite the locus of the fistula. The most common AVM is type I, i.e., dural arteriovenous fistula. Nearly 60% of Type I spinal AVM are spontaneous and approximately 40% are caused by trauma [Beaujeux RL, Reizine DC, Casasco A et al. Endovascular treatment of vertebral arteriovenous fistula. Radiol 1992;183:361-367]

-ETIOLOGY- OF MYELOPATHY

The pathophysiology of these malformation is obscure and five mechanisms have been proposed to explain the clinical features.

1)The most favored mechanism is that of venous congestion, thrombosis and hypertension supported by Aminoff et al and later by Kendall and Logue Increased pressure within the coronal venous plexus causes venous distention, congestion, and stagnation in the radial veins draining the spinal cord. This in turn reduces the arteriovenous pressure gradient between abnormal arteries supplying the region and the efferent veins, resulting in diminished intramedullary blood flow. The resulting ischemic edema causes further reduction in blood flow in the spinal microcirculation thus aggravating the ischemia in a vicious cycle.

2)A vascular steal phenomenon may cause ischemia in some cases but this theory is generally discounted

3)A mass effect may be created by the tangle of abnormal blood vessels , usually distended and arterialized veins and occasionally large and often thrombosed venous aneurysms.[Cogen]

4)Acute hemorrhage into the spinal cord (hematomyelia) or into the subarachnoid space

5)Repeated hemorrhages resulting in spinal cord damage, arachnoiditis, scarring and secondary ischemia to the spinal cord.

Ischemic disease. Because of the limited but critical sources of blood supply to the spinal cord, any pathologic processes that interfere with this crucial blood supply may result in ischemia and/or infarction in the spinal cord. Infarcts of the spinal cord are rare lesions. The majority of arterial infarcts are secondary to occlusion of the anterior spinal artery, which supplies the ventral two thirds of the cord (25,27). The thoracic and thoracolumbar regions are most commonly involved since these levels are primarily supplied by one large anterior medullary vessel, the artery of Adamkiewitz, which has poor collateral circulation.

Anterior Spinal Artery Infarction

The characteristic clinical syndrome of the anterior spinal artery syndrome (anterior myelopathy) is loss of pain and temperature sensation below the level of the lesion and a relative sparing of vibration and proprioceptive sensation

(mediated through the dorsal columns). The ventral gray matter and the corticospinal tracts are affected causing paralysis of motor function. The maximum deficit occurs either immediately or after progression over a few hours. The clinical manifestations of arterial occlusion will, of course, vary with the level of infarction. Bowel and bladder paralysis are common features of the clinical picture. Pain is sometimes a complaint, either diffuse in a segment of the body (e.g. legs) or more often radicular, corresponding to the upper level of the lesion. Initially the limbs are flaccid and areflexic, as in spinal shock from other abrupt transverse leions, followed after several weeks by the development of spasticity, hyperactive tendon reflexes, conus and Babinski signs, and some degree of voluntary bladder control. Rarely, spinal cord infarction is limited to the gray matter and in these patients there is abrupt onset of muscle weakness in the legs, but no pain or sensory loss.

Poterior Spinal Artery Infarction

Poterior spinal artery infarction is rare, most likely due to the increased number of medullary arteries that supply this vessel. The clinical presentation is variable but presenting symptoms include pain, loss of proprioception, and deficits in the lateral corticospinal and spinocerebellar tracts. Infarction is usually bilateral but may be bilateral or unilateral. The posterior horn(s) and lateral corticospinal tract(s) are affected. In a review of the literature, Kaneki et al noted that in 27 cases the posterior columns were affected in all. In 25 of the 27 cases there was bilateral involvement. They reported an unusual case in which thre was sparing of the posterior columns and unilateral involvement of the cord.

Venous Infarction

Acute venous obstruction is rare. A review by Hughes in 1971 revealed only seven reports. The primary causes have included tumor and extensive generalized septicemia. Acute infarction may be caused by thrombophlebitis Ohshio reported a patient with non-Hodgkin's lymphoma who died of Candida sepsis. One week before death he became acutely paraplegic and at autopsy an infarct due to Candida induces venous thrombosis was showhn in the right lateral column. Instances of a subacute or chronic spinal cord syndrome also occur that are produced by either a more gradual venous thrombotic occlusion or a recurrence of episodes of spinal cord damage due to recurrent thrombosis. These instances should be differentiated from spinal vascular malformations, in which venous thrombosis can be found. At necropsy the spinal veins are always greatly distended and are usually extensively thrombosed. The venous obstruction can, however, be sited in the plexus of veins in the spinal canal or in the veins in the pelvis and abdomen. The spinal cord itself is disrupted by severe hemorrhagic necrosis in which small hematomas are conspicuous. The most affected parts of the spinal cord are the central areas, and the infarction is much more hemorrhagic than after arterial occlusion. The extent of the infarct both longitudinally and in cross-sectional area is even greater than that caused by an extensive thrombosis of the anterior spinal artery.

Mechanisms of Spinal Cord Ischemia

There is no obvious explanation for why spinal infarctions occur less frequently than do cerebral infarcts. One explanation that has been advanced is the presence of rich anastomotic channels among the various levels of the cord through coronal vessels. However, well-designed experiments have demonstrated that these anastomotic channels are probably inadequate to perform the task of redistributing blood flow further than one or two adjacent segments and that, at best, they may protect only the peripheral or superficial white matter. Much the same is true of the medullary vessels. Although their capillary bed appears more extensive than that found in the cerebral circulation, they act as end arteries and provide little or no flow between anterior and posterior circulation in the cord. Extraspinal anastomoses do exist and may provide functionally adequate redistribution of flow in instances of occlusion of spinal or segmental vessels. These are most evident in the cervical region and less well defined caudally, a possible explanation for the relatively greater incidence of caudal ischemia. These extraspinal anastomotic channels may also account for the observation that the closer vascular occlusion occurs to the aorta or vertebral arteries, the less likely is the cord to suffer ischemia. In addition to the adequacy of blood supply, another important factor in determining ischemic damage is the tissue's vulnerability to ischemia. In this respect, the spinal cord may have an advantage over the brain. The greater metabolic requirements of the brain than those of the cord is suggested by the former's greater blood flow (both organs autoregulate blood flow) . The total blood flow in the brain is 50 ml/minute/100g. The blood flow in the cord varies according to the spinal level and the species studied, but a rate of 19.7 +/- 1.2 ml/minute/100g of tissue probably represents maximal flow in the resting cord. Indeed, experiments designed to study the spinal cord's resistance to ischemia suggest that no permanent ischemic damage occurs in the cord with as much as 30 minutes of interruption in blood supply by aortic clamping. This experiment, carried out in dogs. is one of many suggesting that the cord can survive relatively long periods of ischemia. Clinical data also suggest that aortic clamping times of less than 30 minutes are attended by a lower incidence of permanent spinal ischemia. It is also clear that when ischemia occurs, the most vulnerable region of the spinal cord is the gray matter, because its metabolic rate may be three to five times greater than the metabolic rate of white matter. This helps to explain the many cases reported in the literature in which there is severe paraparesis with little or no sensory involvement and for instances of progressive lower motor neuron syndrome on an ischemic basis.

-IMAGING.

The role of imaging is to define the site and nature of the vascular occlusion, to determine the reason for the occlusion, and to exclude etiologies such as cord compression and demyelinating, inflammatory, or infectious disease. Magnetic

resonance imaging is fast becoming the examination of choice in spinal cord injury. MR imaging is considered to be the most sensitive examination for detection of spinal cord infarcts. MRI can suggest the presence of meningeal arteriovenous malformation, malignancy, and inflammatory or infectious pathology involving the spinal canal . Acute infarcts are identified as areas of increased signal intensity on T2-weighted images, often associated with spinal cord swelling and enlargement but it may be negative in the earliest stages of infarction. Eventual breakdown in the blood-spinal cord barrier produces enhancement of the infarcted regions. MRI should be performed using double-echo T2w technique, since the geographic distribution of the signal abnormalities within the gray matter cannot be accurately displacyed on sagital scns. In addition, well-position scanss with cardiac gating and motion-suppression techniques, as well as proper patient sedation, are necessary to avoid artifactual signal abnormalities in the center of the cord.

CAUSES OF SPINAL CORD ISCHEMIA
Aortic Disease aortic dissection aortic surgery aortic catheterization Systemic hypotension Atherosclerosis Intercostal artery occlusion thoracotomy trauma compression Embolic disease decompression sickness (nitrogen bubble emboli) fibrocartilagenous emboli thromboemboli antiphospholipid antibody syndrome hypercoagulable states neoplastic emboli parasitic emboli Venous hypertension/infarction Vasculitis

Mawad et al showed four different patterns of signal abnoralities in the distribuiton of the anterior spinal artery. Cases in which the increased signal was limited to the anterior horns of the gray matter had some preservation of motor

function and a better clinical outcome than those in which t he signal abnormalities were more diffuse and involved the adjacent central white matter. The region of the white matter may be supplied by either the centrifugal arterial system dependent on the anterior spinal artery or the centripetal peripheral system arising from the posterior spinal arteries. If MRI is not available, CT, preferably with myelography, should be carried out. CT and myelography are relatively ineffective in evaluation of spinal cord ischemia. Although both techniques can visualize cord enlargement, the amount of cord swelling during acute infarction is variable and may not be easily detected. Subtle changes in cord density have been described on CT but CT has the disadvantage of giving a poor image of the spinal cord and subtle density changes are not reliable.

Hemorrhages originating within the spinal canal may be localized to the spinal cord substance (hematomyelia) or subarachnoid, subdural, or epidural spaces. Patients with *hematomyelia* usually present with a rapidly evolving myelopathy, although Licata et al have reported a gradual and lengthy clinical course in three paients and Wisoff reported a patient with Factor XI deficiency who had a probable prodromal hemorrhage one week prior to the catastrophic bleed. Depending on the level of the lesion, the patient may present with paraplegia or quadriplegia, bowel or bladder dysfunction, and sensory disturbance involving all modalities.

In patients with *subarachnoid hemorrhage* the initial symptom is often the acute paroxysmal onset of severe back or neck pain, termed "le coup de poignard" or the "strike of the dagger", which is known as the hallmark symptom of spinal SAH. There is often a history of multiple episodes of back pain suggesting intermittent hemorrhage. Sudden pain is frequent experienced at the level of the lesion, and pain may radiate into one or both legs, the flanks, or occasionally into the abdomen, thus mimicking a visceral catastrophe. There are frequently no signs of spinal cord or cauda equina dysfunction; however, sudden paraparesis, urinary retention, and leg numbness may occur at the onset. meningismus, with Brudzinski's and Kernig's signs, is usually prominent soon after the hemorrhagic event. Minor SAH can present predominantly as radicular pain that is often mistaken for a musculoskeletal problem. The presence of headache, nausea, or vomiting may provide the only clue to spinal SAH. When evidence of SAH is seen together with neurological signs or symptoms referable to the spinal cord, the possibility of a spinal vascular abnormality must be entertained. Occasionally, auscultation of a bruit over the spine signifies excessive blood flow in an underlying AVM. This sign may be enhanced by exercising the patient prior to the auscultation.

Patients with spinal *epidural hematoma* (EDH) usually present with sudden, acute back pain or neck pain that may have a radicular distribution (Lanzieri, Zilkha). Shortly after the onset of pain, from minutes to hours and sometimes days, the signs and symptoms of cord or cauda equina compression develop. Patients may develop long track sings and a snsory level. Depending upon the

loaction, paraplegia or quadriplegia develops. Less commonly, te progression of disease may be less acute and even have an undulating pattern. Spinal EDH is more likely in older patients although spinal EDH may appear in the child age group.

-ETIOLOGY-

Causes of spinal hemorrhage include arteriovenous malformations, aneurysms and tumors, syringomyelia, bleeding diathesis and spontaneous idiopathic hemorrhage. Wisoff *et al* reported a 34 year old woman with a Factor XI deficiency who developed an acute intramedullary hematoma extending from C3 to C7. Cruickshank reported a patient who developed cervical cord hematomyelia after an intravenous injection of 1.5 million units of steptokinase. Gowers described a series of four patients with presumed syringomyelia who suffered an apoplectic increase in symptoms and signs. One patient at autopsy was noted to have extensive hemorrhage into a preexistent syringomyelia cavity. Perot et al reported a 39-year-old man with known syringomyelia who, 6 years later, developed hematomyelia precipitated by trauma. The patient underwent surgery which revealed bleeding into a cervical cord syrinx cavity . Perot speculated that abnormal vessels in the wall of the syrinx predisposed the patient to hemorrhage due to trauma. Rarely, hemorrhages are spontaneous and of unknown etiology.

SPINAL ANEURYSM

* Isolated spinal artery aneurysm is rare
* Most associated with high flow state
* Most spinal artery aneurysms are associated with AVM
* Most common characteristics include:
  - present with hemorrhage
  - usually extramedullary
  - usually involve anterior spinal artery
  - usually do not have true neck
  - usually do not involve a bifurcation
* Other associations include:
  - coarctation of the aorta (high flow)
  - vasculopathies

The most common cause of spinal subarachnoid hemorrhage is probably AVM of the spinal cord [Swann]. The incidence of spinal hemorrhage (SAH or hematomyelia) with spinal AVMs' is 10%-70%[Cogen, Symon, Aminoff, Tomlinson, Guegen]. AVMs that involve the spinal cord are more likely to be associated with hemorrhage than those AVMs or fistulas that are extradural, however Morimoto reported a 61 year old patient with a cervical dural AVM in whom the initial symptoms indicated a SAH, confirmed by spinal tap. Hemorrhage from a spinal dural AVM might be due to venous hypertension which results when arterial blood passes through the dural AVM into the medullary vein and reaches the valveless coronal venous plexus. Hemorrhage is more common when AVMs are associated with aneurysms and in aneurysms alone. Shephard cautioned that hemorrhage might be a particular risk of a younger age group harboring these lesions. Young patients are more likely to have intramedullary AVMs.

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A selection of case reviews from the teaching file at the Medical College of Ohio is presented demonstrating less common lesions of the neuroaxis. The contributions of Magnetic Resonace Imaging (MRI) are highlighted using the cases as examples.

The utility of MR multiplanar scanning in complex anatomy is illustrated in skull base lesions, in the sacrum, and in complex congenital abnormalities of the brain and orbit. The superior sensitivity of MRI in white matter evaluation is demonstrated in cases of demyelinating disease.

Some newer use in post-operative evaluation of patients with titanium posterior and anterior spinal plates is demonstrated. MR scanning can provide excellent detail in evaluation of post-operative complications in most of the newer titanium orthopedic appliances.

RENAL CYSTIC DISEASE:
FOR THE NON-URORADIOLOGIST

David S. Hartman, M.D.

INTRODUCTION

Renal cystic disease comprises a diverse group of heritable, developmental, and acquired disorders. An encyclopedic description of the clinical, pathological and radiological features the spectrum of renal cystic disease is beyond the scope of this abstract. Rather, the purpose of this presentation is to emphasize the current concepts, controversies and imaging strategies of three renal cystic diseases: 1. several important concepts of autosomal dominant polycystic kidney disease, 2. the .diagnostic criteria for medullary sponge kidney and its relationship to benign tubular ectasia and 3. the pathogenesis, natural history and radiologic evaluation of multicystic dysplastic kidney.

AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE

DEFINITION

Autosomal dominant, or adult, polycystic kidney disease is a genetically transmitted disorder that results in large numbers of variable-sized cortical and medullary cysts and impaired renal function. It is the most common renal cystic disease affecting about 500,000 Americans. The gene is located on the short arm of chromosome sixteen where it can be detected by gene linkage analysis.

PATHOLOGY

The pathologic findings vary with the number and sizes of the cysts. With minimal disease, the kidney is normal in size and its surface is smooth; cysts are discovered only on cut section. As they increase in size and number, the cysts enlarge

the kidney but do not necessarily distort the renal contour. Increase in renal volume usually precedes impaired renal function. As the cysts continue to enlarge, they project beyond the renal margin, causing the renal contour to become bosselated.

CLINICAL

Palpable mass, hypertension, abdominal pain, hematuria and urinary tract infection are common complications of autosomal dominant polycystic kidney disease. Death due to rupture of a cerebral aneurysm into the brain and subarachnoid space occurs in approximately 10% of patients with autosomal dominant polycystic kidney disease.

RADIOLOGY

Autosomal dominant polycystic kidney disease is usually imaged with ultrasound, CT or MRI. With minimal disease, sonographic examination demonstrates small echo-free masses in otherwise normal kidneys. As the disease progresses, the kidneys enlarge, the contour becomes bosselated and numerous cysts are visualized. Marked variation in the size of the cysts is common. The central sinus echo complex is distorted by the larger cysts.

Uncomplicated cysts have CT attenuation values near that of water and do not enhance. Because of the dense parenchymal blush of uninvolved surrounding renal parenchyma, the cysts are accentuated on contrast enhanced scans. Acute hemorrhage into a cyst is recognized as a well-defined hyperdense mass on an unenhanced scan.

Uncomplicated cysts are homogeneous and low intensity on T1-weighted images and homogeneous and high intensity on T2-weighted images, just as are simple cysts. Cysts complicated by hemorrhage are usually hyperintense, but the appearance varies with the amount of time since the bleeding

episode.

EXTRARENAL MANIFESTATIONS

Liver cysts are found in up to 60% of patients with autosomal dominant polycystic kidney disease. Pancreatic cysts are found in approximately 10% and splenic cysts in up to 5% of these patients. Cysts of the thyroid, ovary, endometrium, seminal vesicles, lung, brain, pituitary gland, breasts, peritoneum, parathyroid, pineal, and epididymis have also been reported.

Extrarenal cysts seldom cause symptoms. There is no correlation between the severity of renal cystic disease and the severity of hepatic cysts. Rare complications of hepatic cystic disease include infections, hemorrhage, portal hypertension, cholangiocarcinoma, biliary obstruction and congenital hepatic fibrosis.

Extrarenal cysts are best identified by computed tomography or sonography. The precise role of magnetic resonance imaging relative to established modalities for screening, diagnosis and follow-up of cases of autosomal polycystic kidney disease is unknown.

Approximately 20% of patients with autosomal dominant polycystic kidney disease have intracranial aneurysms at autopsy. The overall prevalence of intracranial aneurysms in the general population is estimated to be 5%. Aneurysm less than 1 cm rarely rupture but demonstrate an increased risk with an increase in size.

There is no good information about the natural history of intracranial aneurysms associated with autosomal dominant polycystic kidney disease. It has been suggested that *routine* angiographic screening should not be performed in patients with autosomal dominant polycystic kidney disease. There is,

however, some evidence that there is clustering of intracranial aneurysms in certain families with autosomal dominant polycystic kidney disease. Magnetic resonance angiography (MRA) is a non-invasive technique which may prove useful in screening. While *routine* screening may not be efficacious, MRA or cerebral angiography should probably be performed in patients with autosomal dominant polycystic kidney disease and 1. a family history of berry aneurysm, 2. individuals who perform activities in which sudden loss of consciousness would endanger the lives of themselves or others, 3. neurological signs and symptoms. Surgery is probably indicated for intracranial aneurysms 10 mm in diameter or larger.

Although the association with autosomal dominant polycystic kidney disease and cerebral aneurysms is well known, there are many other cardiovascular abnormalities associated with autosomal dominant polycystic kidney disease. Approximately 18% of patients hospitalized with autosomal dominant polycystic kidney disease and 27% of autopsied patients have one or more cardiovascular abnormalities. These abnormalities are independent of the hypertension which may complicate autosomal dominant polycystic kidney disease. Aortic aneurysms or dissections, bicuspid aortic valve, aortic coarctation, dilation of the aortic root and mitral valve prolapse have all been reported. Of these, dissecting thoracic aortic aneurysm is the most common vascular association of autosomal dominant polycystic kidney disease. The observed frequency of co-existing autosomal dominant polycystic kidney disease and dissecting thoracic aortic aneurysm is approximately seven times greater than expected from chance association alone.

MEDULLARY SPONGE KIDNEY

DEFINITION

Medullary sponge kidney, an abnormality of unknown etiology,

is characterized by dilation of the terminal portion of the collecting tubules within one or more renal pyramids of one or both kidneys.

PATHOLOGY & PATHOGENESIS

Renal size is usually normal to slightly enlarged. Cut section of the kidney reveals multiple cyst-like cavities ranging from 1 to 7.5 mm in diameter in one or more pyramids. Each of these represents dilation of the terminal portion of the collecting ducts and is confined to the papillary portions of the pyramids. Calculi frequently are discovered within the dilated spaces. In most cases, both kidneys are involved.

Medullary sponge kidney is usually diagnosed radiologically. Because there are differences in the radiologic criteria for medullary sponge kidney and because cases of medullary sponge kidney rarely come to pathologic scrutiny, the true prevalence of medullary sponge kidney is unknown. Likewise, its pathogenesis is purely conjectural (congenital vs acquired). It is uncertain whether ductal dilation predisposes to stone formation or stone formation results in adjacent tubular dilation.

CLINICAL

Uncomplicated medullary sponge kidney usually causes neither symptoms nor impaired renal function, although there have been reports of impaired concentration or acidification of urine. Medullary sponge kidney is associated with an increased frequency of calculi which, in turn, may cause colic, hematuria, fever, dysuria, or flank pain.

RADIOLOGY

In the past, the minimum urographic criterion for the diagnosis of medullary sponge kidney was the demonstration of discrete linear densities in one or more papillae on non-magnified radiographs. This was often referred to as benign

tubular ectasia or minimal medullary sponge kidney and felt to indicate that the collecting ducts were wider than normal (200 to 300 microns). This same phenomenon, however, is seen in approximately 10-12 percent of cases in which low osmolar contrast medium is utilized. There is no good evidence that "benign tubular ectasia" progresses to frank medullary sponge kidney with nephrocalcinosis and/or nephrolithiasis.

A radiologic diagnosis of medullary sponge kidney should probably be made only when nephrocalcinosis or nephrolithiasis can be demonstrated along with visualization of discrete collecting ducts. With minimal disease individual ducts adjacent stones will be barely perceptible. With moderate ductal ectasia, extracalyceal contrast collections become more prominent especially after a stone has passed into the calyx. The most advanced stages of medullary sponge kidney cause severe deformity of the papillae, with beaded or striated cavities and marked distortion of the calyces.

Medullary sponge kidney is best appreciated on excretory urography. Ultrasound and CT are less specific. The sonographic findings of medullary sponge kidney include intensely echogenic shadow-producing deposits of calcium in the pyramids and dilated, anechoic spaces (collecting ducts). Although medullary nephrocalcinosis is fairly easily detected sonographically, demonstration of dilated collecting tubules is much more difficult. Unenhanced CT scans typically demonstrate medullary nephrocalcinosis and/or nephrolithiasis. To distinguish medullary sponge kidney from other causes of medullary nephrocalcinosis, extracalyceal contrast in the dilated collecting ducts must be visualized. This may be impossible utilizing CT when the dilation is minimal or moderate.

