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Section I: Introduction to the Program, Faculty & Staff

Welcome to the Vascular Surgery Fellowship Program at Duke University Department of Surgery

Our goal is to prepare the Vascular Surgery Fellows (VSFs) to function as qualified practitioners of vascular surgery at the high level of competence expected of a board-certified specialist. The program provides educational resources for the development of proficiency in the diagnosis and treatment of diseases of the arterial, venous and lymphatic circulatory systems exclusive of those components intrinsic to the heart and intracranial vessels. In addition to acquiring the above clinical knowledge, VSF’s will develop interpersonal and communications skills, receive training in professional behavior and principles of ethical practice, and learn about the relationship of vascular surgery to the broader health care enterprise as exemplified by the Duke University Health System.

VSFs will be provided adequate time and sufficient facilities for study and be assured of a rotation schedule that provides an equivalent opportunity for each. Service responsibilities will not detract from educational activities. By the end of this Vascular Surgery Fellowship, the VSF will be able to:

- Demonstrate knowledge of the anatomy, physiology and pathophysiology of the vascular system, including congenital and acquired diseases
- Demonstrate the ability to surgically manage the preoperative, operative, and postoperative care of patients with arterial, venous, and lymphatic disease(s)
- Practice independently and competently as a Vascular Surgeon

The Department of Vascular Surgery Faculty & Staff

Cynthia K. Shortell, MD  Chief, Division of Vascular Surgery, Professor of Surgery
Mitchell W. Cox, MD  Program Director
Jeffrey H. Lawson, MD, PhD  Professor of Surgery
Richard L. McCann, MD  Associate Professor of Surgery
Leila Mureebe, M.D.  Associate Professor of Surgery

Associated Faculty
George “Chad” Hughes, M.D.  Associate Professor of Surgery

Honor Sanderford  Program Coordinator

This Program Manual contains specific policies and procedures in vascular surgery. These policies are in addition to the policies and procedures of the Duke GME office as outlined in the GME handbook, available at the GME website, gme.duke.edu.
Section II: Vascular Fellowship Admissions/Selection Criteria

Selection for a fellowship position in vascular surgery is based on a number of factors, which include:

1. As an Independent Program, as described by the ACGME, vascular surgery education in the independent format for the Duke Vascular Surgery Fellowship, the incoming vascular surgery fellow must:
   a. Successfully complete a general surgery residency program accredited by the ACGME.

2. Eligible for Board Certification from the ABS at the start of Fellowship – July 1, respectively.

4. Completion of all USMLE Requirements.

5. Three Letters of Recommendation from ABS Board Certified Surgeons.

6. Professional Statement of Goals and Career Objectives for the reason a candidate wants to be a Vascular Surgery Fellow at Duke.

7. Completed Application to the Program Director no later than February 15 annually.

8. Personal interview with Program Director and Faculty members upon completion of initial review for admission – all materials must be up-to-date prior to interview for consideration or the fellowship will be denied (if graduating from a residency program, you must be eligible for ABS by the time you enter our Vascular Surgery Fellowship – verification must be in the Fellowship Office no later than 10 days prior to the first day of orientation).
Section III: Specific Vascular Policies and Schedules

Duty Hours and Work Environment
All VSFs must log their duty hours each week via MedHub. At the end of each week, the program director and coordinator will review the data submitted to ensure the VSFs adhere to the following:

1. Duty hours are defined as all clinical and academic activities related to the fellowship program; i.e., patient care (both inpatient and outpatient), administrative duties relative to patient care, the provision for transfer of patient care; time spent in-house during call activities, and scheduled activities such as conferences. Duty hours do not include reading and preparation time spent away from the duty site.

2. Duty hours should be logged for time spent at conferences.

3. Duty hours must be limited to 80 hours per week, averaged over a four-week period, inclusive of all call activities.

4. Fellows are provided 1 day in 7 free from all educational and clinical responsibilities, averaged over a four-week period, inclusive of call. One day is defined as 1 continuous 24-hour period free from all clinical, educational, and administrative duties. Each VSF is must take their arranged day off.

5. Adequate time for rest and personal activities must be provided. This should consist of a 10-hour time period provided between all daily duty periods.

On-Call Activities
The objective of on-call activities is to provide fellows with continuity of patient care experiences. VSFs do not take in-house call, just home-call (pager call), which is defined as call taken from outside the assigned institution.

1. The frequency of at home-call is not subject to the every third night limitation. However, home-call is not so frequent as to preclude rest and reasonable personal time for each trainee. Trainees taking home-call are provided with 1 day in 7 completely free from all educational and clinical responsibilities, averaged over a 4-week period.

2. When trainees are called into the hospital from home, the hours trainees spend in-house are counted toward the 80-hour limit.

3. The program director and the faculty monitor the demands of home-call and make scheduling adjustments as necessary to mitigate excessive service demands and/or fatigue.

Any VSF who fails to comply with the ACGME rules place the program at risk. Failure to adhere to program requirements may include administrative leave or a corrective action plan. If
a VSF fails to adhere to the corrective action plan, as a last resort, termination from the program will be considered.

**Supervisory Lines of Responsibility**
The Duke University Hospital Fellowship in Vascular Surgery is designed to provide consistent and outstanding didactic, clinical and technical education to the Vascular Surgery Fellow (VSF). Attaining these goals enables the Fellow to effectively provide superior health care to patients.

In addition, the Duke Fellowship in Vascular Surgery prepares the Fellow to independently manage the preoperative, operative, and postoperative care of patients with arterial, venous and lymphatic disease(s).

All VSFs are required to have a valid North Carolina State Medical License and must be registered with the North Carolina Medical Board.

The VSFs and General Surgery Chief Residents are never assigned to the same service nor do they have the same responsibility for the patients on his/her service.

The Vascular Surgery Fellow is responsible for the preoperative management of the patients with the help of the junior residents assigned to the Vascular Service and advanced practice providers under the supervision of the attending surgeon. The VSF will have first seen the patient in the surgical outpatient experience/clinic, Emergency Room, attending surgeons’ office, or upon admission to the hospital. The VSF is assigned to those operations where the VSF can assume the most senior role commensurate with experience and abilities. Preoperatively, a dialogue is established between the VSF involved and the responsible faculty member to determine the specifics of therapy and the options for management. No patient can be taken to the operating room for any surgical procedure without the faculty member present in the operating room. Anesthesia cannot be induced until the faculty member has related to the patient. The faculty member must remain physically within the operating room area throughout the entire procedure until the patient is transferred to the post-operative care unit. All attestation sheets are signed by the faculty member of record, as are the operative notes. Under the supervision of the attending surgeon, the VSF is responsible for the postoperative in-hospital management of the patient and when possible will see the patient during postoperative visits.

The faculty members of Vascular Surgery share the responsibility of “on call.” There is one faculty member responsible each day for consultation and emergencies at night and on the weekends for the three training sites. Call will be split among the VSF’s and senior residents on service such that all sites with a rotating fellow will be covered. Faculty presence in the hospital can be requested by the VSF at any time, and faculty attendance is mandatory for any operative procedure.

Vascular Surgery Fellow and Attending On-Call Schedules are made monthly, are available at all times, and are provided to the following:
• Page office
• Answering service
• Emergency room
• Admitting office
• All Vascular attending(s)
• All Vascular Fellows
• All Interventional Radiology attendings and residents
• Nurses on Vascular service
• Vascular Clinic
• General Surgery Residency Office

This policy is consistent with the Duke University Hospital GME Supervision Policy.