MULTICYSTIC DYSPLASTIC KIDNEY
DEFINITION

Multicystic dysplastic kidney is a nonhereditary developmental anomaly characterized by various-sized cysts with little or no discernible renal parenchyma.

PATHOLOGY

Multicystic dysplastic kidney is usually unilateral and involves the entire kidney. Rarely, the condition involves part of the kidney (segmental) or both kidneys (bilateral which is lethal). Multicystic dysplastic kidney is most commonly associated with complete atresia of the pelvis or ureter early in fetal life. In this form, there is severe derangement of growth and branching of the ureteral bud. The "kidney" lacks a reniform outline and consists of only a grape-like cluster of thin-walled cysts. The mass of cysts is held together by connective tissue. No cortical or medullary structure can be identified. With severe but incomplete obstruction early in nephrogenesis, pathologic changes are similar to those described previously except that the pelvis is dilated and there is communication with the cysts (hydronephrotic multicystic kidney).

CLINICAL

The clinical features of pelvoinfundibular atresia and severe hydronephrotic multicystic kidney are similar. Males and females are equally affected. Most cases of multicystic dysplastic kidney are of an otherwise asymptomatic neonate who presents with a large mass. In a newborn, the most important clinical consideration with multicystic dysplastic kidney is the recognition of contralateral anomalies, such as ureteropelvic junction obstruction and ureteral anomalies which may be life threatening. While severe anomalies are rare, minor anomalies (e.g. malposition, grade I reflux) can be identified in 10-15% of cases. Multicystic dysplastic kidney is sometimes discovered in the older child or adult. In most of these cases, the malformed kidney is small or normal in size and is detected as an incidental radiographic finding, usually

in the form of ring-like calcifications in the flank rather than as a palpable mass.

In the past a multicystic dysplastic kidney was removed surgically. Recently, however, a nonsurgical approach has been advocated. Approximately two-thirds of cases will demonstrate a spontaneous decrease in size on follow-up studies. Only 10% of cases will increase in volume which may require surgical removal.

RADIOLOGY

The radiologic findings of multicystic dysplastic kidney vary for the following reasons: (a) the patient's age; (b) whether the multicystic dysplastic kidney results from pelvoinfundibular atresia or from severe hyponephrosis; (c) whether the disease is unilateral or bilateral; and (d) whether involvement is segmental or total. In the neonate, an abdominal radiograph demonstrates a large, noncalcified abdominal mass that displaces bowel gas and frequently crosses the midline. Most cases of multicystic dysplastic kidney discovered in the older child or the adult will have one or more ring calcifications in the region of the kidney and no evidence of a functioning kidney.

The sonographic findings of multicystic dysplastic kidney with pelvoinfundibular atresia include cysts that vary in size and shape, with the largest cyst being peripheral in location; absent communication between adjacent cysts on a good-quality real-time study, absent renal parenchyma between cysts, absent central sonolucency corresponding to a renal pelvis and echogenic areas which represent primitive mesenchyme or tiny cysts in an eccentric location. Hydronephrotic multicystic kidney demonstrates numerous peripheral cysts and a large medial fluid-filled structure corresponding to the dilated renal pelvis. The peripheral cysts may communicate with each other or with the renal pelvis. A

reniform shape is preserved to some extent.

In the most severely affected cases, radionuclide studies demonstrate a photon-deficient mass in the renal fossa reflecting absent perfusion. When sufficient mature renal parenchyma is present between cysts, renal function may be demonstrated on delayed images.

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RADIOLOGICAL DIAGNOSIS OF MAXILLOFACIAL INJURIES

Angelo M. DelBalso, M.D.

I. OVERVIEW OF FACIAL FRACTURES

A. Classification of facial fractures

1. isolated
   - a. limited to a single facial structures
   - b. usually the result of a low velocity forces
   - c. commonly isolated fractures include: nasal fractures, tripod, and orbital blow fracture
2. complex
   - a. involve several facial structures
   - b. usually the result of high velocity forces
   - c. includes the Le Fort and nasoethmoid fracture complexes
   - d. Isolated (low velocity) injuries

B. Nasal fractures

1. commonest low velocity injury resulting in a disruption of the nasal arch and/or nasal bones.
2. plain film views used in assessing nasal fractures:
   - a. Waters
     - (1) useful in assessing the status of the nasal arch, nasal septum, and in evaluating structures contiguous
     - (2) structure e.g. lamina papyracea, inferior orbital rim, nasomaxillary articulation
     - (3) hematomas of the soft tissues overlying the nasal arch as well as septum
   - b. bilateral nasal bone view
     - (1) provides information concerning displacement of the nose
     - (2) diastasis of nasofrontal articulation
     - (3) avulsion of lateral nasal cartilages
     - (4) fractures of the nasal spine of the maxilla
   - c. superior inferior view
     - (1) best obtained using dental occlusal film
     - (2) demonstrate lateral displacement of the nasal arch
     - (3) can be used in assessing naso-maxillary articulation
3. computed tomography
   - a. not routinely indicated except when one is concerned about fractures in adjacent structures e.g. naso-ethmoid fracture complex
   - b. nasal fractures are routinely demonstrated on CT studies done for complex facial fractures, and when present the radiologist should adequately and appropriately describe the fractures

C. Zygomatic injuries
  1. anatomy of the zygoma
    a. divided into a central body and two major processes - frontal and temporal processes
    b. five sutural articulations with adjacent structures
      (1) zygomatic process of the temporal bone at the zygomatico-temporal suture
      (2) zygomatic process of the frontal bone at the zygomatico-frontal articulation
      (3) greater wing of the sphenoid bone at the zygomatico-sphenoid articulation in the lateral orbit
      (4) medial maxilla
        (a) inferior orbital rim, orbital floor, lateral wall of maxillary sinus, and maxillary alveolus
        (b) zygomatico-maxillary ariculation is often weakened as result of pneumatization of the medial aspect of the body zygoma by the adjacent maxillary sinus (lateral recess of the maxillary sinus.
  2. zygomatic injuries
    a. isolated
      (1) zygomatic arch injuries
      (2) tripod
    b. part of a complex midfacial fracture
      (1) Le Fort II (zygomatico-maxillary articulation
      (2) Le Fort III (both zygomatico-frontal and zygomatico-temporal sutures and/or zygomatic arch)
    b. zygomatic arch fractures
      (1) usually the result of a direct blow to zygomatic arch, often time resulting a comminuted depressed fractures of the zygomatic arch only. The remainder of the zygoma is not involved
      (2) can result in impingement on coronoid process of mandible leading to limitation of mandibular movement
      (3) imaging of zygomatic arch injuries
        (a) Waters view may demonstrate depression of the zygomatic arch resuling in the "bright band sign"
        (b) submental vertical view will demonstrate depression of the zygomatic arch as well as impingement on the coronoid process
    c. Tripod fractures
      (1) second most common isolated facial fracture
      (2) results from a horizonatally direct blow

to the zygoma resulting in disruption of its main articulations
(a) zygomatico-temporal - zygomatic arch
(b) zygomatico-maxillary articulation usually medial to the infraorbital foramen. Fracture line is circumferential and will involve the orbital floor, anterior and postero-lateral wall of the maxillary sinus
(c) zygomatico-frontal articulation usually at the level of the zygomatico-frontal suture
(d) zygomatico-sphenoid articulation in the lateral aspect of the orbit between zygomatico-frontal articulartion and inferior orbital canal

(3) diagnostic imaging of tripod fractures
(a) plain films
i) Waters view
a) provides best overview for demonstrating tripod fractures. It best defines the zygomatic arch on a frontal projection and provides best visualization of fratures involving the lateral maxillary sinus, inferior orbital rim, and floor of orbit.
ii) Caldwell
a) provides an undistorted view of the frontal process of the zygoma and lateral orbit, and best defines injuries involving the zygomatico-frontal and zygomatico-sphenoid articulation
iii) submento-vertical view
a) best defines fractures and diastases involving the zygomatic arch, impingement on coronoid process, and posterior displacement of the zygoma
b) computed tomography
i) computed tomograph in axial and if possible direct coronal plane is recommended in cases of tripod fracture not only to fully assess the actual tripod injury but also to evaluate the

orbit especially the globe and apex

d. Orbital fractures
  (1) osseous orbital anatomy
    (a) the apex, floor, roof, lateral and medial walls, and base (or orbital rim)
    (b) areas of inherent weakness in the osseous orbit
    (c) lamina papyracea and orbital floor
e. mechanisms of orbital injuries
  (1) direct trauma
  (2) extension of fractures involving adjacent structure
  (3) hydraulic mechanism
f. Orbital blow out fractures
  (1) orbital floor
    (a) third most common isolated facial injury
    (b) occurs primarily through a hydraulic mechanism
    (c) preservation of inferior orbital rim
    (d) can also be associated with medial blow fractures (20 to 50 percent)
    (e) diagnostic imaging of orbital floor blow out fractures
      i) Waters view
        a) demonstrate increased distance between the palpable orbital rim and orbital floor (> 2 mm) when compared to the non-injured side
          a) a hanging osseous fragment "trap door" sign
          b) soft tissue density on the roof of the maxillary sinus
          c) absence of orbital floor
      ii) Caldwell view
        a) demonstrate the actual fracture site
      iii) lateral oblique view
        a) demonstrate minimal, non-displaced fractures
      iv) Towne view
        a) demonstrate the relationship of the fracture to the inferior orbital fissure
    (a) Computed tomography
      i) direct coronal CT imaging
      ii) provides the best demonstration of the lateral and medial extent of the fracture
    (b) direct oblique sagittal projection
      i) provides the best demonstration of

the anterior and posterior extent of the fracture

(c) axial CT section

i) does not optimally demonstrate blowout fractures

ii) most common finding is a prominent bone fragment in the maxillary sinus with preservation of the inferior orbital rim

iii) axial CT studies are useful in assessing the status of the globe and orbital apex

(2) medial (lamina papyracea) blow out fractures

(a) exact incidence of isolated medial blow out fractures is not known; however, often seen in association with orbital floor blow out fractures (20 to 50 percent of cases

(b) imaging of medial blow out fractures

i) Caldwell view

a) disruption of the lamina papyracea

b) opacification of the adjacent ethmoid air cells

c) presence of intraorbital air

(c) computed tomography

i) best demonstration of the full extent of the injury

ii) findings are identical to those noted on Caldwell view

II. Complex Facial Fractures

A. Le Fort system

1. most widely used system for classifying midface fractures
2. simplified approach to the Le Fort classification is to divide the skull into three individual components:
   a. ovoid-shaped cranium
   b. pyramidal-shaped midface composed of the maxillae with the hard palate being the base of the pyramid and the nasofrontal region being the apex of the pyramid
   c. bilateral paired zygomas
   d. all Le Fort fractures must involve the pterygoid plates
3. Radiographic evaluation of Le Fort fractures
   a. plain films
      (1) lateral view important in assessing
         (a) pterygoid plates
         (b) anterior walls of the maxillary sinus

(c) naso-frontal articulation
(d) orbital roofs
(e) anterior and posterior tables of frontal sinus
(e) presence of open bite

(2) Waters view useful in assessing:
(a) zygomatic arhes
(b) zygomatico-maxillary articulation
(c) medial orbital walls (lamina papyracea)
(d) nasofrontal articulation and glabella
(e) zygomatico-frontal articulation

(3) Caldwell view
(a) nasofrontal articulation and glabella
(b) orbital roof
(c) zygomatico-frontal articulation

4. Le Fort fractures

a. Le Fort I

(1) shearing off hard palate/lower maxilla (base of the midfacial pyramid)

(2) Radiographic Evaluation
(a) lateral view
i) disruption of pterygoid plates-posterior wall of maxillary sinus (pterygomaxillary buttress)
ii) fractures of the anterior walls of maxillary sinus
iii) displacement of hard palate
iv) open bite
(b) Waters and Caldwell viewss
i) opacification of maxillary sinuses
ii) preservation of inferior orbital rims, naso-frontal and zygomatico-frontal articulations

b. Le Fort II

(1) commonest Le Fort fracture
(2) separation of the pyramid shaped midfacial skeleton from the remainder of the skull
(3) zygoma remains attached to skull
(4) radiographic evaluation
(a) lateral view
i) disruption of pterygoid plates
ii) disruption of naso-frontal articulation
iii) depression and/or displacement of lower third of face
(b) Waters and Caldwell views
i) fractures involving naso-frontal articulation, lamina papyracea, inferior orbital rims, zygomatico-maxillary articulations
ii) preservation of zygomatico-frontal articulations

c. Le Fort III
   (1) most severe of the Le Fort injuries
   (2) also known as a "cranial-facial separation"
   (3) zygomas are completely separated from cranium
   (4) radiographic evaluation
      (a) lateral view
         i) disruption of pterygoid plates
         ii) depression and/or displacement of midface
         iii) fractures in region of naso-frontal articulation
         iv) fractures of orbital roof
      (b) Waters view
         i) fractures of zygomatic arches
      (c) Waters and Caldwell views
         i) fractures in region of naso-frontal articulation
         ii) fractures of lamina papyracea
         iii) fractures or orbital roof or floor
         iv) fractures or diastasis in region of zygomatico-frontal articulation
   (5) Computed tomography with 3-D reconstruction is the preferred imaging modality of choice in the evaluation of all complex midfacial fractures
      (a) naso-ethmoidal (naso-orbit ethmoid) complex
         i) result from a blow to midface
      (b) depending on area of greatest injury classified as being
         i) naso-ethmoid fracture
         ii) naso-ethmoid maxillary fracture
         iii) naso-ethmoid frontal fracture
   (6) diagnostic imaging of naso-ethmoidal fractures
      (a) plain films
      (b) variable depending on point of impact
         i) depression of midface
         ii) nasal arch disruption
         iii) fracture of lamina papyracea
         iv) fracture of inferior and/or superior orbital rims
         v) fracture of orbital roof and/or floor
         vi) fractures of frontal and/or maxillary sinuses
      (c) computed tomography
         i) essential in evaluating naso-ethmoidal fractures
         ii) same findings as plain films

LOCKER ROOM RADIOLOGY: SKELETAL INJURIES IN SPORTS

LEE F. ROGERS, M.D.

I. Baseball

A. Thrower's fracture of humeral shaft.

B. Elbow - Traction stress medical-impaction lateral.

1. Little leaguer's elbow - separation of medial epicondyle due to pitching.
2. Osteochondral fracture of capitellum and radial head.
3. Chronic stress in professional.

a. Osseous hypertrophy.
b. Overgrowth medial epicondyle.
c. Traction spur on ulna.
d. Free bodies in joint.

C. Shoulder in pitcher.

1. Widening of lateral aspect prox humeral growth plate in adolescent pitcher.
2. Stress fracture first rib.
3. Infraglenoid spur of scapula at triceps insertion.
4. Rotator cuff tears - impingement syndromes.
5. Vascular compression syndromes.

D. Fingers

1. Subject to hyperextension.

a. Baseball finger - fx base of distal phalanx.
b. Volar plate avulsion - fx base of mid or prox phalanx at 1 P joint - may be minute fragment.

II. Track and Jogging

A. Stress Fracture

1. Disruption of bone caused by repeated application of stress.

2. Originally recognized in military recruits by Breithaupt, Austrian Military Surgeon, in 1856, the "March" fracture.

3. Stress is relative - force vs. strength. The greater and the more repetitious the force and the weaker the skeleton, the more likely a stress fracture. May occur in conditioned athlete or beginner.

   a. Conditioned athlete - exceptional exertion or increase in training.
   b. Normal individual - hiking or running.
   c. Osteoporotic female - five days christmas shopping.
   d. Joint replacement or rheumatoid arthritis - ambulation.

4. Cyclical nature of symptoms - characteristic.

   a. Stress - Pain - Rest - Relief, pain created by the stress is relieved by rest.

5. Location

   a. Primarily in lower extremity.

      (1) Running, jogging, ballet dancers.
      (2) Foot:

         (a) Metatarsal
             Neck of 2nd and 3rd.
             Base of 1st, 4th, and 5th.
         (b) Hindfoot
             Calcaneus
             Navicular

(c) Tibia
Proximal third - posterior.
Mid shaft - anterior - short horizontal lucencies confined to cortex.
Lower third.
Plateau
(d) Fibula
Proximal or distal third.
(e) Femur
Mid or distal third - medial cortex.
Femoral neck - basicervical
(f) Inferior Public Ramus

b. Rare in upper extremity

(1) Throwing sports and weightlifting.
(2) Involve 1st rib - mid portion.
(3) Mid shaft of ulna in weightlifters and volleyball players.
(4) Scaphoid stress fracture reported in gymnasts.

c. Spine - Pars interarticularis stress fracture reported in gymnasts and American football linemen.

III. TENNIS

A. No x-ray findings in tennis elbow.

B. In professional findings similar to baseball pitchers

IV. WEIGHTLIFTING

A. In adolescent - separation distal radial epiphyseal.

B. May cause compression fracture spine.

V. ARMWRESTLING

A. Fracture of humeral shaft.

VI. BICYCLING

A. Children who fall from bikes may injure knee.

1. Often anterior cruciate avulsion of ant. tibial spine.

VII. GOLF

A. Fx posterior ribs.

B. Fx hook of hamate.

1. Also seen in raquet sports and baseball.
2. Obscure - impossible to see on routine films.
3. May see on carpal tunnel view.
4. CT best method.

THE MULTIPLE INJURED PATIENT

I. DIAGNOSTIC ERRORS AND OVERSIGHTS ARE COMMON

A. Reasons
1. Patient Unconcious and Uncooperative.
2. Difficult to perform physical examination.
   a. Chest.
   b. Pelvis.
   c. Thoracic and Lumbar Spine.
3. Distraction by pressing needs of patient.
   a. Establishment of airway.
   b. Treatment of hemorrhage and shock.

B. To Prevent Errors and Oversights
1. Look for and exclude presence of common injuries encountered in multiple injured patient.
2. Establish diagnostic routine.
3. Be aware of common associations of injury.

II. COMMON MISSED INJURIES IN THE MULTIPLE INJURED PATIENT

A. Chest
1. Ruptures of the Aorta
2. Myocardial contusion
3. Ruptures of the diaphragm

B. Abdomen
1. Ruptures of Solid Organs
   a. Liver
   b. Spleen

C. Skeletal
1. Most injuries missed centered at joints.
   a. Often joint proximal to obvious distal injury.
   b. Deformity attributed to distal injury and proximal injury overlooked.
2. Fracture of Pelvis
3. Dislocation of hip.
   a. Unconscious patient
   b. Association with fractures of femoral shaft or tibia and fibula
4. Spine Injury
   a. Injury at cervicothoracic junction
   b. Discontiguous, second level fracture of spine in spinal injury patient.

III. RADIOGRAPHIC EXAMINATION IN MULTIPLE INJURED OR UNCONSCIOUS PATIENT

A. Traumatic Survey
1. Minimum examination
   a. AP Chest

b. AP Abdomen
c. AP Pelvis
d. AP and Lateral Cervical Spine

2. If unconscious or head injury add:
   e. AP and Lateral Skull
3. Obvious Extremity injury add:
   f. AP and Lateral of Extremity
4. If Spinal cord injury add:
   g. Lateral Thoracic and Lumbar spine

B. In shaft fractures
   1. Must examine from joint above to the joint below
      a. Dislocation of hip associated with femoral shaft fracture
      b. Monteggia fracture
      c. Galliezzi fracture

C. Extremity Injuries require a minimum of two views.
   a. Never accept single view as adequate

D. In examining cervical spine - must visualize 7th cervical vertebra.
   a. Fracture of C_7 is common
   b. May require swimmer's view
   c. Failure to visualize C_7 most common error in radiographic evaluation of spine.

IV. CLINICAL CAMEOS - THE COMMON ASSOCIATION OF INJURIES

A. The presence of one injury should suggest the possibility of the other.

B. Bone and Bone
   1. Fx os Calcis - Fx Thoracolumar Spine
   2. Fx Femoral Shaft - Fx or Fx Dislocation of the Hip
   3. Fx Anterior Arch Pelvis - Fx About or Dislocation of SI Joint
   4. Fx Chest Wall - Fx Scapula
   5. Fx Ulnar Shaft - Dislocated Radial Head
   6. Fx Radial Shaft - Dislocated Distal Radial Ulnar Joint
   7. Colles Fx in Young - Fx Scaphoid
   8. Colles Fx or Fx Humeral Neck in Elderly - Fx Hip

C. Bone and Vascular
   1. Fx Distal Third Femur - Femoral Artery
   2. Ant. or Post. Disl. Knee - Popliteal Artery
   3. Supracondylar Fx Humerus in Child - Brachial Artery
   4. Fx Pelvis - Pelvic Arterial Tree
   5. Fx Sternum - Myocardial Contusion
   6. Fx Ribs 1,2,3 - Rupture Aorta

D. Bone and Visceral
   1. Fx Pelvis - Rupture Bladder or Urethra
   2. Fx Lower Ribs - Laceration Liver, Spleen, or Kidney
   3. Fx Pelvis - Rupture Diaphragm
   4. Chance Fx Spine - Rupture Mesentery or Small Bowel

V. SPECIFIC INJURIES

A. Traumatic Rupture of Aorta
   1. Radiographic signs
      a. Widened mediastinum - particularly in young patient.
      b. Poorly defined margin of aortic arch.
      c. Displacement of trachea to left.
      d. Depression of left main stem bronchus.
   2. Differential Diagnosis
      a. Venous bleeding
      b. Mediastinal hematoma associated with upper thoracic spine fracture.
   3. Aortogram required for diagnosis.

B. Rupture left hemidiaphragm
   1. Associated with pelvic fracture
   2. Radiographic Signs
      a. Apparent elevation of diaphragm
      b. "Air pocket" in lower left hemithorax. ("Air pocket" represents air in herniated stomach.)
   3. In 90% stomach herniates through central tear in diaphragm.
   4. Course of nasogastric tube helpful.
      a. Esophageal hiatus not involved in tear.
      b. NG tube passes through esophageal hiatus and then coils upward into stomach ("air pocket").

ANGIOGRAPHIC INVESTIGATION OF PENETRATING VASCULAR INJURY IN THE NON-RENAL ABDOMINAL AND PELVIC EXTRAPERITONEUM

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The most common extraperitoneal vascular injuries diagnosed and treated by radiologists are renal, of which penetrating renal injuries are seen at the highest frequency, half of them from street trauma. Penetrating street trauma to the abdominal aorta, and to the non-renal extraperitoneal arteries—— lumbar, inferior phrenic, and internal iliac, and their branches——are much less common.

Given the great difficulty of identifying extraperitoneal vascular injuries by physical examination and laboratory studies, their diagnosis is usually made on the operating table. However, because some of these injuries may be missed at laparotomy even in the hands of the best surgeons, there is a place for either preoperative or postoperative radiologic diagnosis.

Why Angiography?

Whereas CT is the preferred initial examination for blunt renal and other extraperitoneal trauma, *provided the patient is not in shock,* those with penetrating injuries are far better diagnosed by angiography: because, in penetrating trauma, we are looking for vascular injuries and hemorrhage——the domain of diagnostic and interventive angiography——rather than for parenchymal injuries and hematomas, it is in the best interest of the patient not to waste time and contrast media on CT. Certainly, the hemodynamic instability which so often characterizes these injuries, mandates that their diagnosis be made by early and aggressive angiographic exploration——the only radiologic modality which allows the radiologist immediate access to control of hemorrhage.