**Protocol for Common Circumstances Requiring Faculty Involvement**
The Duke Vascular Surgery Fellowship expects Fellows to immediately contact the Attending on call for:

1. Any concern, uncertainty or ambiguity at any time, the fellow may perceive
2. Patient death
3. If an RRT or code is called on a patient
4. Patient clinically unstable or status changed
5. Major change in plan of care
6. Communication of important results/care plan/prognosis to patient
7. If a patient is transferred to a more acute level of care, eg. From the floor to the ICU
8. Consideration of urgent/emergent operation
9. Consideration of taking the patient back to the OR urgently
10. Decision to make a patient DNR
11. Decision to change a patient’s resuscitation status
12. A request from an attending on another service (consult)
13. Concerns, Conflicts expressed by another member of the health care team, a patient, or family member
14. Requests for inter-service transfer

**Fellow Appointment, Reappointment, Promotion & Dismissal**
All Employment Agreement letters are for one (1) year. Contracts are prepared and signed electronically by the Program Director, Fellow and Designated Institutional Officer (DIO).

Completion of the program is based on several factors. Those factors include:

1. Outcome assessment through various formats
2. Operative case experiences in both open and endovascular surgical arenas
3. Accurate and timely logging of all surgical case operative experiences via the ACGME case log entry system and ensuring that the minimum requirements are met before the end of the fellowship.
4. Formal and informal evaluations from Program Director and faculty members
5. Commitment and interest in scholarly activities
6. Commitment and interest in teaching of junior residents, medical students and other health care providers

In addition, at any time during the Fellow’s training, a written or verbal report to the Program Director of inappropriate behavior or actions by the Fellow is received; it will be discussed with him/her. After investigation and evaluation of the allegations appropriate actions may be taken which may include, but are not limited to: advice, warnings, counseling, psychological support, change in rotations, or recommendation to the Institution that the Fellow be given a leave of absence or be dismissed using the appropriate due process policies of Duke University Hospital.

The Program's Clinical Competency Committee meets bi-annually to review evaluations and other assessment tools. The committee will make recommendations to the program director regarding promotion and graduation.

Reappointment and/or graduation for individual Fellows depends upon their ongoing clinical skills evaluation by each attending surgeon, evidence of ethical behavior, and professional characteristics of an individual capable of independent practice in vascular surgery.

If it is decided that the Fellow should take a leave of absence or be dismissed, the Fellow, the Chairman of the Department and the DIO will be notified in writing. Final decisions are subject to Duke University GME House Staff policies and shall always be in writing.

**Vacation, Leave of Absence and Academic Conference Policies**

VSFs will be allotted two (2) one week paid vacations, which must be scheduled at least one month in advance by sending an email request to the Program Director. Failure to request the time off may result in the request being disallowed for that time period. In addition, VSFs are encouraged to attend one (1) meeting per academic year. All travel must occur in the U.S. VSFs are encouraged to be academically productive and will be granted additional meeting time for approved meetings where they have had an abstract accepted. VSFs should check with the Program Director prior to submitting abstracts to determine if the meeting is an approved meeting. Failure to do so may result in the travel to said meeting being disallowed.

VSFs obligation to participate in their educational experience is identical regardless of the clinical rotation they are assigned to. Vacation time should be requested must be scheduled at least one month in advance by sending an email request to the Program Director. Any time away or "tardiness" needed from the typical work day must be approved by the Program Director and a back-up must be established with another fellow. Emergencies are a different story of course; fellows with emergencies should contact the Program Director, the Associate Program Director or the Program Coordinator.

FMLA / Disability Leave: The Family and Medical Leave Act (FMLA) entitles a covered employee to take up to 84 days of unpaid leave in a 12-month period for the birth or adoption of a child, or the "serious health condition" of the employee or the employee's child, spouse, or
parent. If at all possible, the fellow must make the request for FMLA and all associated paperwork prior to the precipitating event. The fellow must inform the Program Director and the Program Coordinator at their earliest awareness of such a need. For FMLA approval, the VSF should seek consultation with the EOHS, or ask his/her treating clinician to send documentation of an FMLA qualifying condition and recommendation for time away to the EOHS. The EOHS will then communicate the approval of the leave to the VSFs program director. FMLA can be taken in a full block or in smaller increments as determined by the clinician who provides care in conjunction with the EOHS or his/her designee.

Absences in excess of the Vascular Surgery Board of the American Board of Surgery (VSBABS) Requirements for Vascular Surgery must be made up by extension of the fellowship training. The VSF must have obtained no fewer than 48 weeks of full-time surgical experience in each fellowship year. For documented medical problems or maternity leave, the VSB-ABS will accept 46 weeks of surgical training in one of the last two (2) years of all approved training pathways.

VSFs requesting any type of leave are required to notify the Program Director as early as possible to arrange for adequate coverage during your absence.

Moonlighting
Because graduate medical education is a full-time endeavor, moonlighting of any type is not permitted.

Fellow Fatigue and Stress
Recognizing that fellows can suffer from fatigue and stress, the Vascular Surgery Fellowship Program does the following to minimize fatigue and stress:

- Adheres to specialty specific duty hour requirements,
- Minimize prolonged work (> 24 hours of clinical duties),
- Protects periods designed to address sleep debt (i.e. the minimum of at least 24 hours off each week free from all clinical responsibilities)
- Reduces non–essential tasks and enhance learning during clinical time,
- Reduces non–essential interruptions (i.e. added ancillary services, triage of phone calls by charge nurse etc)
- Assists fellows to identify co–existent medical issues which impair their sleep (i.e. undiagnosed sleep disorder, depression, stress),
- Educates regarding awareness and management of fatigue
- Critically appraises the best way to implement shift work.
- Provide napping resources
- Provide free car service from hospital to home and back to hospital.

The program director directly asks about issues pertaining to getting adequate sleep, fellow safety such as concerning post–call driving, and fellow concerns about the balance between professionalism and work hour restrictions.
Duke's free and confidential Personal Assistance Service is available free of charge to the fellows to assist them in dealing with the stressors in their life, http://hr.duke.edu/pas/

**Hand-Off Procedure**
A standardized approach to the "hand-off" of care at Duke University Hospital provides an opportunity to ask and respond to questions. Caregivers involved in the hand-off process include, but are not limited to; physicians, nurses, advanced practice providers, therapists, technicians and transporters.

Key elements of patient information are included in the hand-off process as determined by the service or team of caregivers. Patient information related to current condition and present treatment patient information will include at a minimum:

- Patient name
- MR#
- Age and comorbidities
- Diagnosis and operative procedure, if appropriate
- Allergies
- Isolation Status
- Potential changes in condition
- Care plan for patient
- What to watch for or monitor during the next interval of care

Hand-off communication in the Division of Vascular Surgery is every Friday morning at Attending Rounds at 8:00 a.m. in the Endosurgery Conference Room

**Corrective Action and Hearing Procedures**

1. Scope; Other Applicable Procedures. These procedures provide the sole and exclusive process (including all notices, hearings, appeals or other review, if any) for the suspension of, imposition of corrective action against, or nonrenewal of an Associate Member of the Medical Staff ("Associate") of Duke University Hospital with respect to such Associate’s activities or status at (i) Duke University Hospital, Duke Raleigh Hospital or Durham Regional Hospital (each a “Hospital” and collectively the “Hospitals”) or (ii) any other entity with which the Duke University Hospital Graduate Medical Education Program ("Program") has an Affiliation Agreement and or Training Letter of Agreement (each such other entity an “Affiliate” and collectively the “Affiliates”). The Associate expressly acknowledges and agrees to these procedures by applying for and/or entering into the Program or applying for and maintaining Associate Staff membership as described above. Nothing herein should be read as precluding either (i) preliminary or informal discussions with Associates regarding concerns otherwise addressed by these procedures or (ii) action against Associates under other applicable Duke University, Duke University Health System, Hospital or other rules, regulations, policies or procedures; provided, that such discussions and/or actions should be undertaken following consultation with the Program Director and the Director of Graduate Medical Education. Notwithstanding anything
herein to the contrary, however, no Associate is or shall be entitled to avail himself or herself of any corrective action, hearing or appeal procedures set forth in Hospital or Affiliate medical staff bylaws (“Bylaws”).