Angiographic control of hemorrhage is not dealt with here unless absolutely relevant for the discussion. However, several sources are listed for interested readers.

Abdominopelvic Gunshot Wounds

The relatively high mortality (over 20%) in abdominopelvic gunshot wounds is most frequently attributed to uncontrolled hemorrhage or to missed intestinal perforations; hence the mandatory requirement for surgical exploration of all abdominopelvic GSWs. Still, even in best trauma surgeons may overlook injuries, especially in the extraperitoneum, given the complexity and unpredictability of the residual wound tract—or tracts—and the difficult surgical access to certain regions harboring some very critical injuries. Here again the angiographer may be required to assist in both diagnosis and management of vascular injuries and, although angiography is not routinely necessary prior to laparotomy, unless specific preoperative information or intervention mandates it, an **exploratory angiogram** is highly desirable immediately following an especially messy operation for complex wound tracts. The angiogram should be performed as soon as surgery has been completed—or temporarily suspended—while the patient is still under general anesthesia. Angiographic exploration must always begin with a high midstream abdominal aortogram on which the last 10cm of the thoracic aorta are clearly seen. Moreover, since the purpose of the study is to find injuries which were missed at operation *because they were not suspected,* the radiologist must not allow the surgeon to interfere in the procedure or direct it in any way. Indeed, this author has observed that the most successful outcome of patient management is often accomplished when the surgeon leaves his ego in the operating theatre and presents the patient to the radiologist with a request to explore, find, and if possible treat all injuries missed at operation.

Penetrating Trauma to the Abdominal Aorta

Severe and sometimes through-and-through penetrations of the abdominal aorta may be missed during the initial laparotomy, given the need to close the abdomen as soon as possible because of circulatory collapse, hypothermia, and coagulopathy. Patients may tolerate untreated penetrating injuries to the abdominal aorta for a length of time which cannot be predicted. Therefore, a postoperative exploratory

angiogram should be done within hours of the first laparotomy, certainly prior to the "second-look" operation. Also, a penetrating injury to the abdominal aorta may initially be so well controlled by the surrounding tissues that there will be little if anything to see at laparotomy unless the surgeon presumes proximity injury to the aorta and executes the Mattox maneuver to thoroughly explore the whole course of the aorta.

Penetrating Trauma to the Lumbar Arteries

Lumbar arterial hemorrhage—usually the result of stabbing or of blunt trauma—is, definitely, best diagnosed and treated angiographically. Any attempt to make the diagnosis by surgical techniques may be morbid or, sometimes, lethal, since the operative access to the lumbar arteries is most difficult and can be very mutilating, irrespective of the direction of the surgical approach: considerable risk of injury to the ureter exists in the transabdominal and even the lateral-extraperitoneal approach, and to the lumbar plexus in approach from any direction. In addition, because of the often-present hypothermia and coagulopathy, the patient may not tolerate an operation at all. In blunt trauma especially, lumbar arterial hemorrhage defies diagnosis in that too many patients undergo needless exploratory laparotomy which, almost invariably, not only fails to discover the source of bleeding but may aggravate the hemorrhage. Lumbar arterial hemorrhage can also be part of an overall exsanguinating hemorrhage in patients—especially the very old or those with severe brain injury—following extremely violent blunt trauma.

Penetrating Trauma to Other Extraperitoneal Arteries

The internal iliac arteries and their branches are at risk from gunshot wounds to the pelvis and perineum. These are often very difficult to approach surgically and are, as in pelvic fractures from blunt trauma, best diagnosed and treated by the radiologist.

Rarely, hemorrhage from the inferior phrenic, adrenal, pancreaticoduodenal, or inferior epigastric arteries is acutely diagnosed and treated at angiography. In most events these are seen by the angiographer after having been missed at the time of laparotomy, or else as postoperative complications.

Summary and Conclusions

Posttraumatic arterial hemorrhage in the abdominal and pelvic extraperitoneum is counted among the injuries least amenable to diagnosis by physical examination and the common laboratory tests. Moreover, surgical exploration of such patients not only is ineffective, but can be mutilating and—not infrequently—lethal. Angiography is the ideal tool for diagnostic exploration of the extraperitoneum, as well as for effective control of arterial hemorrhage in most extraperitoneal injuries. Angiography and embolization, performed judiciously in the immediate post-impact course, can be instrumental in reducing morbidity, preserving organs, and lowering mortality.

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ANGIOGRAPHIC EVALUATION OF BLUNT CHEST TRAUMA

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Laceration of the thoracic aorta is almost invariably a fatal injury. Over 80% of victims die on impact and/or up to 24 minutes from arrival at the hospital, and even those who live long enough to undergo thoracotomy for this injury have a 50% survival rate. Physical findings on admission are non-specific, often misleading, and could be entirely absent.

Thoracic aortic injuries are caused by a wide variety of blunt mechanisms: the "classic" unrestrained frontal impact to a car-occupant is responsible for only 43% of injuries; 57% are caused by broadside collisions, motorcycle and aircraft crashes, pedestrian accidents, falls, equestrian, and other mishaps.

The rapid lethality of the injury——untreated, 98% of patients will die——calls for a high index of suspicion in all events of violent blunt trauma to the chest, irrespective of physical findings; the presence of pelvic fractures must further heighten the suspicion since in patients with pelvis fractures from anteroposterior compression the likelihood of rupture of the aorta is 9 times greater than in the general trauma population.

preliminary radiologic diagnosis

The chest film offers only a limited amount of gross information regarding the mediastinum and no information at all regarding vascular injuries, and none of the roentgen signs associated with mediastinal hematomas have statistically predictive significance either in confirmation or in refutation of the diagnosis of ABC injury. Therefore, in the presence of even the smallest mediastinal hematoma in a patient after violent blunt impact to the chest, emergency aortography is necessary.

> ***Caveat:*** *since the chest film contains no reliable information to begin with, repeating it at regular intervals will not create more information and may actually contribute the patient's early death.*

Other imaging modalities, especially CT and TEE, currently miss too many aortic and brachiocephalic injuries to be completely reliable, and also have a high rate of false-positive readings. Still, it is possible that, if and when they are accomplished, future improvements in both speed of acquisition and image resolution of CT and TEE may indeed make either or both at least as reliable as aortography——i.e. surgeons will decide whether or not to operate solely on the basis of the non-invasive images——in which event they will replace invasive angiography.

> ***Caveat:*** *until this happens, the introduction into the diagnostic process of CT and TEE must be regarded as very hazardous since it will delay the definite diagnosis and result with increased mortality.*

angiography

Access: femoral or right axillary, via F-7 introducer sheath. Catheter: F-7 pigtail (Royal Flush by COOK, Bloomington, Indiana), capable of administering 50 cc/sec. Guidewire: 0.038", Teflon-coated, LLT, 2mm-J. Projections: RPO at 40°-to-45°, LPO at 45°-to-50°. Contrast medium for films: Nonionic, 370 mgm iodine per 1 ml, straight up, 50 ccs total, injected at 50 cc/sec. Film program: 4/sec for 2 seconds, 1/sec for 4 seconds. Contrast medium for DSA: Nonionic, 370 mgm iodine per 1 ml, diluted with saline to 25%, 50 ccs total, injected at 50 cc/sec. Imaging sequence: 4-6 images/sec until veins are seen.

> ***Caveat:*** *incomplete aortograms and poor angiographic techniques are constant sources of diagnostic errors, especially false negatives.*

the injuries

Any point may be injured along the course of the thoracic aorta (82.3%) and brachiocephalic arteries (17.7%). Most aortic injuries—73.0%—involve the isthmus, less so the ascending (4.8%), arch (15.9%), or descending (6.3%) parts of the aorta. Most brachiocephalic artery trauma—59.3%—is to the subclavians, less so to the innominate (29.6%) and carotids (11.1%).

typical injuries:

Of the many mechanisms of injury and the aortic/brachiocephalic they produce, I have identified five for which the causal relationship between the wounding mechanism and the injury is fairly well established.

1. *Frontal crashes of cars and motorcycles: Isthmic injury* with one or more transection lines involving the intima and media, producing a circumferential but incomplete transection with a subadventitial collection of contrast medium (false aneurysm). Sometimes, what seems to be a well-contained false aneurysm is indeed a completely transected aorta held together by the pleura.

Mechanism of injury: a combination of cephalad stretch of the aorta, compression of the heart and sudden evacuation of its contents into the aorta which has been pinched distally by the taut diaphragmatic ligament. Result: sudden high pulse pressure——water hammer effect——which blows the isthmus, since the isthmus is the weakest segment of the thoracic aorta. Rupture of the aortic isthmus in a frontal collision is a random event, requiring that the impact will coincide with the end-diastolic segment of the cardiac cycle——i.e. a full heart. This explains why, of all victims of virtually identical frontal car collisions, less than 15% will sustain isthmic lacerations.

2. *Broadside collisions: Distal arch injury* with a short, incomplete transverse laceration at the lesser curvature of the arch immediately above the isthmus. The accompanying false aneurysm is usually quite small and may be seen only on the LPO projection. The surgeon must be warned that the proximal location of this laceration leaves a shorter aortic segment with which to work.

Mechanism of injury: a lateral shearing force, directed from either left or right. In contrast with frontal impacts, where the mechanism is primarily a hemodynamic blowout, broadsides are purely mechanical in nature.

3. ***Seatbelt injury: Brachiocephalic injury*** with partial wall laceration or incomplete transection of the innominate or subclavian artery.

Presumed mechanism of injury: direct blow to the subclavian, or a sudden stretching of the innominate artery by the diagonal component of the seatbelt.

4. ***Contact wounding by a fractured sternum: Innominate artery avulsion.*** A rare injury in which the origin of the innominate artery from the arch is partially torn, creating a small false aneurysm, or the wall injury dissects distally and completely occludes the artery as well as its carotid and subclavian branches.

Mechanism of injury: Inward-buckle of a transversely fractured sternum (in the great majority of cases a fractured sternum buckles outwards).

5. ***Crushing of the chest.*** Survivors of violent crushing of the chest show mostly trauma to the brachiocephalic arteries and the top of the aortic arch, sometimes with partial avulsion of the innominate artery from the arch. Rarely, and depending on the direction of force, the descending aorta may be crushed.

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MRI

CNS Ischemic Disease

Conventional MRI

- Occlusive ischemia.
- Nonocclusive ischemia.
- Venoocclusive disease.

New Techniques

- Anatomic CBF.
  - » MRA
- Functional CBF.
  - » exogenous marker
  - » endogenous marker
- Metabolic.
  - » spectroscopy

Stroke

- Annually 500,000.
- Third leading cause of death.
- Stroke related death.
  - » decreased 50% in last three decades

Other CNS Pathology

- Primary brain tumors.
  - » 33 times (15,000)
- Demyelinating disease.
  - » > 100 times
- Trauma.

Stroke - MRI

- Infarcts at 2 hours in lab setting.
- Usual clinical conditions - 12 hours.
- On T2-weighted sequences (long TR).
  - » 80% positive at 24 hours
- Immediate vascular enhancement.

Stroke - Functional

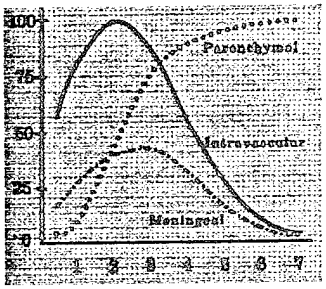
- Usually immediate.
- Usually larger than T2 region.
- Penumbra.

Immediate

- Loss of "flow void".
- Intravascular enhancement.
  - » 75-100% at 48 hours
- Meningeal enhancement.
  - » 30% at 48 hours

Enhancement

- Intravascular 2 days.
- Meningeal similar.
- Delayed parenchymal.



Early - T1

- 12 hours.
- Anatomic.
  - » sulcal effacement
  - » gyral edema
- Loss of G-W interface.
  - » equivalent of loss of insular ribbon

Early - T2

- 12 hours.
- T2 hyperintensity.
- Early mass effect.

Early Subacute

- 1 - 3 days.
- Prominent T2 changes.
- Mass effect.
- Parenchymal enhancement.
  - » rule of 2/3's

Subacute

- 1/2 to 1 week.
- Maximal mass effect.
- Marked parenchymal enhancement.
- Loss of intravascular and meningeal enhancement.
- Hemorrhagic changes.

Late Subacute

- 1 week to 3 months.
- Parenchymal enhancement.
- Resolving mass effect and T2 changes.

Chronic

- Greater than 3 months.
- Encephalomalacia.
- Hemosiderin/ferritin.

Role of Contrast

- Intravascular.
- Meningeal.
- Parenchymal.

Adjuncts

- Magnetization transfer.
- Higher contrast doses.

Enhancement

- CBV has little contribution.
- Interstitial volume > 10X CBV.
- Initially loss of BBB.
  - » up to one week
- After one week.
  - » granualtion tissue
  - » immature proliferating endothelial cells

Occlusive Ischemia

- Atherosclerosis.
  - » trials evaluating carotid bifurcation disease
  - » skull base/arch
  - » Tandem lesions - 2%

Occlusive Ischemia

- Dissection.
- Vasculitis.
- Vasculopathy.
- Extrinsic compression.
- Vasospasm.

Symptomatic Trials

- NASCET.
  - » 70 - 99% stenosis
  - » Alan Fox
- ECST.
- VA.

Asymptomatic Trials

- ACAS.
  - » $\geq$ 60%
  - » male > female
  - » Bruce Dean
- VA.

Stenosis

- Decreased pressure at 60%.
- Decreased flow at 90%.

Stenosis

- ACAS results.
- Indicates need for screening.
  - » male > female
  - » MRA
  - » doppler

Dissection

- **Carotid.**
  - » cervical
  - » skull base

Dissection

- **Vertebral.**
  - » C2 to skull base
  - » origin to C6 less common

Dissection

- **MR perivascular halo.**
- **Angio may be normal.**
  - » subadventitial

Dissection

- **Trauma.**
- **FMD.**
- **Iatrogenic.**
- **Spontaneous.**
- **Vasculopathy.**
- **Atherosclerosis.**

Vasculitis

- **Infectious.**
- **Noninfectious.**
  - » immune complex
    - – lupus
    - – polyarteritis
  - » cell mediated
    - – granulomatous
    - – temporal arteritis

Vasculitis

- **Miscellaneous.**
  - » drug
  - » Behcet's
  - » Buerger's
  - » Kawasaki

Vasculopathy

- Fibromuscular dysplasia.
- Radiation.
- Moya moya.

Vasculopathy

- Hypertension.
- Neurocutaneous syndromes.
  - » NF 1
  - » Sturge-Weber

Extrinsic Compression

- Trauma.
- Nasopharyngeal mass.
- Meningioma.

Vasospasm

- SAH.
- 3 - 10 days.
- Radiographic findings may be reversible.

Nonocclusive Ischemia

- Hypotension.
- CO poisoning.
- Metabolic abnormalities.
- Hypoglycemia.

Stroke Features

- Gyriform enhancement.
- Wedge-shaped involvement.
- "Tincture of time".

Stroke Mimics

- Tumor.
- Encephalitis.
- Status epilepticus.
- Toxemia.
- Malignant hypertension.

Stroke Children

- CHD.
- Dissection.
- Infection.

Stroke Children

- Blood Dyscrasias.
- Radiation.
- Sickle cell.
  - » small and large vessel
  - » endothelial injury

Stroke Neonate

- Edematous brain.
  - » "reversal sign"
  - » decreased G-W distinction
  - » cerebellum less involved

Stroke Neonate

- Cortical laminar necrosis.
- Boundary zones.
  - » periventricular white matter
- Encephalomalacia.
  - » multicystic
  - » mineralization

Wallerian Degeneration

- Initial axonal degeneration.
  - » MR negative
  - » myelin intact
- Myelin involvement at 3 - 4 weeks.
  - » T2 shortening
  - » decreased signal T2-WI

Wallerian Degeneration

- 3 - 6 months.
  - » increased on T2-WI
- 6 - 18 months.
  - » increased on T2
  - » volume loss

Venous Occlusion

- SSS is most common.
- ICV/deep system is usually catastrophic.

Cerebral Venoocclusion

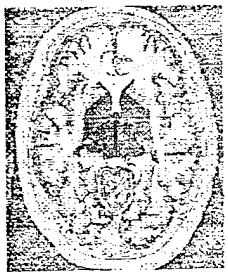
- Not arterial distribution.
- Hemorrhage at G-W junction.

Cerebral Venoocclusion

- Infection.
- Trauma.
- Hypercoagulable.
- Dehydration.
- C-V disease.
- Idiopathic.

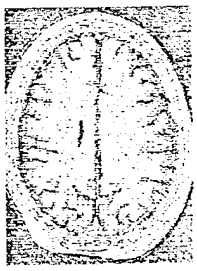
Vascular Territories

- Perforating vessels.
- ACA, MCA, PCA.



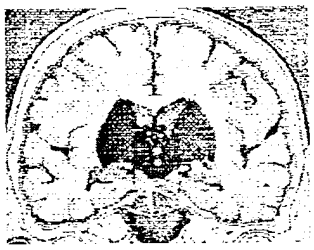
Vascular Territories

- Boundary zones.
- Triple zone posteriorly.



Vascular Territories

- PCA undersurface.
- Anterior choroidal.
- PCA perforators.



MRI of Sports Injuries

Bruce L. Dean

Sports Injuries

Spine.
Joints.
Soft tissues.

Spine Injuries

- 10 - 15% of sports injuries.
- .6 - 10% with neurologic findings.

MRI of Acute Spine Injuries

- Canal status.
  - » bony narrowing
  - » HNP
  - » soft tissue impingement
- Cord status.
  - » edema
  - » hemorrhage
- Soft tissue injuries.

MRI of Acute Spine Injuries

- Thoracolumbar.
  - » high speed injuries
- Cervical injuries.
  - » football
    - – fractures
    - – transient quadriparesis (stenosis)
  - » hockey
    - – 80% cervical
    - – push from behind
  - » gymnast

Chronic Changes

- Endplate irregularity.
- Wedging.
- Ski jumpers - 100%.
- Gymnasts > 50%.

Spondylolysis

- Hyperlordosis.
- Hyperextension and axial loading.
- 5% in general population.
- Some sports 4X the general population.
  - » 1/3 of football linemen
  - » jumping sports (basketball)
  - » gymnasts

HNP and Disk Disease

- Flexion/torsional motions.
- Golfers.
  - » 90% of tournament level golfers had prior cervical or lumbar injuries
- Weightlifters at atypical locations.

Joint Injuries

- Knee is most vulnerable.
- Ankle and low back are second.
- Lower leg and shoulder.

Scope of Problem

- Virtually everyone has had an injury.
- Professional athletes.
- Lost work days.

Scope of Problem

- Majority of Americans participate in sports.
- Overlap with vocational injuries.
- "Big-picture" of pathophysiology.

Acute Injuries

• Sprain	34
• Strain	23
• Contusion	13
• Fracture	10
• Tendonitis	7

High school sports

Overuse Injuries

- Comprise 30-50% of injuries.
- Tendon injuries M>F.
- Sports:
  - » American - variety
  - » Europe and South America - Soccer

Overuse Syndrome

- Achilles tendonitis/bursitis.
- Jumper's knee.
- Patellar chondromalacia.
- Iliotibial tract syndrome.

Overuse Syndrome

- Synovitis.
- Shin splints.
- Meniscal lesion.
- Knee instability.

Joint Pathophysiology

- Bones.
- Cartilage.
- Ligaments.
- Load transmitters.
  - » knee menisci
  - » TFC
  - » TM joint menisci

Bone Trauma

- Subtle fractures.
- Fractures involving articular surfaces or epiphyses.
- Stress fractures.

Bone contusions.

Bone Injuries

- Type 1.
- Type 2.
- Type 3.
- Lynch and Crues.

Bone injuries
More Serious Lesions

- Cortical infraction or impaction.
- Planes or crescents of low signal.
- More focal lesions.

"Contusions"

- Very low percentage of patients will suffer osteochondral sequelae with diffuse lesions.
- High percentage (>50%) will have osteochondral sequelae with geographic, focal lesions.

Osteonecrosis

- Hip.
  - » avascular necrosis
- Knee.
  - » insufficiency fracture of relatively "osteopenic" bone

Chondral Loss

- Iscemic basal layer of cartilage?
- Subchondral plate collapse.
- Direct injury to chondrocytes.
- Humoral factor release.

Treatment

- Animal studies.
  - » trabeculae weakest at 4 weeks
- Crues - rest 4 - 6 weeks if symptomatic.
- Sports medicine physicians - playing time guaged by symptoms.
- Empiric rest for 6 - 8 weeks.

Sequelae

- Chondral lesions.
- Loose bodies.
- Osteonecrosis.
- Degenerative.

Stress Fracture

- Fatigue/insufficiency.
- Bandlike.
- Subperiosteal and sub-Q changes.
- Don't involve articulating surface.

Cartilage

- Load transmission.
- Lubrication.
- Overloading causes cell death and chondromalacia.

Cartilage Evaluation

- T2 (long TR/TE) sequences.
- Intra-articular contrast.
- Large flip GE.
- Fat-saturated intermediate weighted scans.
- MTC.

Cartilage Evaluation

- Increased signal.
  - » edema
  - » influx of fluid
- Fissuring.
- "Crabmeat".

Acute Chondral Injury

- Limited ability to repair (pseudocartilage).
- Edema.
- Lysis.
- Fractures.

Acute Chondral Injury

- Lateral femoral condyle (basketball players).
- Weight-bearing medial condyle.
- Patella.

Chondromalacia

- Closed - grade 1.
- Fissures/ulcers - grade 2.
- Fibrillation - grade 3.
- Craters, subchondral eburnation - grade 4.

Soft Tissue Injuries

- Muscle-tendon unit injuries.
- Soft tissue contusions.

Tendons

- Transmission of muscle contraction to bone.
- Resistant to extension but flexible.
- In general, stronger than muscle.

Tendonosis

- Tendonosis is tendonosis, is tendonosis, is tendonosis.

Tendon Tears

- Achilles and patellar tendon tears - 35 to 45 age group.
- Rotator cuff similar age group, unless accelerated tendon pathology.
- Quadriceps tendon ruptures are rare.

Ligaments

- Joint stabilizers.
- Motion restriction.
- Proper alignment of loads in various ranges of motion.

ACL

- Semilunar shape.
- AMB (larger) (flexion).
- PLB (smaller)(extension).

Ligamentous Injuries

- ACL is crucial in professional athletes.
- High speed injury with hemarthrosis.
- Higher frequency in female athletes.

Ligamentous Injuries

- Untreated.
  - » DJD
  - » Secondary meniscal tears in 25%
- Shape of notch with chronic insufficiency.

ACL Tear

- Loss of continuity.
- Mass effect.
- Retraction.
- Wavy or concave.
- "Wrong" angle.