2. Suspension.

2.1. Summary Suspension. If immediate action is deemed necessary to preserve the interests of patient care or safety, the safety of other individuals at any Hospital or Affiliate or the orderly operation of any Hospital or Affiliate, then the Program Director or the Director of Graduate Medical Education may immediately remove an Associate from all clinical and other duties at all Hospitals and Affiliates pending a final decision (including any hearings, appeals or other review) on a request for corrective action as described below. Such removal shall be hereinafter referred to as “summary suspension”. A request for corrective action relating to the basis for summary suspension must be made in accordance with Section 3.1 below no later than three (3) days following the imposition of a summary suspension.

2.2. Automatic Suspension. An Associate’s staff membership(s) and privileges (if any) may be automatically suspended and the Program Director or the Director of Graduate Medical Education may immediately remove such Associate from all clinical and other duties at all Hospitals and Affiliates in the event of one or more of the following deficiencies, until such time as the Associate cures the deficiency or deficiencies in the time and manner required by, and to the satisfaction of, the affected Hospital or Affiliate, the Program Director and the Office of Graduate Medical Education:

- Failure to timely complete and sign all medical records or other documents requiring the Associate’s signature as set forth in Bylaws or Medical Staff Rules and Regulations, as applicable.
- Failure to maintain on file at the Graduate Medical Education Office a current and active North Carolina medical license and federal and state prescription authority, free from suspension or other limitation as required by the Hospital or Affiliate to fulfill duties as a trainee.
- Engaging in unapproved external moonlighting.
- Failure of the Associate to comply with any request by any Hospital or Affiliate, including to cooperate with peer review or quality assurance activities of any Hospital or Affiliate, or its Medical Staff;
- Failure to comply with any Hospital’s or Affiliate’s request for a physical or psychological examination.
- Failure to comply with any Hospital, Affiliate, Department or Program-mandated educational, safety, legal, health instructional programs or requirements including, but not limited to, tuberculosis screening, OSHA training, hospital safety, HIPAA education, Basis Life Support (BLS), Advanced Cardiac Life Support (ACLS), Pediatric Advanced Life Support (PALS), Neonatal Advanced Life Support (NALS), and Advanced Trauma Life Support (ATLS) (when required).
- Three (3) or more unexcused absences from scheduled training.
2.3. **Notice of Suspension.** The Program Director shall notify the Associate of the imposition or lifting of a summary or automatic suspension within three (3) days thereafter. Copies of such notice shall be provided to the Director of Graduate Medical Education, and the Department Chair. Suspensions lifted after the Associate cures the deficiencies noted shall not be reported to the NC Medical Board, state regional or national databank, nor to any other third party, except as otherwise required by law or contract.

2.4. The Associate shall have no right to request a hearing on, or appeal or otherwise seek review of, a summary or automatic suspension; provided, however, nothing in these procedures precludes an Associate from subsequently meeting with the Program Director or Director of Graduate Medical Education to discuss the circumstances of their suspension and, in the case of automatic suspension, the steps Associate must take to lift it.

3. **Corrective Action.**

3.1. **Who May Request Corrective Action.** A request for corrective action against an Associate may be submitted by any member of the Active Medical Staff or the leadership of the Office of Graduate Medical Education. The request shall be made by notice submitted to the appropriate Program Director, and shall be supported by reference to the specific activities or conduct which constitute the grounds for the request. The Program Director shall promptly forward copies of such notice to the Director of Graduate Medical Education and the Department Chair.

3.2. **Initial Investigation.** Each Department will promptly investigate and evaluate any request for corrective action. As part of this investigation, the Program Director shall, within three (3) days of receiving a notice pursuant to Section 3.1, notify the Associate of the request and the Associate’s opportunity to meet with the Program Director to discuss it. If such a meeting takes place, it shall be informal, and shall not constitute a hearing. The Program Director shall make a record of the meeting consistent with usual Program practices.

3.3. **Decision not to Impose Corrective Action.** Upon completion of any investigation described in Section 3.2 above, the Program Director may decide that the request does not warrant the imposition of any corrective action. In that event, the Program Director shall notify in writing the Associate with a copy thereof to the Director of Graduate Medical Education and the Department Chair of his or her decision within fourteen (14) days of the Program Director’s receipt of notice pursuant to Section 3.1 above. Such decision shall be a final decision.

3.4. **Decision to Impose Corrective Action.** Upon completion of any investigation described in Section 3.2 above, the Program Director may determine that routine or adverse corrective action should be imposed on the Associate. Such decision shall be provided by notice specifying the type of corrective action recommended together with a report specifying the supporting grounds for such action and, where applicable, the necessary remediation steps for the Associate to accomplish in order for such corrective action(s) to cease, in the time and manner further described in Sections 3.5.1 or 3.6.1 below (as applicable).
3.5. **Routine Corrective Action.** Any act or omission by an Associate deemed by the Program Director in his or her discretion as warranting formal remedial measures may constitute grounds for routine corrective action, including, without limitation, continued failure by an Associate to meet Program standards and requirements despite prior attempts to informally address such failure with the Associate. Routine corrective action may include, without limitation, imposition of one or more of the following on an Associate:

(i) additional self-study, repetition of learning assignments or like educational measures (other than non-promotion as defined in Section 10 below);
(ii) required period(s) of individual mentoring and/or increased supervision; or
(iii) structured counseling. Once final, routine corrective action shall be implemented on a written schedule developed by the Program Director and provided to the Associate.

3.5.1. **Notification of Routine Corrective Action.** Where the Program Director determines that routine corrective action is appropriate, the Program Director shall within **fourteen (14) days** of the receipt of notice of investigation request pursuant to Section 3.1, provide the notice and report described in Section 3.4 to the Associate, with a copy to the Director of Graduate Medical Education and the Department Chair. The notice shall also notify the Associate that he or she may request a review of the Program Director’s decision by notifying the Director of Graduate Medical Education of his or her request within **seven (7) days** of delivery of the Program Director’s notice and report. If the Associate does not so request a review, the Program Director’s decision as to routine corrective action shall become a final decision, and the Associate shall have no right to request a hearing on, or to appeal or otherwise seek review of, such decision.

3.5.2. **Routine Corrective Action Review Procedures.** Upon receipt of an Associate’s notice requesting review of a routine corrective action as set forth in Section 3.5.1 above, the Director of Graduate Medical Education shall provide the Program Director and Department Chair with Associate’s request for review. The Department Chair reviews the recommendation and report of the Program Director pursuant to Section 3.4. Such review may include a meeting with the Associate at the request of the Department Chair. The Department Chair shall send a written decision, including a discussion of the rationale for the decision, within **fourteen (14) days** of Associate’s request for review pursuant to Section 3.5.1 above. The Department Chair may affirm the Program Director’s decision, impose an alternative form of routine corrective action no more severe than that recommended by the Program Director, or determine that routine corrective action is not appropriate. If the Department Chair determines that routine corrective action is not appropriate, the appropriate form of responsive action (other than routine corrective action), if any, shall be left to the discretion of the Program Director in consultation with the Department Chair. The Department Chair’s decision shall be shared in writing with the Associate with a copy thereof to the Program Director and Director of Graduate Medical Education. Such decision shall be a final decision, and the Associate shall have no right to further hearings, appeals or other review of such decision; provided, however that nothing in these procedures precludes an Associate from subsequently meeting
with Director of Graduate Medical Education to discuss the circumstances of and process leading up to the imposition of routine corrective action.

3.6. **Adverse Corrective Action.** Corrective action is considered “adverse corrective action” if the Program Director determines, pursuant to Section 3.4, to impose any of the following on an Associate:

1) suspension or revocation of an Associate’s Staff membership or privileges, except in the case of summary or automatic suspension described above,
2) dismissal from the Graduate Medical Education Program, except in connection with a non-renewal described below, or
3) non-promotion (as defined in Section 10). The following are grounds for adverse corrective action:

- Performance, activities or professional conduct of the Associate which are considered to be lower than the standards or aims of the Program or the medical staff of any Hospital (“Medical Staff”), or which are disruptive to the objectives and efficient operations of the Program or any Hospital or Affiliate;
- Failure of the Associate to comply with the Bylaws or Rules and Regulations applicable to the Medical Staff;
- Failure of the Associate to comply with any Hospital, Departmental or Program regulations;
- Failure of the Associate to comply with any request by any Hospital or Affiliate, including to cooperate with peer review or quality assurance activities of any Hospital or Affiliate, or its Medical Staff;
- The Associate’s being charged with or convicted of, or entering of a plea of nolo contendere to, a crime related to the provision of healthcare, or any felony, or any misdemeanor involving intentional violence or assault, theft, or other acts of moral turpitude;
- The (i) commencement of any investigation of Associate with respect to, or the imposition of sanctions on or the exclusion, debarment, suspension or declaration of ineligibility of the Associate from or with respect to, any public healthcare program or (ii) listing of Associate on any General Services Administration or Office of Inspector General excluded parties lists, regardless of any right to appeal; • Any misrepresentation by the Associate in 1) an application for acceptance as a graduate medical trainee in any Hospital Graduate Medical Education Program; or 2) any other documentation submitted to obtain or maintain a position as a graduate medical trainee in any Hospital’s Graduate Medical Education Program. • Any grounds listed in Section 10, Non-Promotion. • Other grounds as set forth in Section 5, Confidentiality and Immunity.

3.6.1. **Notification of Adverse Corrective Action.** Where the Program Director determines that adverse corrective action is appropriate, the Program Director shall within **fourteen (14) days** of the receipt of notice of investigation request pursuant to Section 3.1, provide the notice and report described in Section 3.4 to the Associate with a copy to the Director of Graduate Medical Education and the
Department Chair. The notice shall also notify the Associate that he or she may request a review of the Program Director's decision by notifying the Director of Graduate Medical Education of his or her request within seven (7) days of delivery of the Program Director's notice and report. If the Associate does not so request a review, the Program Director’s decision as to adverse corrective action shall become a final decision, and the Associate shall have no right to request a hearing on, or to appeal or otherwise seek review of, such decision.

3.6.2. Adverse Corrective Action Hearing Procedures.

3.6.2.1. Hearing Panel. Upon receipt of the Associate’s written request for hearing pursuant to Section 3.6.1 above, the Director of Graduate Medical Education shall appoint a subcommittee of the Institutional Committee on Graduate Medical Education as the Hearing Panel. The Hearing Panel shall be composed of five individuals. Four members of the panel shall be members of the Active Medical Staff, at least one of whom is from the Department with which the Associate is affiliated. The fifth member of the panel shall be an Associate Staff member. The Director of Graduate Medical Education shall designate one of the five individuals to serve as Chair of the Hearing Panel. The Hearing Panel shall not include any individual previously involved in any way with the action or actions which resulted in the request for corrective action, or in the previous consideration of the request for corrective action. As necessary, the Director of Graduate Medical Education may appoint members of the Medical Staff who are not members of the Institutional Committee on Graduate Medical Education to serve on the Hearing Panel.

3.6.2.2. Notification and Scheduling. The Director of Graduate Medical Education shall promptly notify the Associate of the time, place and date for the hearing, which shall be held not less than fourteen (14) or more than twenty-eight (28) days from the date of the delivery of the Associate’s request for a hearing pursuant to Section 3.6.1. above.

3.6.2.3. Procedures. The Hearing Panel shall be entitled to call witnesses and to examine them. The Associate shall have the right to be advised (but not represented) by counsel, call witnesses, present relevant written information, cross examine any witnesses testifying at the request of the Hearing Panel, and submit a written statement at the close of the hearing. The Program Director shall appear and present his or her report to the Hearing Panel. The hearing need not be conducted strictly according to the rules of law relating to the examination of witnesses or the presentation of evidence. The Chair of the Hearing Panel shall make all necessary rulings regarding hearing procedure, including any necessary ruling on an Associate’s request that a Hearing Panel member be replaced on the grounds of that member’s bias against the Associate. The Chair shall ensure that an accurate record of the hearing is kept by court reporter, electronic recording, verbatim transcription or by the taking of adequate minutes.

3.6.2.4. Hearing Panel Decision. Following the conclusion of the hearing, the Hearing Panel shall promptly conduct deliberations. The Hearing Panel shall send its written decision, including a discussion of the rationale for the decision, within fourteen (14) days of the conclusion of the hearing, to the Associate with a copy
thereof to the Program Director, Director of Graduate Medical Education and the Department Chair. The Hearing Panel may adopt the Program Director’s recommendation for adverse corrective action, impose an alternative form of adverse corrective action no more severe than that recommended by the Program Director, or determine that adverse corrective action is not appropriate. If the Hearing Panel determines that adverse corrective action is not appropriate, the appropriate form of responsive action (other than adverse corrective action), if any, shall be left to the discretion of the Program Director in consultation with the Department Chair.

3.6.3. Appellate Review of Adverse Corrective Action.

3.6.3.1. Written Submissions. If the Hearing Panel’s decision constitutes “adverse corrective action” as defined in 3.6. above, the Associate may contest the decision by submitting a notice of contest together with a written submission detailing Associate’s objections to such decision. The Associate’s notice and submission must be received by the Director of Graduate Medical Education within seven (7) days of the delivery of the Hearing Panel’s decision to the Associate pursuant to Section 3.6.2.4 above. Upon receipt of the Associate’s notice and submission, the Director of Graduate Medical Education shall provide a copy thereof to the Program Director. The Program Director shall have seven (7) days from the date of receipt of such copy to submit a statement in response to the Associate’s notice and submission to the Director of Graduate Medical Education. The Director of Graduate Medical Education shall notify the Associate of the Program Director’s written submission by providing a copy thereof.

3.6.3.2. Appellate Decision. Within fourteen (14) days of the deadline for receipt of the Program Director’s responsive submission, the Director of Graduate Medical Education shall render his or her decision in writing and send it to the Associate and provide a copy thereof to the Program Director and the Department Chair. The decision shall be based on the record before the Hearing Panel and any written submissions made under Section 3.6.3.1 above. The Director of Graduate Medical Education may affirm the Hearing Panel’s decision, impose an alternative form of adverse corrective action no more severe than that recommended by the Program Director, or determine that adverse corrective action is not appropriate. If the Director of Graduate Medical Education determines that adverse corrective action is not appropriate, the appropriate form of responsive action (other than adverse corrective action), if any, shall be left to the discretion of the Program Director in consultation with the Department Chair. The Director of Graduate Medical Education’s decision shall be a final decision, and the Associate shall have no right to further hearings on, or appeals or other review of, such decision.

4. Non-Renewal. The decision not to renew the yearly contract of an Associate for graduate medical training upon expiration of that contract (“non-renewal”) is not a suspension or corrective action, and the procedures pertinent to those matters do not apply to non-renewal. However, in order to provide a structure for the review
of non-renewal, the following procedures have been implemented and constitute
the sole and exclusive procedures for hearing, appeal or other review thereof.