ACL - Bone Injury

- Bone injuries > 80%.
  - » usually lateral
- Segond.
- Lateral notch > 2mm.

Location ACL Tears

- Proximal - 20%.
- Midsubstance - 75%.
- Distal - 5%.
- Complete > partial.
- Skiers usually proximally.

Partial ACL Tear

- Usually AMB.
- Usually progress to complete tear.

Muscle Injuries

- Common injuries.
- Limited ability of muscle to regenerate without scar.
- Protect from further damage.
- Disruption occurs at muscle-tendon junction or adjacent muscle.

Hematomas

- Hemorrhage.
  - » intramuscular
  - » intermuscular
- Compartment syndrome.
- Severity correlates with clinical sxs.
  - » loss of motion
  - » pain
  - » swelling

Meniscal Function

- Load transmission.
- Joint stabilization.
- Shock absorption.
- Lubrication.
- Proprioception.

Meniscal Tears in Athletes

- Jumping sports - basketball.
  - » radial and parrot-beak tears.
  - » lateral compartment.
  - » junction of body and horns.
- "Bucket-handle" in young athletes.
- "Flap" tears in pitchers - pivot.

PROSTATE CANCER

PRESENT CONCEPTS IN DETECTION AND IMAGING

R.B.FINEGOLD, MD

TO SCREEN OR NOT TO SCREEN

?

Can we justify the costs of screening and intervention in older men who are likely to die of other disease?

Can we detect and treat those cancers which will result in prostate cancer death and morbidity and conservatively follow or ignore those cancers which will not?

LECTURE OUTLINE

- EPIDEMIOLOGY
- ANATOMY / STAGING
- SCREENING TESTS
  - DRE
  - PSA
- IMAGINGTESTS
  - TRUS
  - MRI
- SUMMARY

CAN WE ACCURATELY DETECT THE MAJORITY OF CLINICALLY-IMPORTANT CANCERS?

CAN WE JUSTIFY LARGE SCALE SCREENING FOR EARLY DETECTION OF PROSTATE CANCER IN ASYMPTOMATIC MEN?

"CLINICALLY IMPORTANT" CANCERS

TUMOR SIZE most closely relates to patient survival.

0.5 cc - 3.0 cc lesions are potentially curable.

AUTOPSY DATA: of men >50yrs, 60% had < 0.5cc lesions

DILEMMAS IN SCREENING

LEAD TIME BIAS: cancer detected early w/o change in cancer-related mortality.

LENGTH BIAS: pre-clinically detects slowly growing cancers. *(Prostate cancer takes ~10 years to grow to a volume of 1 cc.)*

DIGITAL RECTAL EXAMINATION

- cancers as small as 0.2cc detected.
- relatively inexpensive.
- most cancers detected are *incurable* : >60% of Stage B tumors upgraded to Stage C or D1 after surgery.
- tumor volume determination poor.

PROSTATE SPECIFIC ANTIGEN

- an immunogenic glycoprotein produced by prostate epithelial cells: normal, hyperplastic, and neoplastic.

PROSTATE SPECIFIC ANTIGEN ASSAY (PSA)

- unadjusted
- PSA density
- age-specific PSA ranges
- PSA velocity
- predicted PSA

unadjusted PSA

Normal mPSA = 0-4.0 ng/ml

Problems :

Many False (+) elevations (e.g. BPH, prostatitis)

2 out of 3 biopsies in patients with uPSA > 4.0 are *benign*.

20 - 25% of men with clinically localized CA have *normal* uPSA.

serum PSA (unadjusted)

Prostate Cancer detection:
- Sensitivity 96%
- Specificity 89%
  (improves to 95% using volume-adjusted criteria.)
- Positive Predictive Value 47%

PSA DENSITY (Benson)

Prostate weight = #1 non-cancer variable in PSA elevation.

premise: serum PSA production ∝ prostate size .

premise: PSA elevations are *10 x* greater per gram CA than per gram BPH .

PSAD = serum PSA / gland volume (TRUS)

PSAD > 0.12 = 10% probability for CA

PSAD

PROBLEMS :

- epithelial / stromal ratio varies
  ∴prostates of similar size can produce different PSAs.
- volume errors exist w ith TRUS

(volume = width x height x length x 0.52)

PSAD and uPSA

PSAD	No. of pts. with uPSA levels (ng/ml) of:		
	<u>< 4.0</u>	<u>4.0-10.0</u>	<u>>10.0</u>
< 0.10	2	0	1
0.10-0.11	0	1	0
0.12-0.13	0	3	0
> 0.14	2	23	8

--Olsen et al. (1994)

PSA velocity (Carter)

serial PSA determinations: detect those cancers with greater malignant potential by the magnitude of the rate of change in the PSA value.

cancer = ↑ PSA > 20% in 1 year
= > 0.7 ng/ml in 1 year

problem: PSA conc. do not increase until a tumor volume of 1 cc is obtained; may miss CA on "1st"

age-specific PSA (Oesterling)

premise: prostate size and PSA ↑ with age.

age 40-49	0 - 2.5 ng/ml
age 50-59	0 - 3.5ng/ml
age 60-69	0 - 4.5 ng/ml
age 70-79	0- 6.5 ng/ml

- ↑ specificity in older men
- ↑ sensitivity in younger men

AMERICAN CANCER SOCIETY NATIONAL PROSTATE CANCER DETECTION PROJECT

- multicenter multidisciplinary evaluation of the efficacy of early prostate cancer detection.
- three thousand men, age 55-70 yrs, evaluated yearly by DRE , PSA , & TRUS x 5 yrs .
- *volunteers* without hx of prostate ca or previous work-up (∴ ≠ gen. pop; but = health care setting)

ACS-NPCDP

	2011 (-) CA	171(+)CA
avg. uPSA (ng/ml)	2.1 (90% < 4.0)	12.0 (72% > 4.0)
mean gland vol (cc)	34	39
av. age (yrs)	64	66
PSAD	.06	.35

ACS-NPCDP

	SENSITIVITY	SPECIFICITY
uPSA	72%	90%
PSAD	75%	83%
agePSA	67%	91%
velPSA	55%	97%

2011 men had DRE - / TRUS - (ie. "normal")
171 men (+) prostate CA

ACS-NPCDP

Unadjusted serum PSA (nl < 4.0) was close to the most specific (velPSA) and most sensitive (PSAD) alternate PSA test.

Aging does not predict ↑ PSA levels in *normal* men.

"PREDICTED" PSA (Lee/Littrup)

- individualizes a pt's PSA relative to glandvolume.
- mPSA production per gram BPH = 0.12 ng/ml .
- predicted PSA = 0.12 x TRUS volume (e.g. =3.6ng/ml for 30cc gland)
- pobability of cancer or prostatitis > *50%* if serum PSA exceeds "predicted" PSA.

"Predicted" PSA

- functions near 95th percentile for glands < 40cc
- objectively detects 70% of cancer in only 10% of screened population.
- Predicted PSA can suggest tumor size on TRUS:
  Excess PSA = serum PSA - predicted PSA
  Potential tumor volume (cc) = $\frac{\text{excess mPSA}}{2.0\text{ng/ml/gmCA}}$
  Mean TRUS lesion diameter (cm) = $\sqrt[3]{\text{tumor vol}}$

TRANSRECTAL ULTRASOUND

- Improves positive predictive values when combined with both DRE & PSA screening *but not enough to justify* its additional cost as a screening tool.
- role: to assess prostate volume and morphology when DRE &/or PSA is abnormal.
- role: to guide prostate biopsies.

MRI Staging

Premise: surgical cure of Prostate CA is possible in > 80% of organ-confined tumors

	STAGING(locals.)		
	SENS.	SPEC.	ACCUR.
TRUS	66%	46%	58%
body coil MR	77%	57%	69%
endorectal MR	87%	85%	79%
phased array FSE	89%	77%	85%
Integrated ER-PPA	83%	98%	77%

TO SCREEN OR NOT TO SCREEN

Prostate cancer is slow-growing.

-1/ 380 men with Prostate CA dies of it.
-can be aggressive and affect younger men.
-35,000 deaths per year!

TO SCREEN OR NOT TO SCREEN

The cure is worse than the disease.

No treatment is best treatment.
(Krahn, JAMA '94)

-screening benefits offset by morbidity of treatment, lack of benefit of treatment, and high cost of screening (~$1.2 billion 1st year)

TO SCREEN OR NOT TO SCREEN

Screening detects insignificant tumors.

False.

1cc tumor volume ≈ 3.6 ng / ml uPSA.

<1cc tumor volume is non-life-threatening.

TO SCREEN OR NOT TO SCREEN

Screening is of unproved benefit.

"True" (as is mammography in women < 50yrs) yet survival curves show upward trend.

- efficacy of treatment not yet established.
- controlled studies (e.g. ACS-NPCDP) needed over 16 years or more.

TO SCREEN OR NOT TO SCREEN

Screening likely best in high risk mortality groups.

- African-American males
- (+) family history of prostate CA
- men with > 15 years life expectency

SUMMARY

- 200,000 cases Prostate CA 1994.
- 35,000 deaths annually and rising.
- Treatment options are controversial, different for localized and advanced ds, and (+) effects on survival not assured.

SUMMARY

- Screening (begining age 40-50)"
  DRE (annually)
  PSA (annually)
- TRUS: If DRE (+) or PSA (+)
  visualized lesions biopsied.
  "random" bxs
- *Continued investigations:*
  screening; staging; therapies; & efficacy.

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RETROPERITONEAL DISEASES: A PRACTICAL APPROACH

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RETROPERITONEAL ADENOPATHY

The retroperitoneal lymph nodes include 1 paraaortic, 2 paracaval, 3 intra-aortocaval, 4 renal hilar and 5 suprahilar. Abnormal lymph nodes are recognized by abnormalities in size, number or internal architecture.

CT is an excellent technique for detecting enlarged retroperitoneal lymph nodes. Accurate assessment by CT is dependent on thorough opacification of bowel loops with dilute oral contrast material. Scans should be obtained in suspended expiration using 10-mm collimation. Intravenous contrast may be used to differentiate adenopathy from vascular anomalies.

Normal lymph nodes (3-10 mm) may be seen as round or oval soft tissue density structures in the paraortic and paracaval areas. Isolated nodes measuring 1-1.5 cm are considered suspicious. Lymph nodes are definitely abnormal if they exceed 1.5 cm in cross-sectional diameter. Between the aorta and left psoas muscle, there is normally no extranodal structure larger than 5 mm. Loss of the normal lateral contour of the aorta strongly suggests paraaortic adenopathy.

Abnormal lymph nodes may be solitary or multiple and may occur in clusters or conglomerate masses. In patients with known lympho-proliferative disease, clusters of more than the usual number of normal

sized lymph nodes are usually abnormal.

Abnormal lymph nodes may be homogeneous or heterogeneous in attenuation on CT. The density of abnormal nodes may be fatty (eg lipoplastic lymphadenopathy), necrotic, tissue density, high density (67-117 H) or may contain calcification. High density nodes have been described in patients with Hodgkin disease, metastatic breast and ovarian carcinoma. Calcifications may occur in patients receiving chemotherapy or patients with testicular neoplasms.

Lymphadenopathy may be recognized as a small number of enlarged nodes, a conglomerate group of contiguous nodes, or a large confluent homogeneous mass in which individual nodes are no longer identifiable. It is impossible to differentiate benign and malignant causes for node enlargement by CT alone. Likewise, CT cannot depict focal architectural change and a negative CT does not exclude lymph node pathology.

Structures which must be differentiated from adenopathy include: bowel loops, left-sided inferior vena cava, dilated normal veins, the diaphragmatic crus, retroperitoneal hemorrhage (especially due to aneurysm) and retroperitoneal fibrosis.

Magnetic resonance imaging may provide useful complementary information, especially if the CT is equivocal. Lymphadenopathy usually

has low or intermediate signal on T1- weighted or proton-density images. On T2-weighted images, lymph nodes have a high signal and may resemble fat. MRI may be useful in differentiating abnormal lymph nodes (high signal) from vessels (low signal) due to flow void phenomenon. Likewise MR may be helpful in differentiating tumorous nodes from fibrosis following chemo- or radiation therapy as fibrosis may have a low signal intensity on T-2 weighted images while tumor will typically have a high signal.

Lymphangiography may also be useful when CT is equivocal or negative. Lymphangiography although seldom performed is more sensitive at detecting architectural abnormalities in normal sized lymph nodes than is CT.

RETROPERITONEAL FIBROSIS

Retroperitoneal fibrosis refers to the proliferation of fibrous tissue in the retroperitoneum. It may be idiopathic (Ormond disease) or be secondary to aortic disease (i.e. hemorrhage, aortitis), malignancy (especially metastatic disease which is desmoplastic such as sclerosing Hodgkin disease) or miscellaneous conditions including drugs (methylsergide), inflammatory disease, extravasation of blood or urine,

surgery, radiation and collagen vascular disease.

Idiopathic retroperitoneal fibrosis most commonly occurs in patients between the ages of 40 and 60 years. Men are more commonly involved than women (2:1). Pathologically the fibrotic mass is most prominent centrally, typically extending from the infra-renal region to the bifurcation of the great vessels. The fibrotic tissue may involve the inferior vena cava, aorta, ureters and occasionally the iliac and renal veins. Involvement may be symmetric or asymmetric. Microscopically idiopathic retroperitoneal fibrosis is characterized by collagen, fibroblasts, and inflammatory cells. The presence of neoplastic cells excludes the diagnosis of idiopathic retroperitoneal fibrosis and confirms that the fibrotic process is a desmoplastic response to malignancy.

The clinical and laboratory findings of idiopathic retroperitoneal fibrosis are non-specific and include fever, back pain, and elevated erythrocyte sedimentation rate. Less common signs and symptoms include lower extremity swelling (venous occlusion), anuria (obstructive uropathy), and constipation (rectal involvement). Surgical management options include stenting, diversion and/or ureterolysis. Corticosteroid therapy may be useful, especially in cases with active inflammation. Non-idiopathic cases which are drug related may regress when the drug is

withdrawn.

Excretory urography typically demonstrates bilateral hydronephrosis which may be asymmetric. Less commonly involvement will be unilateral. Rarely the ureter is not involved and the kidneys are normal. Retrograde studies typically demonstrate smooth tapering of the ureters most pronounced at the pelvic brim. The ureteral narrowing may be focal or involve a long segment of ureter. Contrary to popular belief, the ureters are usually not displaced medially but maintain a normal location.

CT typically demonstrates a homogeneous soft tissue mass encasing the aorta, IVC and ureters. In most cases the soft tissue mass extends from the infrarenal paraaortic region to the bifurcation of the aorta. Ureteral involvement with subsequent proximal ureteral dilatation and hydronephrosis may be asymmetric or unilateral. The aorta is typically not elevated off of the lumbar spine. Contrast enhancement is variable and relates to the degree of vascularity. Enhancement is greater in areas of active inflammation than in dense organized fibrous tissue.

MRI may be helpful in distinguishing benign or non-neoplastic retroperitoneal fibrosis from malignant retroperitoneal fibrosis.

Typically MR in cases of non-malignant retroperitoneal fibrosis shows a low or intermediate signal intensity on T-1 and T-2 weighted images. This is in contrast to most malignancies which demonstrate a higher signal intensity on T-2 weighted images. MR is also useful in evaluating the effect of the fibrotic mass on flowing blood in the aorta and IVC.

Ultrasound may demonstrate the retroperitoneal fibrosis as a poorly marginated, periaortic mass which is typically echo-free or hypoechoic. Any resultant hydronephrosis is easily evaluated as well. Body habitus, bowel gas, adjacent bony structures may interfere with optimal assessment of retroperitoneal fibrosis sonographically. The newer imaging modalities have largely replaced inferior vena cavography. Venacavography is usually reserved for problematic cases in which times it typically demonstrates displacement, extrinsic compression or complete obstruction.

Unusual radiologic features of retroperitoneal fibrosis which may be encountered include: ventral displacement of the aorta disease confined to the pelvis, involvement of the perirenal space, displacement of the retroperitoneal bowel, and vascular occlusion. Rarely the CT examination is normal.

The major diagnostic differential is between " benign"

retroperitoneal fibrosis (eg idiopathic, drug related, inflammation) and a desmoplastic malignancy. Although the radiological appearance and clinical features usually allows differentiation, tissue diagnosis is often required.

RETROPERITONEAL FLUID COLLECTIONS

Perirenal Space

Abnormalities that arise either within the kidney or within structures of the adjacent retroperitoneal spaces may cause fluid to accumulate in the perirenal space. The most common fluid accumulations are blood, pus and urine. Fluid that collects slowly and in small to moderate amounts usually localizes in the posterior and inferior aspects of the perirenal space, behind and below the lower pole of the kidney. Acute distention of the perirenal space by a large amount of fluid transforms the normal inferomedial orientation of the space into a vertical axis; obliterates the outline of the kidney and upper one-half of the psoas muscle; displaces the kidney in an anterior, medial and superior direction; and, when extensive, causes anterior displacement of the descending duodenum (if on the right) or lateral displacement of the retroperitoneal part of the ascending or descending colon. Rapid accumulation of a large perirenal collection of fluid causes a concave

deformity of the renal contour similar to that seen with a subcapsular fluid collection.

Perinephric hemorrhage is usually traumatic but may also result from intrinsic rehal disease (e.g. tumor, vasculitis, infection), adrenal disease (especially neoplasia) or from bleeding diathesis. In cases resulting from trauma, it is necessary to obtain followup-up studies to exclude occult neoplasia. In spontaneous, not traumatic cases, renal or adrenal neoplasia must always be strongly considered.

Pus from suppurative processes, either in the kidney or in the pararenal spaces, may extend into the perirenal space. In pancreatitis, this is facilitated by the release of digestive enzymes, which, at a minimum, increase the density of perirenal fat owing to edema.

Urine may leak into the perirenal space as a result of an acute or subacute outflow obstruction, as occurs with ureteral obstruction or penetrating or blunt trauma. In acute obstruction due to stone, urine enters the perirenal space through rupture of a calyceal fornix that is distended by increased hydrostatic pressure in the pelvocalyceal system. When patients are studied with excretory urography, the contrast material-induced osmotic diuresis adds to the already elevated pressure of urine in the renal pelvis. Thus, the likelihood of forniceal rupture and urine

leakage increases with the amount of contrast material given. Acute leakage of urine into the perirenal space due to trauma occurs at any point where the pelvocalyceal system or proximal ureter is disrupted.

Urine that slowly leaks into the perirenal space over a long period of time creates a unique condition known as a urinoma. This urine collection has also been termed a uriniferous perirenal pseudocyst, pseudo hydronephrosis, hydrocele renalis, perirenal cyst. Here, an encapsulated extrapelvocalyceal collection of urine forms from urine leakage through a rent in the collecting system or the proximal ureter when chronic ureteral obstruction is present. Accidental or surgical trauma, congenital obstruction in children, ureteral tumor, stone, blood clot, and periureteric fibrosis are among the usual causes of the obstruction. As urine leaks into the perirenal space, normal fat is transformed into a dense, reddish-colored fibrous mass that is covered by distended veins. The wall of the pseudocyst contains fatty or fibrous debris, blood clot, and crystals of urine salts. The mature pseudocyst is the end result of this process of lipolysis and fibroblastic round cell stimulation. A false capsule forms within 2 weeks and matures in approximately 6 weeks.

Clinical manifestations of urinoma are a palpable flank mass and vague abdominal distress or tenderness. There is little, if any, fever. The

mass appears slowly after the initiating event, often with a latent period of 4 or more months.

Urinoma has a radiologic appearance of a soft tissue mass whose axis usually, but not always, approximates the cone of renal fascia. Large lesions displace the kidney superiorly and deviate the lower pole laterally. The proximal ureter is sometimes displaced medially, occasionally to an extreme degree. Perirenal fat remote from the pseudocyst remains uninvolved so that both the upper psoas margin and the contour of the superior half of the kidney are preserved. Additionally, radiologic and ultrasonic findings of chronic obstruction are present. Active extravasation is sometimes seen following injection of contrast material. The fluid nature of the urinoma can be accurately defined by ultrasound, computed tomography. Debris or blood may produce low-level echoes or raise the computed tomographic attenuation values.

Subcapsular Space

Traditionally, it was believed that distinction between a subcapsular and perirenal fluid collection was easily made on CT. Fluid was felt to be subcapsular when it was immediately contiguous with a portion of the renal parenchyma and flattened its normal convex border. Typically fat was present between the fluid collection and Gerota's fascia

which was preserved. In contrast a perinephric fluid collection was diagnosed when it (1.) surrounded a major portion of the kidney, (2.) was contiguous with the renal fascia which was obliterated, (3.) replaced the normal perinephric fat, and (4.) typically did not flatten or distort the normal renal contour.

Careful anatomic studies of the perinephric space, however, have demonstrated that the perinephric space is not one compartment, but is subdivided by bridging septa and by the renorenal septum. Perirenal fluid collections localized by these septa can be indistinguishable from fluid collections which are subcapsular. Differentiation in most cases is not important as management is similar.

The most common subcapsular/localized perinephric fluid collection is blood from trauma, tumor, vasculitis, bleeding diathesis or intrinsic renal disease. Although there are many non-neoplastic etiologies, renal cell carcinoma must always be excluded as the etiology for a subcapsular/localized perinephric bleed, especially those which are spontaneous (i.e. non-traumatic). Pus is less likely to accumulate in the subcapsular space or localized perinephric space as the proteolytic enzymes often enable direct extension through the capsule, septa and often through fascia into the paranephric spaces. Occasionally a

subcapsular/localized perinephric hemorrhage may become secondarily infected resulting in a thick walled localized inflammatory mass. Most frequently, these subcapsular/localized perinephric fluid collections are identified following trauma. Management is usually conservative and in most cases they resolve spontaneously. Less commonly, however, these hematomas do not resolve and become chronic. Renal (ischemic) hypertension may follow a phenomenon known as "Page" kidney. Biconvex, lentiform calcification is very characteristic for chronic, subcapsular or loculated perinephric hemorrhage.

Anterior Pararenal Space

Pancreatitis is the most common cause of a fluid collection in the anterior pararenal space. Large fluid collections in the anterior pararenal space extend toward the peritoneal cavity, displacing the small intestine ventrally and the ascending or descending colon laterally. The properitoneal "flank stripe" renal outline, perirenal fat, and psoas margins are maintained. Proteolytic enzymes disrupt the small septal fibers which connect the lateral conal fascia to the anterior renal fascia. Fluid dissects between the two major layers of the posterior renal fascia producing a characteristic wedge shaped appearance of the retrorenal fluid. Typically the fat within the posterior pararenal space is

compressed but preserved.

Posterior Pararenal Space

Abnormalities of the posterior pararenal space develop through the spread of disease from the anterior pararenal space, the retrocrural space, extraperitoneal pelvic structures, the perirenal space, or adjacent organs. Spontaneous hemorrhage, extension of osteomyelitis of the spine, and lymphatic extension of tumor from the pelvis or lower extremities are several examples. Primary tumors of posterior pararenal fat and connective tissue occur as well.