4.1. Notice of Non-Renewal. Notice of non-renewal shall be given by the Program
Director no later than four (4) months prior to the end of the Associate’s then-
current contract year (or, if any reason or reasons for non-renewal occur within
the four (4) month period prior to the end of the contract year, as soon as
circumstances reasonably allow but in no event after the end of such contract
year). The notice shall contain a statement advising that, except in the case of
nonrenewal due to institutional factors as set forth below, the Associate may by
notice to the Director of Graduate Medical Education request a hearing on their
non-renewal within seven (7) days of the delivery of notice of non-renewal from
the Program Director. If the Associate does not so request a hearing, the non-
renewal shall become a final decision, and the Associate shall have no further
hearing, appeal or other review of such decision. Notwithstanding anything
herein to the contrary, in no event shall an Associate be entitled to any hearing,
appeal or other review when nonrenewal is due to Hospital, Affiliate, Program or
Departmental closures, reductions or discontinuances or like institutional factors.

4.2. Non-Renewal Hearing Procedures.
4.2.1. Hearing Panel. Upon receipt of the Associate’s written request for hearing as set
forth in Section 4.1 above, the Director of Graduate Medical Education shall
appoint three (3) Clinical Department Chairs to hear the matter. These individuals
will constitute the Hearing Panel and shall designate one (1) of the three (3) to
serve as Chair of the Hearing Panel. None of the Hearing Panel members shall
be from the Department or Departments in which the Associate is appointed.

4.2.2. Notification and Scheduling. The Director of Graduate Medical Education shall
promptly notify the Associate of the time, place and date for the hearing which
shall be held not less than fourteen (14) or more than twenty-eight (28) days
from the date of the delivery of the Associate’s request for a hearing.

4.2.3. Procedures. The Hearing Panel shall be entitled to call witnesses and to examine
them. The Associate shall have the right to be advised (but not represented) by
counsel, call witnesses, present relevant written information, cross examine any
witness testifying at the request of the Hearing Panel, and submit a written
statement at the close of the hearing. The Director of Graduate Medical
Education and the elected Associate member of the Executive Committee of the
Medical Staff shall be in attendance at the Hearing as ad hoc, nonvoting
attendees, and the Program Director for the Associate shall present any relevant
information to the Hearing Panel. The hearing need not be conducted strictly
according to the rules of law relating to the examination of witnesses or the
presentation of evidence. The Chair of the Hearing Panel shall make all
necessary rulings regarding hearing procedure, including any necessary ruling
on an Associate’s request that a Hearing Panel member be replaced on the
grounds of that member’s bias against the Associate. The Chair of the Hearing
Panel shall ensure that an accurate record of the hearing is kept by court
reporter, electronic recording, verbatim transcription or the taking of adequate
minutes.
4.2.4. **Hearing Panel Decision.** Following the conclusion of the hearing, the Hearing Panel shall promptly conduct deliberations. The Hearing Panel shall send its written decision, including a discussion of the rationale for the decision, within **fourteen (14) days** of the conclusion of the hearing to the Associate with a copy to the Program Director, Director of Graduate Medical Education and the Department Chair. The Hearing Panel may affirm the Program Director’s decision, impose an alternative form of responsive action no more severe than that recommended by the Program Director, or determine that non-renewal is not appropriate and refer the matter back to the Program Director. If the Hearing Panel determines that non-renewal is not appropriate, the appropriate form of responsive action (other than non-renewal), if any, shall be left to the discretion of the Program Director in consultation with the Department Chair. The written decision of the Hearing Panel shall be a final decision, and the Associate shall have no right to further hearings, appeals or other review of such decision; provided, however, that nothing in these procedures precludes an Associate from subsequently meeting with the Director of Graduate Medical Education to discuss the circumstances of and process leading up to such decision.

5. Confidentiality and Immunity.

5.1. Definitions.

5.1.1. "Information" means records of proceedings, minutes, interviews, reports, forms, memoranda, statements, investigations, examinations, hearings, meetings, recommendations, findings, evaluations, opinions, conclusions, actions, data, and other disclosures or communications, whether in written or oral form. Information may relate to an Associate’s professional licensure or certification, education, training, clinical ability, judgment, utilization practices, character, physical or mental health, emotional stability, professional ethics, or any other matters that might directly or indirectly affect the quality, efficiency, or appropriateness of health care services provided in a Hospital or Affiliate, including confidential patient communications, or records.

5.1.2. "Representative" means the Program, its leadership and all of its or their appointed representatives, designees and panelists (specifically including all Duke University personnel involved in any way in the administration of the Program); personnel at any Hospital involved in any way in the administration of the Program at the Hospital; all consultants and independent contractors to the Program; the University Counsel’s Office attorneys and their assistants or designees; and any authorized representative of any of the foregoing.

5.1.3. "Third Parties" means all individuals or entities other than Duke University, the Program, any Hospital, any Representative or the Associate, including without limitation government agencies, organizations, associations, partnerships and corporations, whether hospitals, health care facilities, or otherwise.

5.2. **Associate Information.** Each Associate authorizes, by way of contract, Representatives to solicit, provide, and act upon information bearing on his or her professional ability, utilization practices, and other qualifications, and authorizes all Third Parties to provide Information to the Program or its Representatives, including allowing inspection and copying of any records in the possession or
control of Third Parties. Each Associate shall, upon request of the Program and in such form as requested by Program, execute general and specific authorizations and releases from liability reflecting the provisions of this Section 5; provided, however, that execution of such documents is not a prerequisite to the effectiveness of this Section 5. Failure to execute such documents on any initial application to the Program shall result in the application being deemed incomplete and it shall not be considered. There shall be no entitlement to procedural rights of review as a result of non-consideration of an application. Failure to execute such documents at any time shall also constitute grounds for suspension, corrective action or non-renewal hereunder.

5.3. Confidentiality. All Information submitted, collected or prepared in connection with activities described in these procedures shall be privileged and confidential. Maintenance of and access to such Information shall be in accord with all applicable institutional and legal requirements to maintain all applicable privileges and confidentiality. Such Information shall not be disclosed to any Third Party except
(i) by the Program Director or the Director of Graduate Medical Education or a Hearing Panel appointed hereunder;
(ii) to legal counsel or others as necessary to carry out their functions or these procedures or
(iii) as authorized by Associate or
(iv) as authorized or required by law.
In no event shall any access to or disclosure of such Information constitute a waiver of applicable privileges or confidentiality requirements. Associates who breach confidentiality as set forth in this Section 5.3 may be subject to corrective action as described herein.

5.4. Immunity. Representatives participating in, and Representatives and Third Parties providing Information in connection with, activities described in these procedures shall be entitled to the same immunities with respect to Associates as are “Representatives” and “Third Parties” with respect to “Practitioners” pursuant to Section 12.5 of the Duke Hospital Medical Staff Bylaws.

5.5. Reporting Requirements. Nothing herein shall affect or interfere with any right or obligation of Duke University, the Program, any Hospital or the Associate to make any report pursuant to state or federal law.

5.6. Cumulative Effect. The provisions in these procedures and in any related forms pertaining to authorization, confidentiality of information, and immunities from liability are in addition to other protections provided by relevant state and federal law, not in limitation. A finding by a court of law or administrative agency that all or any portion of any such provision is not enforceable shall not affect the legality or enforceability of the remainder of the provision or any other provision.

6. Designees. Actions required or authorized to be taken by the Program Director or the Director of Graduate Medical Education under these procedures may be performed by that individual’s designee in the event that individual is unavailable, provided the designee has had no involvement in the events giving rise to the need for such required or authorized action. The Director of Graduate Medical
Education shall appoint a designee to fulfill the duties of the Department Chair under Section 3.5.2 where the Department Chair is also the Program Director.

7. **Procedural Defects or Irregularities.** The failure of any individual or panel described herein to observe required procedures for requesting, or reaching or imposing a final decision (including any hearing, appeal or other review) with regard to, a suspension, corrective action or non-renewal where any such failure is harmless error shall neither affect the validity of such final decision nor constitute a sufficient basis for rehearing or reconsideration thereof.

8. **Time.** For purposes of these procedures, days are defined as Sunday through Saturday. In computing any period of time prescribed or allowed by these procedures, the day of the act, omission or event after which time begins to run shall not be included. The last day of the period so computed shall be included.

9. **Form of Notice.** Any notice required under these procedures must be in writing and
   (i) given in person using means providing for verification of delivery,
   (ii) sent by overnight express delivery service or mailed first class mail, postage prepaid and return receipt requested, to the office of Graduate Medical Education (if to Program personnel) or to the last-known address on file with such Office (if to Associate); or
   (iii) sent to the recipient’s e-mail address listed in the Duke University Hospital e-mail system. The Director of Graduate Medical Education may include other Program, Hospital or Affiliate personnel or any Representative having a need to know in any matter described herein, as deemed applicable and appropriate in his or her sole discretion.

10. **Non-Promotion.** Promotion to the next graduate medical education training level (e.g., PGY-1 to PGY-2) or to program completion is based on the achievement of program-specific competence and performance parameters, including specific cognitive, clinical, technical skills, and professional and ethical conduct, as measured in regular evaluations. Non-promotion to the next level or program completion means that the Associate fails to perform at an acceptable level in the period of current appointment or cannot reasonably function satisfactorily at the next level and is not advanced to a higher rank or title or to program completion. Remediation steps, as noted by the Program Director, must be accomplished prior to the Associate’s advancement to the next level or program completion.
Section IV: Record Keeping

Attendance Records
VSFs are required to attend and sign in legibly at all Grand Rounds, Conferences and Journal Clubs. Attendance of 75% of Grand Rounds, Conferences and Journal Clubs will be considered as part of the promotion/graduation process.

Operative Case Logs
Annual Operative Case Logs must be submitted online via the ACGME. The Program Coordinator gathers operative case logs from ACGME and sends to the Program Director and Associate Program Director. The Program Director will carefully monitor this data on a quarterly basis. If a VSF falls behind or fails to meet the minimum RRC requirements, the Program Director will identify appropriate cases to perform in order to bring the number of surgical cases in-line.

Failure to maintain the operative log on a monthly basis may result in suspension of clinical privileges until the log is updated.

Duty Hour
All VSFs must maintain a log of their daily duty hours. Each week, the VSF will enter their duty hour data via MedHub. At the end of each week, the program director and coordinator will review the data submitted. A monthly calculation will be tabulated and tracked to ensure ACGME compliance as follows:

1 Duty hours are defined as all clinical and academic activities related to the fellowship program; i.e., patient care (both inpatient and outpatient), administrative duties relative to patient care, the provision for transfer of patient care, time spent in-house during call activities, and scheduled activities such as conferences. Duty hours do not include reading and preparation time spent away from the duty site.

2 Duty hours should be logged for time spent at conferences.

3 Duty hours must be limited to 80 hours per week, averaged over a four-week period, inclusive of all in-house call activities.

4 Fellows must be provided with 1 day in 7 free from all educational and clinical responsibilities, averaged over a four-week period, inclusive of call. One day is defined as 1 continuous 24-hour period free from all clinical, educational, and administrative duties. Each VSF is must take their arranged day off.

5 Adequate time for rest and personal activities must be provided. This should consist of a 10-hour time period provided between all daily duty periods and after in-house call.
Any VSF who fails to comply with the ACGME rules place the program at risk. Failure to adhere to program requirements may include administrative leave or a corrective action plan. If a VSF fails to adhere to the corrective action plan, as a last resort, termination from the program will be considered.

Each VSF will be assessed each workday by supervising faculty regarding their previous night work hours, alertness and well-being after night of taking call at home. If the VSF shows signs of lack of alertness or well-being, the VSF will be sent home early the next day. If a fellow has failed to obtain at least four (4) hours of sleep, the VSF will be required to leave no later than 12:00 p.m. (noon) that day. VSFs and faculty are required to review a one-hour video annually on alertness and well-being and duty hour assessment.

**Safety Training and HIPAA**
Safety Training includes ACLS, BCLS, HIPAA, OSHA and others. These must be updated online annually, and the GME Office will send reminders via email. You are required to maintain compliance at all times. Failure to comply includes Hospital mandated administrative leave and temporary loss of training privileges.

**Evaluations**
As a Vascular Surgery Fellow, you will be required to participate in the Division’s evaluation requirements.

Rotation Evaluation – to provide the Program with continual feedback on the value/educational merit of each rotation VSFs are to complete evaluations on MedHub.

Faculty Evaluation – after each rotation VSFs complete evaluations via MedHub on the faculty with valuable feedback concerning:
- Teaching abilities
- Commitment to the Educational Program
- Research/Scholarly Activity
- Clinical Acumen/Knowledge

Fellow Evaluation – after each rotation faculty provide the fellow with appropriate feedback via MedHub evaluations concerning his/her abilities related to the six (6) General Competencies, which are:
- Medical Knowledge
- Patient Care
- Interpersonal & Communication Skills
- Professionalism
- Practice-Based Learning & Improvement
- Systems-Based Practice

Self-evaluation – The fellow completes a self-evaluation form twice yearly.

Semi-Annual Evaluation Form – This is completed by the program director to provide the fellow with a semi-annual review for promotion/graduation purposes.
360 Degree Evaluation – Midpoint during the year, a 360 Degree Evaluation will be distributed to peers, medical students, nursing staff and technologists, and patients and/or families whom the VSF interacts with. The results of the evaluations will be anonymously tabulated and discussed by the Program Director and VSF.

Speaker/Objectives Evaluation Form – to provide the Fellow or speaker with feedback concerning his/her didactic delivery related to:
- Medical Knowledge
- Professionalism
- Interpersonal & Communication Skills
- Practice-Based Learning & Improvement

Final Evaluation - The program director will provide a final evaluation for each VSF who completes the program. This evaluation will include a review of the VSF’s performance during the final period of education, and should verify that the VSF has demonstrated sufficient professional ability to practice competently and independently. The final evaluation must be part of the VSF’s permanent record maintained by the institution.

Negative feedback about program from trainees is taken very seriously. Any negative feedback regarding a rotation will be addressed by the faculty at the quarterly educational retreat. Negative feedback regarding a faculty member or peer will be addressed by the Program Director or an alternate faculty member if the Program Director is named. Interpersonal issues that are not easily reconciled will be taken to the EOHS for additional input.
Section V: Rotation Schedule(s) and Information

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Section VI: Goals and Objectives

The objectives of the Duke Vascular Surgery Fellowship Training Program are to provide a learning and educational environment that facilitates the mentorship and development of expert and competent surgeon-scientists who will have the tools and abilities to become leaders in the field of vascular surgery. The fellowship is a two-year program encompassing training in surgical, endovascular, and medical treatment for vascular disease.