Abnormalities that extend into the posterior pararenal space obliterate the lower one-half of the psoas muscle and the properitoneal flank stripe. The kidney may be displaced in an anterior and superior direction (Fig. 24-29). Fluid collections have an inferomedial axis similar to that of the inferior cone of the perirenal fascia.

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Screening for Ovarian Carcinoma

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There are no accepted screening techniques for ovarian carcinoma. Despite widespread publicity, current "screening" techniques are essentially experimental protocols.

In the past 60 years, there has been no significant diagnostic or therapeutic advances in ovarian carcinoma. Ovarian cancer is the leading cause of death from gynecologic malignancy in the United States. Seventy-five percent of ovarian carcinoma patients present with advanced disease. The overall 5 year survival rate is 38%. The difficulty with making an early diagnosis is the lack of symptoms associated with the stage I and stage II disease and the difficulty in detecting an increase in ovarian size by the bimanual pelvic examination. These facts clearly point to a need for an effective screening tool to detect the early stages of ovarian carcinoma. Two avenues of approach are available for screening: serum tumor markers (CA 125) and ultrasound (transabdominal and transvaginal).

Since ovarian cancer is not a common disease, universal screening is not cost-effective. Current screening strategies advocate screening only those patients considered high risk such as a family history of ovarian cancer. Evaluation of such patients include a detailed physical examination, testing for tumor markers and endovaginal ultrasound. Endovaginal ultrasound provides superior visualization of the ovaries. The ovary is examined for abnormality in size or evidence of cystic or solid ovarian masses. Color and duplex Doppler are then used to search for abnormal flow or neovascularity. Abnormal flow is characterized by low resistance signals in the absence of a corpus luteum.

Drs. Ken Taylor and John Pellerito of Yale University performed a retrospective review of ultrasound findings in patients with known ovarian cancer. They reviewed both sonographic morphology and Doppler waveform. Currently, they are applying these criteria prospectively in order to diagnose ovarian cancer.

Criteria are as follows:

- Age - postmenopausal
- Ascites
- Cyst size - > 5 cm
- Sonographic morphology
  - cyst with thick septations
  - cyst with papillary projections
  - solid mass
- Peak systolic velocity - > 25 cm/sec
- PI $\leq$ 1.0
- RI $\leq$ 0.4

If 3 out of 5 criteria are fulfilled, there is a high probability for ovarian carcinoma.

Sonographic morphology is more sensitive than Doppler wavefrom.

ABDOMINAL ULTRASOUND: BILIARY CONSIDERATIONS

Charles J. Fagan, M.D.

The traditional role of biliary sonography is threefold: (1) evaluate the signs and symptoms of cholecystitis; (2) search for the cause of jaundice and abnormal liver function tests and (3) attempt to show a biliary etiology for non-alcoholic pancreatitis. Most cases are straight forward but biliary sonography cannot be taken for granted - it is not a static subject.

To emphasize this point, the author presents a preview of biliary publications to be featured in the 1995 Year Book of Ultrasound slated for showing at the RSNA meeting in December, 1995.

OLD SUBJECTS REVISITED

- Gallbladder sludge must be considered an important pathologic entity because of the significant incidence of cholelithiasis and/or acute cholecystitis following sludge detection.[1]
- Ultrasound is a highly accurate method for delineating the level of biliary obstruction (88% sensitivity) but fails to define the etiology of biliary obstruction, especially choledochalithiasis and cholangiocarcinoma with an acceptable degree of confidence.[2]
- CBD caliber does not normally change after cholecystectomy.[3]
- Extrahepatic bile duct diameter varies during respiration - potential pitfall in sonographic misdiagnosis of common bile duct obstruction.[4]
- Hepatic arterial calcifications can result in an erroneous sonographic diagnosis of intrahepatic biliary stones.[5]

NEW CONCEPTS OF PHYSIOLOGY, DISEASE PROCESSES, AND CONTRIBUTIONS OF NEW TECHNOLOGY

- Contrast medium solution given before an abdominal CT generally does not cause sufficient gallbladder contracture to preclude post-CT cholecystosonogram if the need arises.[1]

- Duplex sonograms show gallbladder varices are common in patients with portal thrombosis.[2]

- Color Coppler interrogation of the inflamed gallbladder has, at this time, limited value in the diagnosis of cholecystitis.[3]

- The differential diagnosis of focal defects of the gallbladder wall should include osteomas[4] and ectopic liver tissue.[5]

- CT is superior to ultrasound for the diagnosis of gallbladder perforation.[6]

- Bile duct hamartomas[7] and granular cell tumors of the biliary tract[8] are detected by ultrasound.

ROLE OF ULTRASOUND IN NEW BILIARY THERAPEUTIC PROCEDURES

- Percutaneous cholecystolithotomy remains justified in the management of selected patients with gallstones.[1]

- Ultrasound findings can be utilized as predictors of potential operative difficulties for laparoscopic cholecystectomy.[2]

- Laparoscopic sonography has the potential to replace intraoperative cholangiography.

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Doppler Ultrasound in the Evaluation of the Hepato-Portal System and Mesenteric Vessels

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General Considerations:

Abdominal Doppler has become a practical tool because image directed duplex scanning and color Doppler has allowed abdominal blood flow information to be obtained noninvasively. Doppler can simply determine the presence or absence of blood flow or the direction of blood flow.

The shape of the Doppler waveform and the distribution of RBC velocities within the waveform can be evaluated. The shape of the waveform in the arterial system is a function of the downstream circulation and central effects. For instance, a low resistance waveform such as the hepatic artery has much diastolic flow in the forward direction. A higher resistance waveform such as the SMA in a fasting patient has little or reversed flow during diastole. Venous waveforms such as the IVC and hepatic veins are affected by cardiac events and respiration. However the portal vein which delivers blood flow to the liver, does not normally show the effects of cardiac contraction because the intervening liver filters out those effects.

Doppler can investigate the presence of abnormal velocity or turbulence which may indicate stenoses. Significant stenoses are characterized by increased velocity through a narrowed lumen and turbulence beyond the stenosis. Further downstream the flow may return to normal but may show a blunted waveform. This waveform has diminished pulsatility and a slower upstroke of its systolic component.

Portal Vein:

Blood in the portal vein is towards the liver (hepatopetal). It is typically wavy or flat but in a small percentage of normal patients it may be pulsatile (pulsatility has been described in cardiac disease such as elevated RA pressure or tricuspid regurgitation). In portal hypertension the blood may become hepatofugal with blood flowing away from the liver. Hepatofugal flow in the main portal vein is rare even with profound portal hypertension.

In portal hypertension the increased resistance to flow reduces the amount of portal venous flow. Compensatory increased flow through the hepatic artery is noted. As the pressure increases in the portal system, collateral vessels from the splanchnic vessels will open to bypass the liver and deliver the blood directly to the systemic circulation. The common sites of collaterals are the coronary vein and gastro-esophageal varices, spleno-renal veins, patent paraumbilical veins, retroperitoneal veins, and pelvis veins to iliac veins.

Portal vein thrombosis is both a cause and a sequela of portal hypertension. It is typically seen in cirrhotics, those with hypercoagulable states and in patients with hepatomas. The echogenicity of the thrombus is variable and the vein may be expanded in acute thrombus or in tumoral thrombus. Once identified in the cirrhotic, one should always look for a hepatoma to explain why portal vein thrombosis occurred. Duplex Doppler and color Doppler reveals no flow but sluggish flow in cirrhosis may simulate occlusion. One should always confirm the color impression of no flow with Duplex scan to rule out a technical cause for finding. If portal vein thrombosis is longstanding periportal collaterals

may develop over time and reconstitute flow to the liver through the liver hilum.. This collection of small vessels is called cavernous transformation of the portal vein.

Hepatic Veins:

All three hepatic veins should be routinely visualized and blood in the hepatic veins is towards the IVC. The normal hepatic vein signal is pulsatile and shows changes from cardiac events.

Hepatic vein obstruction (Budd-Chiari syndrome) is a complex set of diseases. Different sites of involvement are possible. The IVC may be primarily involved obstructing the hepatic veins secondarily. Alternatively, one or more of the hepatic veins may be primarily obstructed or thrombosed. Causes may consist of congenital or acquired webs, tumor thrombus or bland thrombus. In Budd-Chiari, absent signals or loss of pulsatility in a hepatic vein proximal to the obstruction may be detected by Doppler. Unusual collaterals may develop in the liver parenchyma or capsule. Color or Duplex Doppler may show vessel flow not directly to the IVC but through collaterals. The identification of a bicolored hepatic vein bifurcation is pathognomonic for Budd-Chiari and indicates proximal blockage. Hepatofugal portal flow may be present. IVC webs or stenosis can sometimes be imaged.

Porto-Caval Shunts:

Duplex Doppler and color Doppler are helpful in the evaluation of patency of surgical porto-systemic shunts. Ultrasound may identify the shunt and the Doppler flow within the shunt. In patent portal-caval shunts, pulsed Doppler will show that the portal vein has a pulsatile flow pattern like the IVC (systemic rather than portal vein type pattern). Hepatofugal flow in the portal vein is presumptive evidence that the shunt is open.

TIPS - Transjugular Intrahepatic Portosystemic Shunts:

TIPS consists of a metal wire mesh tube connecting the portal venous system (usually the right branch of the portal vein) to a hepatic vein (usually the right hepatic vein). In portal hypertension, the portal venous system is under high pressure. TIPS provides decompression of the portal venous system through the shunt into the low pressure systemic hepatic vein.

Shunt occlusion may occur without any symptoms. Shunt surveillance should be performed to identify a failing shunt. Revision of a failing shunt may prevent shunt occlusion. We perform a baseline study prior to discharge followed by shunt surveillance every 3 months for the first year and then every 6 months thereafter.

Shunt surveillance should include:

- angle corrected velocity in the main portal vein
- direction of flow right and left portal vein branches
- angle corrected velocity in shunt (proximal, mid and distal portions of shunt)
- visualization of all 3 hepatic veins, direction of flow and phasic waveform

Ultrasound of failing TIPS:

- peak systolic velocity in shunt < 85 cm/sec. (normal velocity 100-180cm/sec)
- focal stenotic jet in shunt or hepatic veins
- visualized narrowing with color flow or gray scale

- change in direction of flow of the right or left portal vein branch or hepatic veins from the baseline study. Normally flow is directed toward the shunt. Flow directed away from the shunt in either portal vein branch or hepatic vein suggests failing shunt.
- new or increased ascites
- increased hepatic artery flow
- new visualization of umbilical vein

Mesenteric Vessels and Ischemia:

There are three major vessels supplying the gut: celiac axis, superior mesenteric artery and inferior mesenteric artery. The IMA is not typically seen unless it its hypertrophied.

Mesenteric ischemia typically requires multivessel disease because of the collateral pathways available and identifying stenosing lesions are not equivalent to diagnosing mesenteric ischemia. Doppler is most helpful to triage patients with symptoms to or away from angiography.

Different groups have found different criteria for diagnosing significant celiac or SMA stenoses. In our laboratory we use the following values used by Dr. Laurence Needleman, Thomas Jefferson University.

- Fasting celiac velocity $\geq$ 200 cm/sec indicates greater than 70% stenosis
- Fasting SMA velocity $\geq$ 275 cm/sec indicates greater than 70& stenosis
- The absence of the SMA to increase systolic and diastolic velocity and diminish pulsatility 30 minutes after a standard meal (eight ounces of Ensure).

False positive diagnoses of celiac stenosis may be due to extrinsic compression by the median arcuate ligament. The median arcuate ligament s a fibrous arch that unites the crura on either side of the aortic hiatus. Usually the ligament passes posteriorly to the celiac axis. If the ligament passes anteriorly, it compresses the celiac axis against the aorta. The compression is seen with expiration. Deep inspiration leads to downward displacement of the celiac axis and relieves the compression. Therefore the celiac trunk should be interrogated during both expiration and inspiration to distinguish intrinsic vascular disease from extrinsic compression.

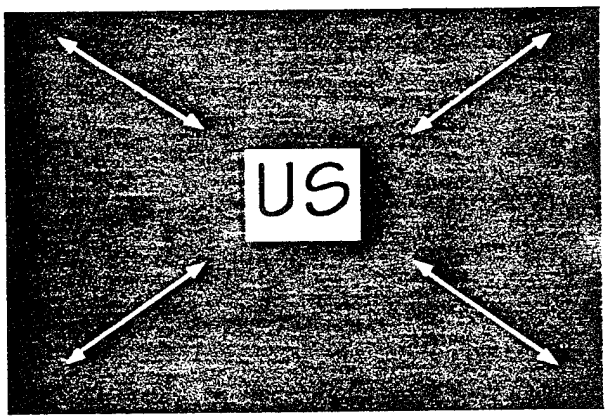
Mesenteric ischemia remains a difficult diagnostic dilemma and the exact role of Doppler is still being sought.

PROSTATE IMAGING

ROBERT B. FINEGOLD, M.D.

PROSTATE IMAGING LECTURE OUTLINE

- ✓ TRANSRECTAL ULTRASOUND
  - —INDICATIONS
  - —TECHNIQUE
  - —US ANATOMY OF THE PROSTATE
  - —US APPEARANCE OF PROSTATE CA
  - —US STAGING OF PROSTATE CA
  - —TRUS: APPLICATIONS
- ✓ PROSTATE MRI
  - —INDICATIONS
  - —TECHNIQUE
  - —MRI ANATOMY OF THE PROSTATE
  - —MRI APPEARANCE OF PROSTATE CA
  - —MRI STAGING OF PROSTATE CA
- ✓ SUMMARY



TRUS INDICATIONS

1. ABNORMAL DRE
2. ABNORMAL PSA
3. BIOPSY GUIDANCE
4. CA STAGING
5. CRYOABLATION GUIDANCE
6. INFERTILITY ASSESSMENT
7. ABCESS DRAINAGE

TRUS TECHNIQUE

- OUTPATIENT
- NO PREP NECESSARY (EXCEPT FOR BX)
- PATIENT: LEFT DECUBITUS POSITION
- CORRELATIVE DRE
- HIGH FREQUENCY (~7 MHZ) TRANSDUCER
- EXAMINE IN AT LEAST TWO IMAGING PLANES

TRUS ANATOMY (MCNEAL)

NON-GLANDULAR ZONE
ANTERIOR FIBROMUSCULAR STROMA

GLANDULAR ZONES	[%]	[%CA]	[%BPH]
PERIURETHRAL	1	–	5
TRANSITIONAL	5	20	95
CENTRAL	24	10	–
PERIPHERAL	70	70	–

US APPEARANCE OF PROSTATE CA

FINDINGS SUSPICIOUS FOR TARGETED BIOPSY:

- HYPOECHOIC PERIPHERAL LESION
- ECHOGENICALLY ASSYMETRIC PZ
- FOCAL BULGE
- CAPSULAR / PERICAPSULAR IRREGULARITY

HISTOLOGIC PATTERNS & US IMAGING

- NODULAR (40%)
  2D = HOMOGENEOUS & HYPOECHOIC (PAUCITY OF INTERFACES)
  CDI = HYPERVASCULAR
- NODULAR-INFILTRATING (30%)
- INFILTRATING (30%)
  2D = ISOECHOIC (TUMOR MIXED WITH NORMAL REFLECTIVE TISSUE)
  CDI = ISOVASCULAR

COLOR DOPPLER IMAGING OF PROSTATE CA

- HELPS SELECT SITES FOR BIOPSY WHEN MULTIPLE HYPOECHOIC LESIONS ARE PRESENT.
- HELPS IDENTIFY SMALL VOLUME CANCERS OF HIGH GLEASON GRADE IN PTS WITH NORMAL PSA'S.
- PROBLEM: INFLAMATION (PROSTATITIS) MAY ALSO BE HYPERVASCULAR

STAGING OF PROSTATE CA (MODIFIED JEWITT-WHITMORE)

A (NONPALPABLE)
A1 = < 1 cc
A2 = > 1cc

B (PALPABLE)
B1 = < 50% gland
B2 = > 50% gland

C (EXTRACAPSULAR EXTENSION)

D (METASTATES): LYMPH NODES, BONE

TRUS STAGING

- 2D IMAGING CAN DETECT GROSS EXTRACAPSULAR EXTENSION BUT OVERALL STAGING ACCURRACY ~ 58% (RIFKIN).
- SEXTANT RANDOM BIOPSIES (HODGE)
- TAILORED BIOPSIES (LEE)
  ASSESS "WEAK ANATOMIC AREAS":
  - PERINEURAL SPACES OF THE NVB
  - APEX AT JXN OF CAPSULE & AFS
  - BASE NEAR BLADDER NECK & CZ (IES)

TRUS-GUIDED BIOPSY TECHNIQUE

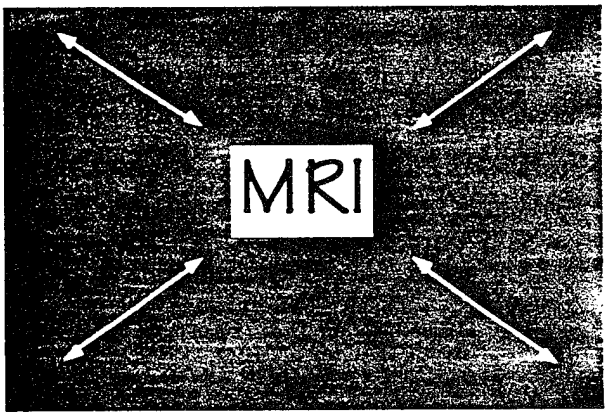
- ANTIMICROBIAL PROPHYLAXIS (e.g. Cipro 500 mg)
- D/C ANTICOAGULANTS
- PATIENT AFEBRILE
- INFORMED CONSENT (RISKS: hemorrhage, infection, inability to urinate)
- REAL TIME US-GUIDED 18 GAUGE BIOPSY CORES (by biopsy gun)
- POST-PROCEDURAL INSTRUCTIONS

TRUS APPLICATIONS

PROSTATE:
- Volume assessment
- CA Detection (DRE+ &/or PSA+)
- US-Guided Biopsies (Directed, Random, Staging)
- Evaluation for Post-operative tumor recurrence
- US-Guided Cryoablation of CA
- Infertility

RECTAL:
- Cancer Staging

PELVIC:
- Node Biopsy
- Abcess Drainage



PROSTATE MRI INDICATIONS

- PRE-OPERATIVE STAGING OF PTS WITH BIOPSY PROVEN CANCER:
  - TRANSCAPSULAR SPREAD (NVB)
  - SEMINAL VESICLE INVASION
  - LYMPH NODE METASTASES
- EVALUATION OF COMPLEX CONGENITAL ANOMALIES

PROSTATE MRI INDICATIONS

- INFERTILITY (SEMINAL VESICLE ANOMALIES)
- HEMATOSPERMIA

MRI TECHNIQUE: BODY COIL

- T1W AXIAL (600/20)
  - HIGH ORGAN / FAT CONTRAST
  - TISSUE CHARACTERIZATION (BLOOD, FLUID, FAT)
  - NODE DETECTION, BONY METS
- T2W AXIAL (2500/40,80)
  - INTRINSIC PATHOLOGY
- SUPPLEMENTAL T2W CORONAL / SAGITTAL

MRI TECHNIQUE: BODY COIL

- FOV ~ 36 cm
- NEX = 2
- MATRIX = 128 X 256
- THICKNESS =
  10 mm (T1) / 2.5 mm gap
  5 mm (T2) / 1.0 mm gap
- GLUCAGON 1 mg IV

MRI TECHNIQUE: ENDORECTAL

- ENDORECTAL COIL (~ $100)
  1.5 T SIGNA GE
- INITIAL SAGITTAL LOCALIZER
- FOV ~ 10 cm
- THICKNESS = 3 mm / 0.5 mm gap

ADVANTAGES OF ENDORECTAL MRI

BETTER RESOLUTION OF:

- NEUROVASCULAR BUNDLE
- SEMINAL VESICLE ARCHITECTURE
- PROSTATE CAPSULE
- PUBOCOCCYGEOUS MUSCLE

MRI TECHNIQUE: FAST SE

FAST SE, SINGLE ECHO, EIGHT-ECHO TRAIN

4000 / 105 EFFECTIVE

CONTIGUOUS INTERLEAVED 3mm-THICK SECTIONS

RIGHT-TO-LEFT PHASE ENCODING

2 SIGNAL AVERAGES

FOV: 16-22cm (PPA)
14cm (INTEGRATED ER-PPA)

GLUCAGON 0.5-1.0mg IV

FAST SE / PHASED ARRAY

FSE:
Reduces acquisition time for T2W imaging
Reduces motion artifacts
Alternate plane imaging readily available

PPA Coil: Higher Signal-to-Noise ratio

FSE + PPA = Higher resolution; More signal averages possible (despite shorter imaging times)

3 ORTHOGONAL PLANES

- AXIAL: TZ vs. PZ optimized
- SAGITTAL: CZ vs. PZ,
  BLADDER BASE,
  RECTUM
- CORONAL: CZ vs. PZ,
  PROSTATE APEX,
  SEMINAL VESICLES,
  LEVATOR ANI MUSCLES

MRI ANATOMY: PROSTATE

T1W: HOMOGENEOUS
INTERMEDIATE SI

T2W: PZ = HIGH SI
CZ / TZ = LOWER SI
AFM = LOW SI
CAPSULE = LOW SI (1 mm)

MRI ANATOMY: SEMINAL VESICLES *

T1W: INTERMEDIATE SI

T2W: HIGH SI FLUID
LOW SI WALLS

NORMAL VARIABILITY OF SI (~ FAT)
BUT SI SHOULD BE SYMMETRIC

CONGENITAL ANOMALIES

AGENESIS

HYPOPLASIA

CONGENITAL CYSTS
—MIDLINE: UTRICLE CYST
MÜLLERIAN DUCT CYST
—LATERAL: PARENCHYMAL CYST
SV CYST

BPH

TZ & PERIURETHRAL TISSUE

—DIFFUSE FORM
—FOCAL ADENOMA

HYPERPLASTIC TISSUE SEPERATED FROM PERIPHERAL ZONE BY THE SURGICAL PSEUDOCAPSULE

PDM: PERIURETHRAL INVOLVEMENT
⇒ MEDIAN LOBE ENLARGEMENT

BPH: MRI

T1W = HOMOGENOUS LOW SI
T2W = VARIABLE:
HOMO- VS. HETEROGENOUS,
MEDIUM TO ↑ SI

PSEUDOCAPSULE = LOW SI STRIPE

SAGITTAL: ✓FOR MEDIAN LOBE ↑
✓FOR F/U OF TURP

CARCINOMA: MRI

- FOR STAGING OF BIOPSY-PROVEN PROSTATE CANCER
- NO ROLE IN CANCER DETECTION
- T2W: ↓ SI IN HIGH SI PZ
(↗ mucous-producing cancers ~1%)
- TUMORS OUTSIDE PZ ARE NOT OFTEN DETECTED

CARCINOMA: MRI

- A NORMAL APPEARING GLAND DOES NOT EXCLUDE THE PRESENCE OF CARCINOMA
- POST-BX HEMORRHAGE IMPAIRS TUMOR DETECTION
(≤ 1 WEEK: ↓ T1; ↑ T2)
(> 1 WEEK: ↑ T1; ↑ T2)
—IMAGE ~2-3 wks POST-BIOPSY

STAGING WITH MRI

TNM	JEWITT-WHITMORE
T1a: <5% gland	A1
T1b: >5% gland	A2
T2a: <50% lobe	B1
T2b: ≥50% lobe	B1
T2c: both lobes	B2
T3a: unil. extra-capsular spread	C
T3b: bilat. spread	C
T3c: SV invasion	C

CANCER STAGING: MRI

- EVALUATE LOCAL TUMOR
  - —TRANSCAPSULAR EXTENSION
    - √ CONTOUR BULGE
    - √ ↓ SI PERIPROSTATIC FAT
  - —SEMINAL VESICLE INVASION
    - √ ↓ SI
- DETECT NODAL & DISTANT METASTASES

CRITERIA FOR EXTRACAPSULAR EXTENSION

- INVASION OF PERIPROSTATIC FAT (T1W)
- PROSTATE CAPSULE DISRUPTION (T2W)
- INVASION OF NEUROVASCULAR BUNDLE (T1-T2)
- LOW SI IN SEMINAL VESICLE (T2W)
- LYMPH NODE ENLARGEMENT (T1W)
- BONE METASTATSIS (T1W)

SEMINAL VESICLE INVASION

- PERIPHERAL (VIA NVB)
  CENTRAL (VIA EJACULATORY DUCTS)
- LOW SI ON T2W
- THICKENING OF TUBULES = EARLIEST SIGN
  (SEEN WITH ENDORECTAL MRI)

NOTE: DO NOT BE FOOLED BY NORMAL VAS DEFERENS AMPULLA

STAGE — THERAPY

T A-B — SURGICAL (NERVE-SPARING)
JOHN HOPKINS POST-OP POTENCY
~ 68 % (>1000 CASES)

T C-D — RADIATION THERAPY
HORMONAL THERAPY

MRI (BODY COIL)

STAGING ACCURACY	SENSIT	SPECIF	# PTS
.78 [1]	.84	.72	81
.69 [2] (.58)	.77 (.66)	.57 (.46)	234
.55 [3]	.48	.66	100

US

1. BEZZI, RADIOLOGY 169:339-346, NOV 1988
2. RIFKIN (RDOG), NEJM 323(10): 612-618 SEP 1990
3. SCHLEIBER, AJR 154: 559-562, MAR 1990

CAUSES OF WORSENING STATISTICS

- INITIAL STUDIES INCLUDED PATIENTS WITH ADVANCED DISEASE (STAGE C & D) WHILE CURRENT STUDIES FOCUS ON MORE SUBTLE LESIONS.
- INITIAL STUDIES INTERPRETED BY SUB-SPECIALISTS WHILE CURRENT STUDIES ARE NOW INTERPRETED BY GENERALISTS.
- INCLUSION OF MICROSCOPIC CAPSULAR INVASION IN STATISTICS.

MRI (ENDORECTAL) [1]

STAGING (B-C) ACCURACY	SENSIT	SPECIF	# PTS
.82 [.69] BODY	.93 [.77] T8	.84 [.57]	22
.63 TC			

1. SCHNALL, RADIOL 178:797-802 MAR 1991

SENSITIVITY FOR CAPSULAR INVASION

MRI (BODY COIL) .44[1] - .62[2]

MRI (ENDORECTAL) .67[3]*

1. BEZZI, RADIOL 169: 339-346 NOV 88
2. RIFKIN (RDOG) NEJ M 323 (10): 612-616 SEP 1990
3. SCHNALL RADIOL 178: 797-802 MAR 91

* 1.00 FOR GROSS INVASION

MRI (ENDORECTAL)

- ↑ SIGNAL / NOISE
- ↓ FIELD OF VIEW (10-12 cm)
- ↓ SLICE THICKNESS
- ↓ PIXEL SIZE
- ↓ CHEMICAL SHIFT ARTIFACT

PHASED ARRAY COILS

- SEPARATE EXTERNAL SURFACE COILS EACH WITH ITS OWN DIGITAL RECEIVER.
- DATA IS COMPOSED INTO A SINGLE COMPOSITE IMAGE.
- NO ↑ ACQUISITION TIME OVER SINGLE-COIL STUDIES.

PHASED ARRAY COILS

- ↑ FIELD OF VIEW (18 cm)
- MATRIX = 512 X 512
- HIGH S / N (DUE TO SEPARATE IMAGE COLLECTION)
- FAST SPIN ECHO (T2W: 4500 / 126)
  - ↑ ACQUISITION TIME (~ 11 SLICES IN 2.5 min)
  - ↑ SPATIAL RESOLUTION

MRI-PATHOLOGY CORRELATION: EXTENT OF PROSTATE CA (FSE-PPA)

	MRI ACCURACY (%)
EXTRACAPSULAR SPREAD	85
SV INVASION	100
NODAL METASTASES	85

Kier, AJR 1993; 161:60

COMBINED PHASED ARRAY SURFACE COILS AND ENDORECTAL COIL

- ENTIRE PELVIS IMAGED (WITH HIGH SENSITIVITY MAINTAINED IN POSTERIOR ASPECT OF PROSTATE GLAND).
- UNIFORM SI FIELD WITH VERY HIGH RESOLUTION (i.e. PREVENTS FAR FIELD DROP-OFF)

COMBINED PHASED ARRAY SURFACE COILS AND ENDORECTAL COIL

- ↑ NODAL DETECTION (OBTURATOR, PERIPROSTATIC, . . .
- IMPROVED STAGING ACCURACY (? SENSITIVITY FOR MICROINVASION OF PROSTATE CAPSULE)

MULTI-ARRAY SURFACE COIL ACCURRACY [1]

	PATHOLOGICAL STAGE ST B	ST C
MRI ST B	22	2
MRI ST C	2	14

ACCURACY: 36 / 40 = 90%*

*I.E. MICROSCOPIC INVASION INCLUDED

[1] POLLACK, HUP

MRI-PATHOLOGY CORRELATION: EXTRACAPSULAR EXTENSION

	PPA	Integrated ER-PPA
Sensitivity (%)	75	84
Specificity (%)	67	80
PPV (%)	48	65
NPV (%)	86	92

Hricak, RAD 1994; 193:703-8

SUMMARY: MRI PROBLEMS

- HIGHLY TECHNIQUE DEPENDENT
- INTERPRETATION LEARNING CURVE IS LONG AND NARROW
- CENTRAL GLAND (TZ-CZ) BLIND SPOT; APEX DIFFICULT TO IMAGE
- CANNOT EXCLUDE MICROCAPSULAR INVASION

PROSTATE IMAGING

THE CLINICAL IMPACT OF PROSTATE IMAGING REMAINS TO BE DETERMINED.

WE HAVE THE ABILITY TO DETECT PROSTATE CA EARLY AND ARE MAKING IMPROVEMENTS IN STAGING ACCURACY.

IMPROVEMENTS IN PROSTATE CA TREATMENTS AND PROPERLY CONDUCTED OUTCOME STUDIES ARE NECESSARY TO ULTIMATELY DEFINE THE ROLE OF PROSTATE IMAGING.

SUMMARY: PROSTATE CA

1. SCREENING DRE AND PSA
2. TRUS (WITH BIOPSY)
3. MRI (PRE-OP STAGING)

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Abstract:

Army Radiology Resident Research Competition 1995

TITLE: Colonic Perforation During Air Enema in the Porcine Model: Relative Risk of Perforation in the neonate

PERSONNEL:

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Introduction: Current recommendations regarding radiologic therapy of intussusception in the 0-3 month old infant suggest the use of lower maximum intraluminal pressure than that used with older infants. Such an approach may result in a disproportionate number of patients undergoing surgical reduction, a procedure with greater cost and morbidity than non-surgical therapy. There is no experimental data available to support such an approach.

The Law of Laplace states that the wall tension of a closed system is directly related to the pressure and the radius of that system by the equation T=Pr. If it can be assumed that children have narrower intestinal lumina than the adults, the wall tension at a given pressure would be less in the narrower viscus.

Based on the above premise, we hypothesized that the younger subject would be at decreased risk for perforation, other factors being equal. Relative risk of perforation was determined, utilizing the porcine model, in varied aged animals.

Materials and Methods: Under general anesthesia and after construction of a closed colonic loop via surgical ligation of the terminal ileum, air enema was performed with fluoroscopic and continuous intraluminal pressure monitoring in three groups of animals (ages 10-20 days, 4-8 weeks and 9-16 weeks). Perforation pressures were documented.

Results: The younger animals demonstrated higher perforating pressures than the older groups ($P<0.05$); 10-20 days - P=136.9 (sd=2.6), 4-8 weeks - P=98.8 (sd=3.9), 9-16 weeks - 82.2 (sd=2.8).

Conclusion: The younger animal is at decreased risk for colonic perforation during air enema when compared to the older subject. Application of this data to the practice of pneumatic reduction of intussusception suggests that use of lower pressures in the 0-3 month age group may be both unnecessary and inappropriate. If other clinical parameters known to alter the risk of perforation are accounted for, the use of lower pressures may decrease the rate of successful non-operative reduction and subject a disproportionate number of these patients to the higher morbidity and mortality of surgical treatment.

Cost effectiveness of screening knee MRI: A prospective evaluation of 50 consecutive patients.

Purpose: To determine the cost effectiveness of screening knee MRIs prior to arthroscopies of the knee.

Materials and Methods: This study involves 50 patients. It is single-blinded and prospective. The patients have met the surgical indications monitoring for appropriateness (SIM-A criteria) for arthroscopy of the knee and have had a knee MRI within 2 weeks prior to the procedure.

MRI and arthroscopy findings have been compared. Based on a cost of $1,000 per MRI and $4,000 per diagnostic arthroscopy, cost effective MRI requires 25% true negativity.

Results: The sensitivity (SN) and specificity (SP) of the knee MRI for the detection of anterior cruciate ligament interruption are 100% and 90%, respectively; posterior cruciate ligament tear, 100% and 82%; medial meniscal tear, 90% and 93%; lateral meniscal tear, 60% and 100%; and composite injury (one or more of the above abnormalities), 97% and 93%. There were 12 (24%) avoidable diagnostic arthroscopies, as prospectively predicted by unremarkable MRI examinations.

Conclusion: Despite very stringent clinical criteria in selecting arthroscopy patients, 24% of the patients could have been spared the procedure, based on MRI screening. Our prospective study indicates that screening MRI is cost effective.

CPI Yong T. Bradley 3rd year resident

Glenohumeral Labral-Ligamentous Complex: Evaluation Using MR Arthrography

Y. Bradley, V Chandnani, T. Murnane, J. Gagliardi

PURPOSE: MR arthrography of the shoulder has enabled more accurate imaging of the structures of the capsular mechanism. In patients with shoulder instability, some have emphasized the role of a deficient inferior glenohumeral ligaments(GHL), while others have proposed a lax capsule, a shallow glenoid, or a deficient labrum as being the causative factors. We sought to determine the incidence and the locations of these and other structural abnormalities in patients with shoulder instability.

MATERIALS AND METHODS: MR arthrographic images of the shoulders of 46 patients with the clinical diagnosis of either instability or pain of unknown etiology were evaluated for the presence and the integrity of the superior, middle, and inferior GHL. The joint capsule type, and the presence and the location of labral tears were sought. All patients underwent further surgical evaluation and surgical findings were correlated with imaging findings.

RESULTS: The superior, middle, and inferior GHL's were identified and found to be torn (identified, torn) in (39,3), (39,18), and (42,8) patients, respectively. There were 9 Type I, 9 Type II, and 28 Type III capsules. Thirty-eight patients had labral tears which were found to be in the superior, anterior, inferior, and posterior portions in (19, 38, 24, 3) patients, respectively.

CONCLUSIONS: The static and dynamic restraints of the glenohumeral joint are reliably visualized in the normal and abnormal state using MR arthrography. Tears of the inferior and middle glenohumeral ligaments were closely correlated with antero-inferior shoulder instability.

Chronic Ankle Instability: Evaluation with Magnetic Resonance Arthrography, Magnetic Resonance Imaging, and Stress Radiography

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Submitted for consideration for oral presentation at BAMC Research Symposium, April, 1995

Harper, Michael T., CPT, MC. Chronic Ankle Instability: Evaluation with MR Arthrography, MR Imaging, and Stress Radiography

Introduction: Conventional imaging of chronic ankle instability (CI) has relied on stress radiographs (SR), which have reported sensitivities of 50-70%. This technique is relatively insensitive, and is dependent on the magnitude of the force applied, as well as on the patient's ability to withstand the force. Moreover, it results in a large amount of radiation exposure to both patient and physician. Magnetic resonance imaging (MRI) of the lateral ankle ligaments has been described; MRI may be less accurate in diagnosing ligamentous tears in CI than in acute injuries because edema, hemorrhage and effusion are usually absent. We hypothesized that Magnetic Resonance Arthrography (MRAG), a previously undescribed technique in the ankle that combines the primary sign of direct ligament visualization offered by MRI and the secondary sign of contrast extravasation, would be more accurate than MRI and SR. The efficacy of these three methods was compared in patients with CI.

Patients and Methods: We prospectively evaluated 17 patients with the history of CI by SR, MRI, and MRAG following intra-articular injection of Gd-DTPA. The imaging findings were correlated with surgical results, which were used as the standard of reference.

Results: The anterior talofibular ligament (ATF) was abnormal in 14, and the calcaneofibular ligament (CF) was abnormal in 10 patients. SR were abnormal in 8 (57%) patients. MRI detected the ATF tear in 7 (50%) and the CF tear in 5 (50%) patients. MRAG detected the ATF tear in 14 (100%) and the CF tear in 9 (90%) patients.

Conclusion: MRAG was more accurate than MRI and SR in diagnosing chronic ligamentous injuries. MRAG enabled direct visualization of abnormal ligament morphology. Secondary signs including contrast extravasation into the soft tissues and peroneal sheath were present. MRI and MRAG also demonstrated associated injuries, including tendon tears, osteochondral fractures and fragments.

CT EVALUATION OF INTRATHORACIC LYMPHOMA

Herman I. Libshitz, M.D.

The staging and follow-up of Hodgkin's Disease (HD) and non-Hodgkin's Lymphoma (NHL) depends on imaging. Intrathoracic involvement is more common in HD than in NHD. The chest radiograph remains the basic examination for evaluation of intrathoracic disease. It demonstrates moderate to extensive adenopathy readily but there is no question that CT is much more sensitive in demonstrating subtle disease.

Value of CT

CT of the thorax in Hodgkin's Disease (HD) can change both the staging and the treatment in the disease. The change is usually an upstaging due to identification of additional nodal groups or evidence of extranodal extension of disease outside of the lymph nodes. Less frequently CT will disprove the presence of suspected disease and alter therapy in this way.

In Castellino's series dealing with the effect of CT on therapy in HD patients, treatment was altered in 9.4% (19 of 203) of patients. The group in whom therapy was most frequently altered was those receiving radiation therapy alone. Nine of 65 (13.8%) in this group had treatment altered. In those receiving both chemotherapy and radiotherapy, therapy was altered in 10 of 122 (8.2%) patients. CT scans had no influence on the therapy decisions in those patients treated with chemotherapy alone.

Additionally 14 of 47 (30%) patients with "normal" chest radiographs were found to have occult disease at CT. However, in only one of these 14 patients was treatment altered. This serves to make the distinction between new information and information that alters therapy.

Based on the data above CT of the thorax is recommended in all patients with HD who are to receive radiotherapy either alone or in combination with chemotherapy. CT of the chest has also been of benefit in staging of patients with (NHL). However, the NHLs are generally treated by chemotherapy and the detection of small foci of disease may be less critical than in HD. CT is also extremely valuable in the follow-up of patients with lymphoma.

With few exceptions magnetic resonance imaging (MR) has not demonstrated an advantage over CT in evaluating intrathoracic lymphoma. MR has not yet been able to satisfactorily distinguish between viable and sterilized tumor.

APPEARANCE

Mediastinum

All of the mediastinal nodal groups except the anterior diaphragmatic (paracardiac) and posterior mediastinal nodes are more frequently involved in HD than NHL. Typically, HD involves nodes by contiguity. The superior mediastinum is almost invariably involved before or in association with hilar or lower mediastinal disease. HD infrequently presents with bilateral hilar and right paratracheal adenopathy similar to sarcoidosis. Isolated hilar adenopathy is only rarely seen.

Intrathoracic disease of NHL may follow the patterns described for HD. However, contiguity of nodal disease typical for HD is frequently absent in NHL. Mediastinal masses of large cell lymphoma may mimic HD, but atypical intrathoracic presentations, particularly in small cell lymphoma, are not unusual.

Significant tracheobronchial compression by the nodes in HD is not common but can be seen with bulky disease. Bulky mediastinal disease is less common in patients with NHL. Primary bronchogenic carcinoma is more likely to cause tracheobronchial narrowing than HD. Superior vena cava syndrome and phrenic nerve paralysis also are more common in bronchogenic carcinoma than in HD.

Involvement of the paratracheal nodes and/or prevascular nodal groups is the most common thoracic lymph node manifestation and occurs in 84% of patients with HD. Hilar involvement ranks next and is seen in about 28% of patients. These are followed by subcarinal nodes (22%), cardiophrenic angle nodes (8%), internal mammary nodes (5%). Castellino considered any node >1.0 cm in transverse diameter abnormal. Unfortunately, there are no absolute size measurements available for any mediastinal nodal groups. There is usually no question of nodal involvement when the adenopathy is bulky, but this determination can be an extremely difficult question for paratracheal, tracheobronchial angle, prevascular and subcarinal nodes in the 1 to 2 cm range.

Subtle changes in the left prevascular region may be appreciated on the conventional radiograph by obscuration of all or part of the aortic knob on the P-A projection. A convex bulge into the lung at the junction of the descending aorta and the left pulmonary artery also is an indication of prevascular adenopathy. The area may be concave or straight, but should not have a convex bulge.

The identification of subtle right tracheobronchial and right paratracheal adenopathy may be quite difficult. Using various criteria with chest radiographs, adenopathy could only be identified

in 89% of patients who had nodes >15 mm. The increased sensitivity of CT is evident.

In Castellino's series, 7% of patients with HD had adenopathy in the paratracheal or prevascular areas identified with CT alone. In 77% the disease was evident with both CT and chest radiographs.

Subcarinal adenopathy is the most difficult to identity of the more frequently involved nodal groups unless the disease is extensive. Only 25-33% of patients with subcarinal adenopathy will be identified on chest radiographs. CT is far more helpful.

Internal mammary adenopathy is not usually seen in frontal chest radiographs unless bulky and is more frequently seen in the lateral view immediately behind the sternum. Even in the lateral view, internal mammary nodes are not identifiable unless moderately enlarged. At CT we consider the node enlarged when it is larger than the accompanying vessels.

Anterior diaphragmatic adenopathy is also more frequently seen at CT than on conventional studies. The paracardiac nodes (the medial component of the anterior diaphragmatic group) when significantly enlarged are seen as masses in the cardiophrenic angles. The more lateral anterior diaphragmatic nodes are rarely seen except with CT. The identification of anterior diaphragmatic nodes suggests that they are enlarged. Identification of these nodes is particularly important in patients to be treated with radiation therapy. These nodes might otherwise be excluded from the radiation portals in order to spare the heart. The nodes are a not uncommon site of recurrence.

The posterior mediastinal nodes include the paraspinous nodes, and the periesophageal and periaortic nodes. These are much more frequently seen at CT. Identification of nodes in this area speaks for their enlargement.

Thymic enlargement is not uncommon in HD and has been found in 30% of patients who had intrathoracic disease at presentation. Identifying thymic enlargement is easier at CT than with conventional chest radiographs. Following therapy, enlargement can be the result of recurrent disease, thymic rebound, or the development or persistence of thymic cysts.

Lung

Pulmonary parenchymal involvement in HD is usually associated with hilar and/or mediastinal nodal disease. In a review by Kaplan of 340 consecutive previously untreated patients, there were "no cases in which HD has been confined to the parenchyma of the lung without associated hilar and mediastinal adenopathy." Filly

found a similar association.

In a series of 203 patients with HD investigated at presentation with CT, 8% had pulmonary involvement and all had hilar and/or mediastinal nodal disease. All cases of parenchymal involvement were seen with chest radiographs although CT scans, at times, showed more extensive parenchymal disease. CT will occasionally identify hilar and/or pulmonary parenchymal disease extending from the hili that is obscured by bulky mediastinal mass.

In the untreated patient with HD, a lung lesion without mediastinal disease should be evaluated for a second process. In the treated patient, relapse in the chest is commonly seen in the lungs. With recurrent disease, pulmonary involvement without nodal disease is more common than it is at presentation. Primary pulmonary parenchymal lymphoma is rare.

In NHL, pulmonary or pleural lesions may be seen without mediastinal or hilar adenopathy. However, it is more common to find some manifestation of mediastinal disease when the lungs are involved.

The bronchovascular bundle is the area most frequently involved. Radiographically ill-defined densities following the bronchovascular bundles are seen. End on, the involvement appears as nodules while in profile it appears more linear. Discrete nodules can also be seen. They may be less well defined than typical pulmonary metastasis and can be seen in both HD and NHL as a stage IV manifestation in an untreated patient or as an indicator of relapse. Cavitation may occur.

The appearance of pulmonary lymphomas also include consolidation and atelectasis of a lobe or segment, due to bronchial compression by nodes or endobronchial disease. If the lung clears and is re-aerated within a few days of initiation of therapy, the findings were likely due to atelectasis. Actual invasion of the lung requires longer to clear, and usually leaves some residual scarring and loss of volume. Air-bronchogram may be present due to masses about the patent bronchi.

The least common parenchymal manifestation of the lymphomas is an interstitial pattern which represents disease along the peripheral lymphatics. This is so uncommon, particularly in HD, that other causes for the radiographic appearance should be excluded.

Pleura & Pericardium

In the Stanford experience, 7% of patients with HD and 10% of patients with NHL had pleural effusions on presentation. However,

at CT it is not unusual to see small amounts of fluid in patients with HD, especially in those with large mediastinal masses. The effusions clear promptly with therapy and are thought to be benign and related to lymphatic and/or venous obstruction.

Solid pleural involvement at autopsy (26%) and 30% in a group of patients with advanced or recurrent disease has also been reported. It is far less frequent at presentation. It may occur anywhere along the pleural surface and is frequently accompanied by fluid. It may appear as plaques, as discrete nodules, or both in conjunction with other pulmonary parenchymal involvement or alone. Solid pleural involvement may rarely be the sole manifestation of recurrent disease.

Pericardial effusion may be seen in patients with lymphoma, particularly those with HD with large mediastinal masses. These usually resolve quickly following institution of therapy. Frequently, masses are seen along the pericardium with intact fat planes within the pericardium and actual pericardial invasion is not often seen.

Chest Wall

Chest wall involvement of Hodgkin's disease is not uncommon. It may be either an initial manifestation of the disease or of recurrence. It was seen in 6.4% (13 of 203) of new patients in Castellino's series and the disease was identified only at CT in 12 of 13.