Year 1

The First Year Vascular Surgery Fellow ("VSF1") year consists of rotations at Duke University Hospital (DUH), a rotation centered around the vascular non-invasive laboratory, a learning basic endovascular intervention, and a rotation managing the vascular service at the Durham VA Medical Center (DVAMC). At DUH, the VSF1 obtains a continued and progressive operative experience in all aspects of Vascular and Endovascular Surgery and gains inpatient experience by overseeing the in-patient care of all vascular patients and all hospital vascular consults. In addition, the VSF1 participates weekly in patient evaluations in the outpatient clinic, which includes, but is not limited to: cerebrovascular occlusive disease, peripheral arterial occlusive disease, peripheral arterial aneurismatic disease, visceral ischemic disorders and treatment of acute and chronic venous disease as well as endovenous laser therapy for management of chronic venous insufficiency and lymphatic disorders. On the DVAMC rotation, the VSF1s will be expected to manage the outpatient clinic and inpatient services independently, with greater independence with regard to faculty oversight. Clinical focus is on end-stage limb salvage, carotid disease, and aortic aneurysm repair. During the vascular non-invasive laboratory rotation, VSFs are expected to master the performance and interpretation of duplex ultrasound studies of the carotids, abdominal aorta, peripheral arteries, vascular bypass grafts, mesenteric arteries, and veins of the upper and lower extremities. Use of duplex ultrasound with endovascular intervention is emphasized, and expertise with ultrasound-guided arterial and venous access is developed. Trainees are expected to sit for and pass the RPVI exam during the first year. Trainees on the ultrasound rotation spend one day per week in the endovascular suite learning techniques of arterial access, imaging strategies, diagnostic angiography, and basic catheter and wire skills.

Duke University Hospital Vascular Service

Patient Care

The VSF1 must demonstrate the ability to manage the Vascular Surgery Service at Duke University Hospital including:

- Direct supervision of a team comprised of general surgery residents, advanced practice providers, and medical students, and coordination of nursing, social services, and administration to optimize patient care.
- Participate in the development of the treatment plan for all patients undergoing medical or vascular surgical care on the Vascular Surgery Service. For in-patients and out-patients, assist in formulating the diagnostic and therapeutic plan for patients having any of the
common vascular diseases including cerebrovascular occlusive disease, peripheral arterial occlusive disease, aneurismal disease of the thoracic aorta, abdominal aorta, and peripheral vessels, cerebrovascular occlusive disease, visceral ischemic disorders and treatment of acute and chronic lymphatic and venous disease as well as endovenous therapies for venous reflux.

- Contribute to the presentation of appropriate patients during Vascular Morbidity and Mortality Conference and during hand-offs.
- The VSF1 should utilize the Year 1 rotations to both build upon and acquire new patient management skills as outlined under Medical Knowledge and those skills required for completion of prior years of training.
- Perform the following procedures with appropriate supervision:
  - **Endovascular Training**
    - Complex cases requiring a combined endovascular and open surgical approach.
    - Ultrasound-guided interventions such as bedside insertion of IVC filters and ultrasound guided treatment of femoral pseudoaneurysms.
    - Endovascular grafts to treat aneurismal disease of the thoracic and abdominal aorta.
  - **Open Vascular Surgery**
    - Thoracoabdominal and infrarenal abdominal aortic repair.
    - Operative treatment of extracranial cerebrovascular disease.
    - Operative treatment of great vessels.
    - Operative treatment of peripheral arterial occlusive disease (aortic and infrainguinal).
    - Operative treatment of visceral and renal lesions.
    - Extremity amputations.
    - Arterial and venous reconstructions after cancer resection.
    - Creation and maintenance of dialysis access, including complex dialysis access procedures
    - During Year 1, the VSF1 will gain an understanding of the operative techniques and exposures required to perform the above procedures, and will, as ability allows, progress towards mastery, which should be achieved in Year 2.

**Medical Knowledge**

The VSF1 should:

- Demonstrate mastery of all knowledge acquired in prior years (vascular knowledge accrued from general surgery training).
- Demonstrate application of acquired knowledge to the preoperative selection, operative and perioperative care, and avoidance and management of complications of patients on the Vascular Surgery Service.
- Discuss the skills necessary to direct the care of patients and supervise general surgery residents and medical students in an acute care specialty
- Discuss, in detail, the management of:
- Extracranial cerebrovascular disease
- Abdominal, thoracic and thoracoabdominal aneurysms
- Peripheral arterial occlusive disease
- Visceral and renal disorders
- Venous disorders
- Dialysis access

- Imaging modalities (CTA, MRI/MRA, vascular non-invasive lab)
- Understand the treatment plan for patients with wound and graft infection
- Evaluate and manage patients with traumatic vascular injuries
- Understand the indications for arteriography in blunt and penetrating arterial injuries

**Practice Based Learning and Improvement**

The VSF1 should demonstrate the ability to:

- Critically evaluate published literature regarding the diseases managed on the Vascular Surgery Service, and formulate evidenced-based therapeutic plan.
- Analyze the surgical complications in patients on the service in which he or she have been involved, and present them at the Morbidity and Mortality Conference in a constructive and educational manner.
- Take considerable initiative in facilitating the learning of general surgical residents and medical students.
- Apply clinical trials data to patient management.
- Lead academic and clinical discussions.
- Attend and actively participate, and direct teaching conferences as appropriate.

**Interpersonal and Communications Skill**

The VSF1 should demonstrate the ability to:

- Establish and maintain professional and therapeutic relationships with patients and healthcare team members.
- Manage and maintain efficiency of the team.
- Effectively counsel patients regarding options for surgical and non-surgical therapies.
- Demonstrate behaviors that reflect an ongoing commitment to continuous professional development, ethical practice, sensitivity to diversity and responsible attitudes.

**Professionalism**

The VSF1 should:

- Demonstrate commitment to their patients superceding personal self-interests, including readiness to provide appropriate bedside and operative care based on severity of illness.
• Demonstrate sensitivity to age, gender, and culture of patients and other health care professionals.
• Actively seek and be receptive to feedback on performance.
• Be involved in end-of-life discussions and decisions.
• Demonstrate leadership of the multidisciplinary team

Systems Based Practice

The VSF1 should:

• Demonstrate the ability to efficiently and effectively organize the care of the surgical and non-surgical vascular patient in a cost-effective and evidenced-based manner.
• Appropriately recruit other specialists and health care professionals to optimize the efficiency of care of the vascular surgery patient.
• Be sensitive to medical-legal issues.
• Be facile in the use of technological and computer-based resources.

Endovascular Therapy

Patient Care

The VSF 1 will be primarily focusing on performance of Endovascular Procedures in a dedicated imaging suite, and acquiring experience with multiple imaging modalities relevant to vascular disease. There will also be more focus on evaluation of patients in the outpatient setting with application of the relevant imaging to the clinical problem.

• Assist faculty with supervision of the vascular clinics.
• Participate in the development of a therapeutic plan from the standpoint of endovascular intervention. This would include review of previous studies to determine appropriate imaging and intervention strategies.
• Contribute to the presentation of appropriate patients to the weekly Vascular Conference.
• Perform the following procedures with appropriate supervision:
  ○ Endovascular Training
    ▪ All aspects of endovascular management including basic and advanced catheterization skills, principles of diagnosis, and therapeutic endovascular procedures including angioplasty, stenting, embolization and endografting.
    ▪ Ultrasound-guided interventions such as bedside insertion of IVC filters and ultrasound guided treatment of femoral pseudoaneurysms.
    ▪ Endovascular grafts to treat failed dialysis access and aneurysmal disease
    ▪ During Year 1, the VSF1 will focus on basic and intermediate skills, such as diagnostic arteriography of the aorta, lower extremities, and aortic arch, iliac stenting and angioplasty, and venography and venous interventions.
Venous Disease: The VSF1 will participate in the evaluation of patients with venous disease in the venous clinic as well as learning basic techniques of venous ablation for the greater saphenous vein and sclerotherapy of varicosities.