The most common type of chest wall involvement in HD is direct extension from anterior mediastinal disease with internal mammary node involvement. Occasionally masses are seen beneath or between the pectoral muscles without contiguous mediastinal or axillary adenopathy. Direct invasion may occur in association with pleural masses. Soft tissue presentation without intrathoracic adenopathy may occur and is more common in NHL, particularly large cell lymphoma.

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IMAGING OF AORTIC DISSECTION AND ANEURYSM

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I. Aortic Dissection

Dissection is the most common acute emergency of the thoracic aorta, and accurate diagnoses and classification are essential for both diagnosis and management.

Type A dissection (Debakey Type 1 and 2) involves the ascending aorta. Complications include proximal extension into the aortic valve (leading to aortic insufficiency), the coronary arteries (potential for myocardial infarction), and intrapericardial rupture (potential cardiac tamponade). Surgical treatment is usually mandatory; survival is $<5\%$ without surgical therapy but $>95\%$ with appropriate early intervention.

Type B dissection (Debakey Type 3) involves the descending thoracic aorta and is usually managed medically with antihypertensive drugs unless there is proximal extension, ischemia to major organs, or rupture.

Noninvasive (nonangiographic) diagnosis of aortic dissection has become the technique of choice for several reasons. The clinical diagnosis is frequently in doubt; Type B dissections are managed medically and rarely require angiographic evaluation; and angiography is less accurate in many cases than cross sectional imaging. When strong clinical indicators of Type A dissection exist (e.g., new aortic insufficiency murmur or pericardial effusion accompanying severe chest pain) some investigators go directly to aortography which can help evaluate aortic insufficiency, coronary artery and other branch vessel involvement, and entry and reentry points. Angiography also carries the potential for catheter-induced damage to the vessel and compromise of renal function secondary to the iodine contrast load.

Echocardiography using a transesophageal transducer is a recent innovation with reported sensitivity and specificity for descending thoracic aortic dissections of over 90%. Aortic insufficiency and hemopericardium are also accurately depicted. Limitations include suboptimal evaluation of the coronary arteries and other branches and the abdominal aorta and its branches. The main advantage of transesophageal echocardiography is probably in the acute evaluation of the hemodynamically unstable patient.

Computed Tomography

CT, in experienced hands and using modern scanners with a bolus injection of contrast material, is a simple and extremely accurate means of diagnosing aortic dissection. CT is

faster, less expensive, and more readily available than MRI. A bolus injection of contrast is essential for accurate diagnosis, but contrast doses are much less than with angiography, particularly when using modern high speed scanners. A preliminary scout radiograph of the chest is taken along with several noncontrast axial sections. The latter are useful to identify displaced calcified intimal flaps and also the hyperdense clotted false lumen that may be the only indicator of dissection in patients who lack flowing blood in the false lumen. Subsequently, a dynamic bolus-enhanced series of axial scans is performed throughout the thoracic aorta, with continuation into the abdomen if a dissection is detected. Modern scanners, even without "helical" modification, are capable of scanning the entire thoracic aorta with a single bolus of contrast (2 ml per second) with the total contrast load not exceeding 100 ml.

CT findings include, on precontrast studies, internal displacement of calcified intimal flap, high density of acutely thrombosed false channel, and pericardial and/or mediastinal hemorrhage.

The most diagnostic feature of aortic dissection is identification of two contrast-filled channels with an intervening lower attenuation intimal flap. The false lumen is usually anterior and to the right in the ascending aorta, superior and slightly posterior in the arch, and posterior and to the left in the descending aorta. The true lumen is often compressed and deformed rather than dilated. (The term "dissecting aneurysm" is a misnomer.) False positive and false negative studies are minimal with proper attention to technique, especially a bolus infusion of contrast material, and avoidance of artifact-producing structures such as ECG leads overlying the chest. An intimal flap can be mimicked by normal structures such as the left innominate vein or superior intercostal vein. Pseudodisplacement of intimal calcification can be caused by partial volume averaging of a tortuous aorta or calcification within luminal thrombus in aneurysms. The latter is rarely a problem due to the coexisting presence of true intimal calcification on the periphery of the aneurysm. Thickening of pleura and atelectatic lung adjacent to the aorta can mimic a thick aortic wall.

Aortic valve insufficiency and the status of the coronary arteries are not accurately depicted by CT. However, involvement of the great vessels and abdominal aorta, pericardial hemorrhage, and perfusion to vital organs such as the kidneys are well assessed with contrast-enhanced CT.

MR Imaging

MR techniques for diagnosing aortic dissection are rapidly evolving and include spin-echo, cine, and velocity-encoded cine sequences. MRI is performed without intravenous contrast, making it safe for patients with compromised renal function. While imaging times are decreasing and monitoring capabilities improving, MR is still less attractive in the acutely ill patient than CT. Accuracy of MR diagnosis of dissection is probably comparable to that of CT, assuming optimal equipment performance and an experienced observer. The potential for artifacts and misinterpretation is much greater, however, in my opinion.

As with other imaging modalities, the hallmark for dissection is visualization of an intimal flap. MR has the advantage over CT of demonstrating aortic insufficiency and shares the benefits of accurately visualizing pericardial hemorrhage and extension into the abdominal aorta. MR has the disadvantage of not visualizing intimal calcifications, making evaluation of type B dissections with a clotted false lumen difficult or impossible.

II. Aortic aneurysms

Abdominal aortic aneurysm is an increasingly common and important entity. Operative morbidity and mortality for elective surgery has diminished sharply, while rupture of an abdominal aortic aneurysm remains a deadly disease. Accurate evaluation of suspected AAA in the stable asymptomatic patient can be made by ultrasound. In the patient suspected of having a leak or rupture (AAA rarely "dissects"), CT is the imaging procedure of choice, assuming that the patient is stable enough to warrant any imaging procedure. Aortography has little or no role in the acute evaluation of abdominal aneurysms. Cross sectional imaging is more accurate than angiography in identifying the true size of an aneurysm, the presence of intraluminal thrombus, and the presence of a leak or rupture. Angiography rarely may be necessary to establish involvement of the renal arteries by the aneurysm.

The first sign of an impending or contained rupture is an asymmetric bulge along the posterolateral margin of the aorta where retroperitoneal fascial planes may temporarily contain the bleeding. We have seen cases present with chronic back pain where contained ruptures have massively enlarged the psoas muscles and even eroded the spine. Blood leaking from a ruptured AAA may dissect in any direction. Adherent posterior parietal peritoneum may result in massive hemoperitoneum with essentially no retroperitoneal bleeding, while the perirenal and anterior and posterior pararenal spaces are commonly involved. Injection of iodinated contrast material is not essential for diagnosis, although it may be useful to establish perfusion of vital organs.

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1 HRCT of Airways Disease

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2 Normal Anatomy

- Lung has greater than 300,000 airways
- Conductive Airway:
  - Trachea
  - Bronchi
  - Membranous (nonalveolated) bronchioles
- Transitional Zone
  - Respiratory bronchioles
  - Alveolar ducts
- Respiratory Zone
  - Alveolar Sacs
  - Alveoli

3 Small Airways

- Terminal (membranous) bronchiole
  - occurs between the 8th and 14th order airways
  - no cartilage
  - contains smooth muscle
  - # of divisions to alveolar sac 2 - 12 with 6 - 8 average
- Respiratory bronchiole
  - ciliated epithelium and alveolar cells
  - no smooth muscle

4 Lung Units

- Primary Pulmonary Lobule
  - all the alveolar ducts, sacs and alveoli, i.e. everything distal to the respiratory bronchiole
- Secondary (Reid) Pulmonary Lobule
  - the smallest discrete portion of lung surrounded by connective tissue. This will contain 3-5 terminal bronchioles with the transitional airways and parenchyma. (one secondary lobule contains 30-50 primary lobules)
- Pulmonary Acinus
  - that portion of lung distal to a terminal bronchiole (mean diameter of 6-10 mm)

5 Bronchiectasis: HRCT Protocol

- Kvp 120 , mA 140
- 1.5 mm thickness at 1 cm intervals
- Bone (high spatial frequency) algorithm
- Supine
- Inspiratory
- Range: apex to diaphragms
- Consider 20 degrees of cranial angulation of gantry per Grenier et al JTI 1993 ; 8: 213-229

6 Bronchiectasis

Types:

- Fusiform
- Varicose
- Cystic
- Traction

7 ☐ **Bronchiectasis: Etiologies**

- Postinfectious
- Bronchial Obstruction
- Pulmonary Fibrosis
- Mounier-Kuhn Syndrome
- Cystic Fibrosis
- Kartagener Syndrome
- Allergic Bronchopulmonary Aspergillosis
- Swyer - James - Macleod Syndrome
- Chronic Granulomatous Disease of Childhood
- Congenital Immunodeficiency Sydromes (IgG)
- Williams-Campbell Syndrome

8 ☐ **Allergic Bronchopulmonary Aspergillosis**

Clinical: hx of asthma, recurrent infiltrates, precipitating Ab's to A. fumigatus, elevated serum IgE levels, peripheral blood eosinophilia and increased aspergillus specific IgE levels.

HRCT Findings: central bronchiectasis (varicose or cystic) with an upper lobe predominance, mucoid impaction, distal atelectasis or hyperinflation.

Treatment: bronchiectasis dictates the need for aggressive corticosteroid therapy

9 ☐ **Cystic Fibrosis**

Clinical: the most common lethal genetically transmitted disease of white children, autosomal recessive with prevalence of 1/2000 live births. Caused by a mutation of a gene termed the CFTR (cystic fibrosis transmembrane conductance regulator), a chloride channel. Symptoms reflect a combination of bronchiectasis and obstructive airway disease.

Labs: abnormal sweat chloride, PFT's demonstrate progressive decreased DLCO with decreasing VC and FEV1 with increasing RV.

10 ☐ **Cystic Fibrosis**

HRCT Findings:

- bronchial wall thickening
- bronchiectasis (usually cylindrical occasionally varicose or cystic)
- upper lobe distribution
- +/- mucous plugs
- parenchymal atelectasis/consolidation
- eventual emphysematous change

11 ☐ **Williams-Campbell Syndrome**

Clinical: a congenital disorder of cartilage in the 4th to 6th generatiaon bronchi

HRCT Findings: cystic bronchiectasis distal to the third generation bronchi, emphysematous lung distal to the bronchi, with insp / exp HRCT ballooning and subsequent collapse may be demonstrated.

12 ☐ **Pulmonary Fibrosis**

- Increased elastic recoil leads to bronchial dilation on the basis of mechanical distortion
- Any process resulting in pulmonary fibrosis can thus result in traction bronchiectasis
- Clinically, this is most often seen with UIP
- In association with ground glass in UIP the presence of traction bronchiectasis

implies fibrosis rather than active alveolitis. Remy-Jardin M et al, Radiology 1993; 189: 693-698.

13 Bronchial Obstruction

Causes:

- foreign bodies
- stenosis / atresia
- neoplastic disease
- granulomatous disease
- broncholith

14 Bronchiolitis Obliterans: Clinical Classification

- Toxic-fume bronchiolitis obliterans
- Postinfectious bronchiolitis obliterans
- Bronchiolitis obliterans associated with connective tissue disease or organ transplantation
- Localized lesions with bronchiolitis obliterans
- Idiopathic bronchiolitis obliterans with organizing pneumonia

15 Bronchiolitis Obliterans

Two morphologic types:

- proliferative bronchiolitis obliterans (granulation tissue polyps filling the bonchiolar lumen)
- constrictive bronchiolitis (concentrically scarred, stenotic or obliterated bronchioles)

16 Proliferative Bronchiolitis Obliterans

Histology: a nonspecific reparative reaction to bronchiolar injury manifest by organizing intraluminal exudate of fibroblasts and a mucopolysaccharide matrix with mixed chronic inflammatory cells. This usually extends into the adjacent alveoli resulting in organizing pneumonia.

17 PBO: Assoc. Pulmonary Disorders

- Organizing DAD (ARDS organizing phase)
- Organizing infection (viral, mycoplasma, bacterial, fungal, PCP)
- Distal to an obstruction
- Organizing aspiration pneumonia
- Organizing phase of fume or toxic exposure
- Collagen vascular disease (esp. RA)
- Extrinsic allergic alveolitis
- Eosinophilic pneumonia (acute and chronic)

18 PBO: Assoc. Pulmonary Disorders

- Drug reactions
- Bone marrow, heart-lung transplant
- Diffuse panbronchiolitis
- Bronchiolitis obliterans organizing pneumonia

19 Bronchiolitis Obliterans Organizing Pneumonia

The commonest example of proliferative bronchiolitis

Clinical: prodrome of 2 - 12 weeks history of flu-like illness with cough, fever, and dyspnea. Patients age 40 - 60 yrs. No sex predilection.

Histology: that of proliferative bronchiolitis in association with organizing pneumonia in the alveoli.

20 ☐ **Bronchiolitis Obliterans Organizing Pneumonia**

HRCT Findings:

- air space consolidation / nodules (50% are subpleural)
- bronchial dilatation
- pleural effusion
- ground glass attenuation

21 ☐ **Constrictive Bronchiolitis**

Histology: irreversible scarring and alteration of the bronchioles with a range of abnormalities to include complete fibrous obliteration, stenosis from mural/adventitial scarring, dilation with mucous stasis, acute or chronic mural inflammation, or acute lumenal inflammation. Distal airspaces may show obstructive changes.

22 ☐ **CB: Assoc. Pulmonary Disorders**

- Healed infections (esp. viral, mycoplasma)
- Chronic bronchitis,CF,asthma,bronchiectasis
- Healed toxic of fume exposure
- Collagen vascualr disease esp. RA
- Bone marrow, heart lung transplants
- Drug reaction (i.e. penicillamine)
- Healed DAD (healed ARDS)
- Chronic allergic alveolitis
- Diffuse panbronchiolitis
- Mineral dust airways disease
- Idiopathic

23 ☐ **Cryptogenic Bronchiolitis Obliterans**

Classic example of constrictive bronchiolitis

Clinical: a rare condition affecting primarily women age 40 - 60 yr. PFT's reflect severe progressive airway obstruction

24 ☐ **Cryptogenic Bronchiolitis Obliterans**

HRCT Findings:

- lobular/segmental decreased lung attenuation
- interspersed areas of normal/increased attenuation/frank consolidation
- peripheral or central bronchiectasis

25 ☐ **Panbronchiolitis**

Clinical: Japanese patient with progressive shortness of breath and history of chronic paranasal sinusitis. 75% are nonsmokers. M:F ratio of 2:1. Age 30 - 60

Lab: PFT's demonstrate principally obstruction with decreased DLCO

Pathology: aggregates of foamy and lymphoid cells within the walls of respiratory bronchioles, adjacent alveolar ducts and alveoli

26 ☐ **Panbronchiolitis**

HRCT Findings:

- centrilobular branching structures
- centrilobular nodules
- mosaic perfusion
- air trapping
- bronchial dilatation

27 ☐ **Asthma**

Clinical: increased resistance to movement of air within the intrapulmonary airways

Pathophysiology of airway narrowing:

- bronchi/iole wall smooth muscle contraction
- inflammatory airway edema
- lumenal occlusion secondary to mucus hypersecretion and plugging

28 Asthma

HRCT Findings:

- bronchial wall thickening (92%)
- bronchial dilatation (77% had at least one dilated bronchus, 36% of 429 bronchi assessed were dilated) Lynch DA et al, Radiology 1993; 188: 829
- focal / diffuse air trapping

29 Respiratory Bronchiolitis

Clinical: young heavy smoker complaining of cough and progressive shortness of breath. Equal sex predilection.

Labs: Mixed PFT's with mild obstruction and restriction. Decreased DLCO

Pathology: bronchiolocentric pigmented macrophages within the respiratory bronchioles, adjacent alveolar ducts and alveoli. Alveolar septa often show infiltration with chronic inflammatory cells but minimal fibrosis

30 Respiratory Bronchiolitis

HRCT Findings:

- normal (majority)**
- subpleural micronodules
- ground glass attenuation
- bronchial dilatation

**Normal HRCT due to the fact that respiratory bronchioles/ducts/alveoli are one to two orders of magnitude smaller than currently resolvable structures. A normal HRCT does not obviate the need for biopsy.

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How Did I Ever Miss That?
Problems in Visual Perception

Thomas S. Harle, M.D.

Visual perception is a complex process involving optical, anatomical, physiological, and psychological components. Previous studies showed even experienced radiologists sometimes have difficulty in perceiving abnormalities. Errors often stem largely from a failure to organize effectively the large number of shadows on the films. The learning process for radiologists depends on being able to call the important from the irrelevant. Errors in perception may be classified as due to: a) sampling and search; b) perception, detection, or recognition; and c) decision-making or interpretation. Cases will be presented which demonstrate errors.

THE RADIOLOGY OF TUBERCULOSIS - 1995

Albert S. Hale, M.D.

Following the introduction of effective antituberculous drugs in the 1950's, the incidence of tuberculosis (TB) began to decline sharply until 1985; since then, there has been an increasing incidence due chiefly to the advent of AIDS. Worldwide, 8,000,000 people develop TB each year, and 2,900,000 people die of TB. An estimated 1,700,000,000 are infected with TB (i.e., have a positive reaction to the PPD skin test), according to WHO estimates.

The disease is caused by Mycobacterium tuberculosis, and is transmitted by droplet nuclei. Transmission usually requires close personal contact or defective T-cell immunity, and is often transmitted among family members and other closed populations. Predisposing factors include:

HIV/AIDS
Diabetes
Mainutrition
Alcoholism and IV drug abuse
Old age
Prisoners
Residents of nursing homes and long term care hospitals
Homeless persons
Refugees and immigrants from high prevalence countries
Inner city residents
Hispanics and Blacks
Chronic diseases, including silicosis, sarcoidosis, renal failure, malignancies, etc.

The basic lesion of tuberculosis is the tubercle, a focus of granulomatous inflammation consisting of lymphocytes, epithelioid cells, macrophages, and giant cells. The tubercle may undergo necrosis ("caseation"); a cavity is formed when the tubercle erodes into a bronchus and expels its contents. The contents of the tubercle may be carried into other portions of the lungs by endobronchial dissemination, or may be expectorated and

swallowed. Spread of disease may also occur by direct extension, or by hematogenous or lymphatic dissemination. The tubercle heals by fibrosis, encapsulation, scar formation, and calcification. "Healed" lesions of tuberculosis, even when calcified, may contain viable tubercle bacilli. Although over 80% of patients have lung involvement only, TB can involve practically any organ or tissue in the body. Common sites of extrapulmonary involvement are the chest wall, skeleton, genitourinary tract, and gastrointestinal tract.

Clinically, pulmonary tuberculosis presents in two forms:

1. Primary (or "childhood") TB: Often involves children and may cause no symptoms. Most cases are self-limited and undetected. Radiographically, primary TB presents as lymphadenopathy and pulmonary infiltrates, often involving the right middle lobe. The parenchymal focus is termed the Ghon focus, and the combination of parenchymal and lymphatic foci is termed the Ranke complex. The foci may calcify.
2. Reinfection (or "adult" ar "secondary") TB: Usually preceded by primary TB by months, years, or decades. In the immunocompetent patient, the disease typically presents radiographically as linear and finely nodular infiltrations, or as patchy confluent infiltrations, in the apical and posterior segments of the upper lobes and in the superior segment of the lower lobes. Cavitation occurs in approximately 50% of patients. Other presentations include segmental or lobar consolidation, multiple small nodules (best seen with CT), or macronodular structures. Effusion may occur in either primary or reinfection TB,

In AIDS, unusual radiographic presentations are the rule. The chest radiograph may be normal, or resemble primary tuberculosis with predominant lymphadenopathy. Infiltrations are often rather sparse, and do not occur in the familiar upper lobe/superior segment distribution. Cavities are unusual. Extrapulmonary dissemination is frequent. The PPD skin test is usually negative.

Miliary TB is massive hematogeneous dissemination, with lesions occurring in all portions of the body, not just the lungs. Typically lesions are 2-4 mm in diameter and uniformly distributed throughout the lungs. The lesions may not be visible in chest radiographs for 4-6 weeks, and the patient may die before the radiograph becomes positive. The lesions clear with treatment and do not calcify. A pleural effusion may occur in some cases. Miliary disease may occur in either primary or reinfection tuberculosis, and in AIDS.

Several thoracic complications of tuberculosis are encountered, including:

1. Pneumothorax and/or tuberculous empyema: Due to rupture of a tuberculous cavity into the pleural space. A bronchopleural fistula may result.
2. Chest wall abscess ("cold" abscess)
3. Empyema necessitans: Chest wall abscess ocurring due to extension of tuberculous empyema through the chest wall; may occur simultaneously with acute empyema, or years later.
4. Hemoptysis: May be due to a ruptured Rasmussen's aneurysm (erosion of a pulmonary artery branch by an enlarging tuberculous cavity, producing a pseudoaneurysm).
5. Fungus ball (aspergilloma): Saprophytic growth of Aspergillus in a pre-existing tuberculous cavity, forming a mobile spherical structure with a typical overlying air crescent.
6. Bronchostenosis: Due to endobronchial tuberculosis. Wall thickening with concentric narrowing of lumen, long segment. May have distal atelectasis.
7. Broncholithiasis: Calcified node or granuloma erodes into a bronchus. May cause hemoptysis, bronchial obstruction, or reactivation of tuberculosis.

Extrathoracic tuberculosis can involve almost every organ or tissue in the body, and can imitate virtually any other process. Frequently encountered locations include:

1. Central nervous system: Meningitis with communicating hydrocephalus, focal cerebritis, tuberculomas with ring enhancement; meninges may show marked contrast enhancement.

2. Larynx: Vocal cord thickening; may mimic carcinoma of the larynx.

3. Cervical lymphatics (scrofula): CT shows enlarged lymph nodes with typical peripheral contrast enhancement. Late calcification.

4. Gastrointestinal tract: Ileocecal region in 90% of cases. Wall thickening and matting of enlarged nodes to form inflammatory mass.

5. Renal: Hematonegeous dissemination from lung in most cases, with formation of multiple tubercles in kidney; may progress to destruction of papillae with irregular cavities seen adjacent to calyces. If not treated, may eventually destroy the entire kidney. Tuberculous autonephrectomy = small calcified kidney. Ureters and bladder may become involved.

6. Serous surfaces: Pleura may become involved by rupture of cavities into pleural space, or by hematogeneous route. pericardium and peritoneum chiefly by hematogeneous dissemination. Manifested by pleural, pericardial, or ascitic fluid.

7. Spine (Pott's Disease): Initially involves anterior aspect of a vertebral body, usually in lower thoracic or upper lumbar area; often involves two adjacent vertebrae and intervening intervertebral disc. Progressive bone destruction and collapse of vertebral bodies, leading to gibbus formation. May spread into adjacent musculature, causing abscesses. May spread up or down spine under anterior or posterior longitudinal ligaments. May extend into epidural space,

8. Skeleton: Femur, tibia, and small bones of hands and feet most commonly involved. Osteolytic foci with fairly well defined rounded margins and surrounding sclerosis. May enter nearby joints.

9. Tuberculous dactylitis: Involvement of small tubular bones of hands and feet. Soft tissue swelling, expansion of bone, and periosteal new bone are seen.

10. Other organs and tissues: Tuberculosis has been reported in virtually every organ, including:

Skin	Ovaries	Fallopian tubes
Mastoids	Uterus	Middle ear
Sinuses	Eyes	Seminal vesicles
Prostate	Adrenals	Vas deferens
Liver	Spleen	Salivary glands
Esophagus		

FOOTNOTES

1. Tuberculosis synonyms:

Consumption
Galloping Consumption
White plague
"Captain of all ships of death"
"The King's evil" (Scrofula)

2. Famous people who had tuberculosis:

Composers Frederic Chopin and Niccolo Paganini
Poets Elizabeth Barrett Browning, Percy Bysshe Shelley, and John Keats
Novelists Charlotte and Emily Bronte
Actress Vivien Leigh (Scarlett, in Gone With the Wind)
Mimi, the heroine of Puccini's La Boheme
Violetta, the heroine of Verdi's La Traviata
Camille, the heroine of Alexandre (fils) Dumas' Camille

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3D Imaging of Head & Neck Trauma

Dr. Lennard A. Nadalo

While plain film x-rays and axial CT provide important information concerning the evaluation of trauma, image manipulation (post processing) of CT images obtained following serious injury results in more complete understanding of the injury, better defines the surgical plan and improves patient care. This is particularly true in the assessment of the complex anatomic relationships of the skull and face. The presentation will include an introduction to workstation terminology. The clinical work presented represents experience using two different independent workstations (Allegro and VoxelQ) in the post-processing of CT images of trauma of the face, cranium, and neck.

Goals for Imaging Trauma

- Define the area and nature of injury.
- Describe the extent of injury.
- Relate important landmarks.
- Evaluate post-op anatomic relationships.
- Review / Rx post traumatic complications.

Methods of Imaging Trauma

- Plain Film x-rays: Review First
- Linear or Poly Tomography
- Sonography including Duplex
- CT using Spiral Mode
- Angiography for Chest / Extremity
- MRI for acute spinal cord injury
- 2D and 3D Reconstructions

Introduction

- Conventional Images: 2D.
- Most anatomic concepts: 3D.
- Analysis in 3D results in better understanding.
- Measurement of irregular objects.
- Organization of large amounts of information.

The Basics

- Basic computer/imaging terminology.
  - Pixels and Voxels
  - MIPS
  - Ethernet and Bandwidth
- CT, MR, and SPECT

- » Individual Image is 2D with slice thickness.
- » The thickness can't be appreciated.
- » The depth of the pixel remains as information.

Types of Remote Workstations

- View Only - Slave Monitor
- Dependent - Shares CPU
- Independent - Remote CPU
- Network - Limited Shared Resources
- PACS - Fully Shared Resources

Workstation Functions

- View Only - Quick Review
- Dependent - Reconstructions
- Independent - Analysis
- Network - Remote Interpretations
- PACS - Archival and Viewing Functions

Connectivity

- Same Manufacturers most successful.
  - » Different image types.
  - » Networking standards.
- Seek common image format.
  - » GIF, TIFF, VECTOR
- DIACOM 3.
  - » Common standard?

Methods

All images were obtained using the thinnest scan sections practical with a 50% overlapping table advancement. The data sets were processed on either the VoxelQ or Allegro workstations. The results and the images were reviewed ***directly*** with the referring surgeons.

Methods

- Obtain multiple overlapping thin images.
  - » i.e. 3mm thick, 2mm interval
- Reduce artifacts to a minimum.
  - » movement, metal, barium
- Transfer data to workstation.
  - » Ethernet, CD WORM, Tape

Cervical Spine Trauma Basics

- Trauma to the upper Cervical Spine
  - x-rays
  - Flex-Exten
  - CT for ?
- Reconstructions
  - Dens FX
  - Subluxations

Workstations

- CPU and Memory
  - CISC vs RISC
  - Linear memory vs Graphic Engine.
- Video Display
  - Large display with high resolution.
- Applications
  - 2D (MPR)
  - 3D (Solid, shaded, and cut)
  - Angio, Dental, Overlays

Lower Cervical Trauma

- **Difficult in Large Pt**
- Alignment: Lat. View
- Spiral CT Useful
  - Axial Images
  - 2D Sagittal
  - 3D Oblique
  - 3D Cut Away

Workstation Standards

- CPU
  - SPEC CPU benchmarks: SPEC92
  - Whetstone, 1976
  - Dhrystone, 1980's
- Video Display
  - Size of Screen: (larger), Dot Pitch: (smaller).
  - Refresh Rate: (faster).
- Operating System: UNIX

Application Standards

- Interpolation: math model
- Gray scale or color depth
- Graphic Array Processor
  - ISG Allegro

- Linear Memory
  - Voxel Q
- Shared Resources
  - CT or MR CPU and Memory: may slow imager.

Voxel Q

- Sun Sparc Host
- 64 MB Object Memory Board
- 1000 MIP Volume Processor
- 104 MB Interleaved Memory
- 10 Million Trilear Interpolations per second (TRIPS)

Voxel Q (cont.)

- PQ Imager
- PQ Disk.....Ether Net LAN
- VoxelQ Disk
- Sun
- Object Memory Board
- Voxel Buffer.....Frame Buffer
- Display: 910 x 910 View Port

Color Functions

- Cardiac and regional blood flow
- Contour and region segmentation
- Multimodality superposition
- 3-D surface presentation
- Region/volume of interest identification
- Scale representation
- Adds another "dimension"

Display Functions

- Multiple viewport display
- High resolution monitors
- Cine with fusion rate
- "Slide show" presentation
- Alternative "back lighting"
- SVHS Tape useful for referral
- Tape useful for patient education

Applications: 2D (MPR)

- Satisfactory for most cases.
  - Isolated Fractures.
  - Spinal Stenosis.
- Ease of performance.

- Daily Use.
- Few Commands.

- Planar display.
  - Less well understood.
  - Less well accepted.

Applications: 3D (Innerview)

- **More demanding technique.**
  - Thin over lapped sections.
  - No false data tolerated.
- **Sensitive to artifacts.**
- **Understanding rather than new information.**
  - Fractures are already known.
  - Pre Op Planning.
  - Post Op Evaluation.

Applications: 3D (Surface)

- Demanding Technique
- Surface Shading
- Rotation Important
- Cut Views Needed
- Useful to Locate Fragments
- General Orientation

2D Clinical Examples

- **Cranio-Facial Trauma.**
  - Coronal from Axial Images
  - Bone fragments.
- **Pelvic Trauma.**
  - Acetabular Roof.
  - Complex, multiple.
- **Multiple Trauma**
  - Cranial, C Sp., Chest, Abd & Pelvis, CT Angio

3D Clinical Examples

- **Cranio-Facial Trauma.**
  - Complex orbital Fx
  - Mandibular Fxs
- **Pelvic Trauma.**
  - Acetabular / Femoral Relationship.
  - Degree of Displacement.
  - Over View.
- **Multiple Trauma**
  - Cranial, C Sp., Chest, Abd & Pelvis, CT Angio

Angiography

- Consider CT Angio in Trauma.
- MR and CT Angiography
  - Maximum Intensity Projection.
  - Flow related artifacts.
  - Tends to over estimate stenosis.

CT Angiography

- Rapid injection rates are needed for MIP.
- 3 - 5 cc. / sec.
- Consider Central Venous Line.
- 3D, Inner View, vs. MIP, vs. MPR
- MPR requires less contrast density.
- 1.5 - 2.0 cc. / sec.

Dental

- Post Facial Trauma
- Dental Implant Preview
  - Maxillary and Mandibular bone thickness.
  - Depth of nerve canal.
  - Anomalies
  - Effects of Prior Infections

Multimodality

- **Interventional Radiology**
  - Planning, Guidance
  - Follow-up Assessment
- **Multimodality Overlay**
  - CT, MR, NucMed.
  - Stroke vs ischemia.
  - Tumor staging.

Multimodality (cont.)

- Anatomic/physiologic correlation
- Planar measurements
- Surgical planning
- Therapy planning
- Multimodality z-axis linked review
- Quantitative tumor/pathology analysis

Analysis of the information provided by post-processed CT images was performed by comparison of plane x-rays, axial CT images, and post-processed 2D and 3D images. We attempted to answer three questions:

1. Does post-processing result in distortion?

2. In what specific ways does 2D and 3D CT contribute to patient care?

3. Is this art or science?

Does 2D & 3D Distort?

- The further removed from the anatomy, the move likely that distortion, or artifact will result in error in measurements.
- 2D & 3D Images are Tertiary in origin.
- **Comparison with known structures needed.**

Material & Methods

- Dry Prep Human Skull
- 2 mm. thickness at 1.5 mm
  - 0.5 mm overlap
- 2D & 3D Reconstructions
- Measurements on the VoxelQ & Allegro
  - Foramen Magnum
  - Zygomatic Arch
  - Foramen Ovale

Material & Methods (cont.)

- Measurements of Skull
  - Foramen Magnum
  - Zygomatic Arch
  - Foramen Ovale
- Comparison: Skull, Allegro, VoxelQ
  - Foramen Magnum
  - Zygomatic Arch
  - Foramen Ovale
- Error Analysis

Does post-processing result in distortion?

In order to answer the first question we performed thin section CT of a dry, human skull. Multiple methods of reconstruction and reformatting were applied to the axial data. The results of our investigation will be presented.

In what specific ways does 2D and 3D CT contribute to patient care?

A review of cases of head and neck trauma evaluated at Methodist Medical Center since we began to apply workstation analysis (1992 - 1995) indicates the following:

1. Additional information which is provided by 2D Multiplanar Reformatting is useful to both radiologists and surgeons but is of primary interest to radiologists. Images which are three dimensional (surface 3D and Inner View) are of primary interest to the surgeons.

2. The more complex the injury, the more valuable was the additional information which was provided by both 2D and 3D images.

3. The value of the reformatted and reconstructed images is increased by consultation between the managing surgeon and the radiologist.

. ***Is this art or science?***

1. Artifacts must be avoided if at all possible. Most "pseudo images" arise from streak or movement artifacts. Metal dental artifacts should be removed prior to imaging.

2. The more "cutting and pasting" the operator must perform, the more likely that an artifact will be generated. Disarticulation and the removal of surface structures must be used with care.

3. This work is most properly performed by the radiologist or by a very well training technologist with much involvement by the radiologist. Frequent comparison with the axial CT and with plain film x-rays is necessary to avoid "pseudo" fragments or "pseudo" fractures.

Results

- **Systematic Error ?**
  - Allegro
  - VoxelQ
  - Dry Skull
- **Random Error ?**
  - Allegro
  - VoxelQ
  - Dry Skull

Summary: Imaging of Solids

- Understanding complex injuries.

- Increased Confidence: Surgeon.
- Tumor Staging.
- Radiologist can MEASURE VOLUME.
- Need for Standardization.
- Beware Artifacts.
- Art meets science.

Is it worth it?

The effort and expense required to perform 2D and 3D CT imaging of trauma provides a better understanding of the nature of complex injury and can result in improvements in patient care provided that the following guidelines are met:

1. ***Garbage in: garbage out***. High quality, thin sectioned, over lapping axial images are needed.

2. ***Avoid Pseudo Lesion.*** The radiologist must be actively involved in the entire process.

3. ***Communicate.*** The images are complex and the findings are best communicated directly to the operating surgeon.

Where do we go from here?

- On-line Patient Record.
  - » Fracture Alignment
  - » Post-Op Complications
- PACS Networks.
  - » Better Communication.
- Fully Digital Image Archives.
- Common final archive media.

CT Evaluation of Blunt Abdominal Trauma

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Introduction

Blunt abdominal trauma remains one of the most frequent causes of morbidity and mortality particularly affecting children and young adults. Clinical evaluation of patients is unreliable in the diagnosis and management of trauma, leaving an important role for imaging. Radiography plays an essential role in initial screening of the trauma patient for life threatening injuries such as cervical or pelvic fractures and pneumothorax. Abdominal films may suggest hemoperitoneum but are insensitive and nonspecific. Excretory urography retains an important role in evaluation of suspected isolated renal trauma, and contrast studies of the gut accurately depict most traumatic injuries. However, even these well established imaging studies have assumed a less important and less frequent role since computed tomography (CT) has become established.

CT is indicated in evaluation of victims of blunt abdominal trauma who are hemodynamically stable following initial evaluation and fluid resuscitation. The victim of abdominal or multiple trauma who is not stable is best evaluated by diagnostic peritoneal lavage (DPL). In such cases DPL can be done quickly and relatively inexpensively in the emergency department or in the operating room if preparing for urgent extraabdominal surgery, as for head or orthopaedic trauma. For patients who are stable, including those with an unreliable physical exam (e.g. intoxication, head or spine trauma) CT has several advantages over DPL; CT is non-invasive, detects both intraperitoneal and extraperitoneal injuries, and can usually depict the quantity and source of bleeding.[2,3] The greatest limitation of DPL is the lack of useful information provided by a "positive" or "equivocal" result in a hemodynamically stable patient. Such patients may have iatrogenic sources of bleeding (e.g. introduction of the DPL trocar), intraperitoneal blood from an extraperitoneal injury, or bleeding from a self limited minor intraperitoneal visceral injury. DPL has played a diminishing role in pediatric patients because "positive" results of DPL led to non-therapeutic laparotomy rates of about 33%; this is due largely to the effectiveness of nonoperative management of most abdominal injuries in children. There has been a growing trend toward nonoperative management of renal, splenic and especially hepatic trauma in adults as well. For these reasons CT is the most effective means of initially evaluating the trauma patient and in subsequent evaluation of patients managed nonoperatively.

Abdominal CT scanning technique is critical to success. A scanner capable of generating artifact-free sections in less than three seconds is mandatory for optimal results. All extraneous artifact-producing objects should be removed from the scanning field or immobilized against the body. Examples include ECG leads, IV tubing, and fractured arms. Following endotracheal inbutation a nasogastric tube should be used in obtunded or seriously injured patients to evacuate stomach contents prior to scanning; following installation of oral contrast medium the NG tube is withdrawn into the distal esophagus to eliminate it as a source of artifact in the upper abdomen. Alert patients are asked to drink several hundred ml of iodinated contrast medium to opacify stomach and gut. This is a safe

procedure and is essential for accurate diagnosis of bowel, mesenteric, and pancreatic injuries.[4] Intravenous contrast medium is used to better detect visceral lacerations, vascular injuries, and renal collecting system disruption. We use a bolus infusion of 140 - 180 ml of 60% contrast (automated injector, 2 - 3 ml/sec) in adults, and 2 ml/kg in children. Nonionic or low osmolar agents are preferable to decrease untoward reactions, including vomiting.

Sections are taken from the top of the diaphragm to the iliac crest by 1 cm intervals, and through the symphysis by 2 cm intervals. Some authors advocate non-contrast scans through the splenic area to avoid the problem of the isodense perisplenic hematoma that is rarely encountered after contrast infusion. This may be a useful adjunct, particularly early in one's experience in interpreting trauma CT scans.

Hemoperitoneum

CT accurately detects any clinically significant amount of intraperitoneal bleeding, and can quantitate the bleeding better than diagnostic peritoneal lavage.[6] In most instances CT also indicates the source of bleeding by demonstrating the visceral laceration itself and/or a "sentinel" clot sign.[7] The sentinel clot refers to a focal heterogeneous collection of high density (CT attenuation >60 HU) that tends to accumulate at the site of bleeding. Liquified nonclotting blood flows freely to the dependent portions of the peritoneal cavity, including Morison's pouch, paracolic gutters, and the pelvis. Such blood has an attenuation of 35 - 50 HU unless it has been present for more than 48 hours, has occurred in a chronically anemic patient, or is being diluted by ascites, urine, bile or bowel contents.

Several CT signs have been reported as important clues to significant hypovolemia and hypotension, including constricted major arteries, and a flattened inferior vena cava.[8,9] Splenic enhancement may be decreased, presumably by vasoconstriction, and may simulate splenic injury. Conversely, abnormally bright and persistent enhancement of the bowel wall, adrenal glands and kidneys may be seen. These signs are indicative of severe hypovolemia even if a supine brachial blood pressure reading is said to be normal, and urgent fluid resuscitation is indicated.

Visceral Injuries

The spleen, liver and kidney each has been reported as the most common visceral injury in blunt trauma in various studies. Injuries to these organs are easily and accurately demonstrated by CT, with sensitivity and specificity of about 95% in experienced hands. The major focus of recent investigations has been to correlate specific CT and clinical criteria in an attempt to manage traumatized patients.

The efficacy of nonoperative management of most abdominal injuries in children has been well established. The positive byproducts of reduced hospital stay and expense, decreased blood transfusion requirements, decreased complications (delayed bleeding, infection, urine or bile leaks), and greater salvage of functioning parenchyma are strong inducements to apply the nonoperative approach to adult patients whenever possible.

CT accurately demonstrates the important elements of splenic trauma including the extent of parenchymal laceration or hematoma, perisplenic or subcapsular hematoma, and intraperitoneal bleeding.[10] These elements can be combined in a "staging" scheme, minor variations of which have

been proposed or discussed by several investigators.[11,12] Variable predictive values have been reported, but a consensus has developed that the ultimate management decision is based on clinical criteria, grounded in close monitoring in a trauma center prepared to mobilize for urgent surgery should it be warranted. Nonoperative management of splenic trauma in adults is growing in popularity but remains risky unless carried out in such a controlled medical environment.

Nonoperative management of hepatic trauma in both adults and children, has become an accepted practice.[13] More than 20 recent investigations have proved its efficacy and have disproved the older theory that liver lacerations do not heal spontaneously. The successful nonoperative management of liver lacerations also reinforces the point that a decision to operate solely on the basis of a positive DPL results in unnecessary laparotomy with its attendant complications, as well as a higher incidence of infection, bile leaks and delayed hemorrhage. It must be noted that many cases of hepatic injury result in gross hemoperitoneum, peritonitis, or other obvious and immediate indications for surgical intervention without CT evaluation. Of the hemodynamically stable patients appropriately selected for CT evaluation, more than 80% will not require surgery, even for extensive parenchymal disruption and hemoperitoneum.[14-16]

Clinical stability and evidence of resolution on repeat CT scan provide a confident basis for nonoperative management in the closely monitored trauma center setting. Within 5 - 10 days, hemoperitoneum will resolve unless there is ongoing hemorrhage. Parenchymal lacerations also heal quickly, though residual intraparenchymal fluid collections (seromas and bilomas) may persist for months. There is no need to intervene surgically or to percutaneously drain such collections unless infection is suspected.

One of the most dreaded hepatic injuries is deep parenchymal laceration involving the vena cava or hepatic veins. Operative intervention is often accompanied by exsanguination unless the vena cava is clamped and bypassed during surgery in a combined abdominal/thoracic approach. Recent investigations favor angiographic evaluation and embolotherapy for arterial bleeding and close surveillance.[13,16] These lesions may not heal promptly and late complications may require surgical management. Renal traumatic lesions are also managed nonoperatively in more than 80% of cases. While suspected isolated renal trauma may be effectively evaluated by excretory urography, CT is optimal for evaluation of suspected major or combined visceral trauma. The decision for operative versus nonoperative therapy is based on the type and extent of renal parenchymal disruption, the presence or absence of extravasation of urine, the extent of perirenal hemorrhage and the presence or absence of renal vascular pedicle injury. All these criteria are better evaluated by CT than by urography.[17] Again, the combination of CT information and clinical evaluation has allowed for confident nonsurgical management in most patients, while prompting early intervention for traumatic lesions that demand surgery or catheter embolotherapy.

Blunt trauma results in pancreatic injury in only 1 - 9% of cases. While uncommon, such injuries are difficult to diagnose by any means, including DPL and CT, and may result in considerable morbidity and mortality. Pancreatic contusion is usually of little clinical significance, while pancreatic fracture with disruption of the pancreatic duct results in pancreatic necrosis, abscess and/or pseudocyst if not detected and treated. Pancreatic trauma results from direct compression of the gland against the spine, causing contusion or fracture of the pancreatic neck or body.

Reports of CT diagnosis of pancreatic injury are scarce, but indicate that diagnostic accuracy is well below that achieved for other solid visceral injuries.[18,19] Almost all reported cases have

demonstrated signs of pancreatic inflammation including peripancreatic edema or infiltrated fat and thickened perirenal (Gerota) fascia. The main pitfall of CT is the nonspecificity of these findings. Nonpancreatic trauma, such as bowel or mesenteric injury, may produce similar findings. Pancreatic contusion is difficult to distinguish from pancreatic fracture, unless an actual fracture plane can be visualized through the substance of the gland. Five such cases were reported recently by Dodds and colleagues who noted the importance of bolus dynamic infusion of contrast medium, thin (5 mm) contiguous CT sections, and use of oral contrast for optimal detection.[19] Direct cholangiography, via ERCP or direct cannulation at surgery, may be required in some cases.

The most difficult and controversial aspect of abdominal trauma CT is accurate diagnosis of bowel and mesenteric injuries.[20,21] Review of the widely divergent results suggests, however, that proper selection of patients and proper technique are important factors. CT is not accurate and is not indicated, in my opinion, in evaluation of patients with penetrating peritoneal trauma. Inclusion of such patients in some series has diminished the apparent accuracy of CT, which cannot detect reliably puncture or simple laceration of the bowel. Similarly, many centers insist on scanning patients without oral contrast medium which makes evaluation of bowel laceration or hematoma very difficult. Finally, some reports include CT cases performed after diagnostic peritoneal lavage. DPL, especially using the "open" technique, results in free intraperitoneal gas and retained lavage fluid, eliminating two of the most valuable CT criteria for recognition of bowel injury.

We have experience with over 100 cases of bowel and mesenteric injuries diagnosed by CT.[20] In the absence of other intraperitoneal injuries, CT reliably demonstrates bowel trauma and provides important information for management decisions. Unequivocal indications for surgical intervention include extraluminal extravasation of bowel contents or free air, or the combination of bowel wall or mesenteric hematoma and free peritoneal fluid. It is important to note that proximal small bowel laceration, the most common blunt injury, is usually not accompanied by CT evidence of extravasated oral contrast medium and may not have free peritoneal air, since the proximal gut is largely gasless prior to trauma. Important additional clues that should prompt close scrutiny for bowel trauma include: the presence of free peritoneal fluid (blood or lower density fluid) without an apparent visceral source; the "sentinel clot" sign, a heterogeneous high density hematoma in the bowel wall or mesentery; and clinical or radiographic indications of severe compression or deceleration trauma to the abdomen. There should be no hesitation to repeat a CT scan, with optimal bowel opacification, to perform a standard fluoroscopic contrast study of the gut, or even to surgically explore equivocal cases. While minor bowel wall and mesenteric hematomas can be managed nonoperatively, bowel perforation or mesenteric arterial disruption can be catastrophic if therapy is delayed, and it is preferable to error on the side of early surgery in difficult cases.

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