- Use of closure devices at vascular access sites.
- Percutaneous thrombin injection for post-catherization pseudoaneurysms

**Medical Knowledge:**

- Principles of radiation safety, including the concepts of time, distance, and shielding in limiting patient and staff radiation exposures.
- Knowledge of digital subtraction angiography and post processing methods of image enhancement.
- Techniques of endovascular intervention, including balloon angioplasty, intravascular stent, stent-graft placement, thrombolysis, transcatheter occlusion, and intravascular foreign body retrieval.
- Indications for diagnostic angiography and endovascular intervention.
- Results and limitations of endovascular therapies.
- Experience with percutaneous arterial and venous access, including femoral, brachial, and popliteal punctures, both retrograde and antegrade.
- Knowledge of intravascular contrast agents; iodinated contrast, carbon dioxide, gadolinium, including dosage, use and complications.
- Knowledge of endovascular instruments: catheters, guidewires, catheter-mounted balloons, balloon-expandable and self-expanding intravascular stents, stent-grafts, infusion catheters, embolization coils, and snares.
- Knowledge of the complications of vascular access and endovascular interventions, and experience with management of complications.

**Practice Based Learning**

- Develop ability to critically analyze the endovascular literature in order to practice evidence-based medicine.
- Organize with attending input pre-procedure and post-procedure care of patients undergoing endovascular procedures.
- Present and discuss endovascular patient management at preop vascular conference, attending rounds and morbidity and mortality conference.

**Interpersonal Relationships and Communication**

- Work effectively with angiographic health care personnel (nurses, clerical, technical staff).
- Provide leadership and organization of patient's receiving endovascular interventions.
- Provide patients with clear informed consent regarding endovascular procedures.

**Systems Based Practice**
• Understand the organization of the OR endovascular suite.
• Demonstrate cost effective endovascular care by being knowledgeable in the costs of endovascular devices and pharmaceuticals.
• Know how to partner with angiography directors, nurses and technologists in order to provide optimal delivery of endovascular care.
• Follow established practices, procedures, and policies of the Department of Surgery and Division of Vascular Surgery concerning endovascular therapy.
• Understand principles of endovascular therapy and quality assurance practices

**Durham VA Medical Center**

The VA rotation offers the opportunity for the VSF1 to directly manage all phases of vascular surgical care in a population with a heavy burden of very advanced vascular disease. VSF 1 will be expected to take primary responsibility for decision-making and surgical planning with the assistance of the faculty.

**Patient Care**

• Manage the Vascular Surgery Clinic including surgical resident and clerical staff
• Efficiently use mid-level providers to facilitate patient care
• Efficiently complete and direct patient care activities on the ward
• Demonstrate the ability to gather essential and accurate patient information in the Emergency Department
• Develop appropriate diagnostic workups of vascular disease with a full understanding of the tests to be ordered
• Independently run daily rounds and implement a daily care plan
• Make informed decisions about diagnostic and therapeutic interventions.
• Implementation of a plan of care for lower extremity amputation

**Medical Knowledge and Skills**

• Manage Complex aneurysmal disease in a patient population with multiple comorbidities
• Learn to independently perform exposures of the supraceliac and juxtavisceral aorta as related to complex aneurysm repair
• Learn standard and complex carotid exposure as well as various approaches to carotid endarterectomy
• Understand options for hemodialysis access as well as current practice guidelines for maximizing the use of arteriovenous fistulas
• Understand decision-making around open versus endovascular management of aneurysmal and occlusive disease
• Directly manage the immediate post-operative ICU care in patients with complex vascular reconstructions
Practice-based learning

- Presentation of patients to the entire surgical team at weekly attending rounds
- Direct the teaching of general surgery residents with regard to vascular disease
- Educate the mid-level providers on the finer points of management of the vascular patient
- Present VA deaths and complications at vascular conference

Systems based practice

- Coordinate the weekly surgical schedule with regard to OR and attending availability
- Learn to provide safe patient care in an environment with distinctly limited resources
- Recognize patients requiring a higher level of care and facilitate transfer
- Effectively advocate for appropriate patient care when encountering multiple bureaucratic obstacles
- Ensure completion of medical records in a timely fashion

Interpersonal Relationships and Communication

- Effectively communicate with support staff in the OR to advocate for appropriate patient care
- Communicate with multiple vascular surgery attendings regarding therapeutic plans to ensure smooth operation of the service
- Manage resident staff to minimize conflicts and improve morale in a stressful, resource-limited environment

Vascular Non-Invasive Laboratory

Patient Care

- Demonstrate the ability to gather essential patient information prior to performing a vascular lab study.
- Make informed decisions about the limitations and accuracy of noninvasive ultrasound and physiologic vascular studies.
- Acquire skills for selected noninvasive ultrasound and physiologic vascular studies.

Medical Knowledge & Skills

- Knowledge of the physics of blood flow.
- Interpretation of extremity arterial and venous physiologic studies using standardized criteria.
- Interpretation of carotid, renal, visceral, aortic and extremity arterial duplex studies using video-imaging review.
- Interpretation of vena cava and venous duplex studies using video-imaging review.
• Knowledge of technical skills required for diagnostic scanning.
• Understand the indications, accuracy and diagnostic utility of specific noninvasive vascular tests.
• Knowledge of statistical analysis used to assess the accuracy of vascular studies, including receiver operator curves, kappa statistic, specificity, sensitivity, positive predictive value, negative predictive value and accuracy.

Practice Based Learning

• Pursue a personal program of self-study and professional growth with guidance from the teaching staff and vascular lab medical and technical directors. This will likely be a course taken in preparation to pass the RPVI.
• Participate in general surgery resident and medical student teaching concerning noninvasive vascular studies.

Systems Based Practice

• Understand how an effective, efficient vascular laboratory is organized.
• Demonstrate knowledge of the costs of the various vascular studies as well as the equipment and disposables required for those studies.
• Recognize the importance of allied health care personnel (clerical and technical staff) to a properly functioning vascular laboratory
• Follow established practices, procedures, and policies of the Vascular Laboratory.
• Complete vascular laboratory documentation and medical records promptly.
• Understand principles of Vascular Laboratory quality assurance practices.
• Demonstrate the knowledge required to implement and coordinate a vascular laboratory quality assurance program.

Interpersonal relationships and communication

• Develop a working relationship with the vascular technologists to facilitate learning duplex technique
• Coordinate with attending staff for supervised study interpretation

Year 2

The objectives of the Duke Vascular Surgery Fellowship Training Program are to provide a learning and educational environment that facilitates the mentorship and development of expert and competent surgeon-scientists who will have the tools and abilities to become leaders in the field of vascular surgery. The fellowship is a two-year program encompassing training in surgical and endovascular treatment for vascular disease.
The Second Year Vascular Surgery Fellow ("VSF2") year consists of rotations in endovascular intervention, at Duke University Hospital (DUH), and the Durham VA Medical Center. During the endovascular rotation, the second-year fellow will be expected to develop and demonstrate competency with advanced interventions such as coil embolization, renal and visceral angioplasty and stenting, and thrombolysis. Emphasis will be on independent decision-making, as fellows are expected to master basic technical skills in the first year. At DUH, the VSF2 obtains a continued and progressive operative experience in all aspects of Vascular and Endovascular Surgery and gains inpatient experience by overseeing the in-patient care of all vascular patients and all hospital vascular consults building on the fundamentals from the first year. In addition, the VSF2, with appropriate supervision, is primarily responsible for the management of the outpatient clinic, which includes, but is not limited to: cerebrovascular occlusive disease, peripheral arterial occlusive disease, aneurysmal disease of the thoracic aorta, abdominal aorta, and peripheral vessels, cerebrovascular occlusive disease, visceral ischemic disorders and treatment of acute and chronic lymphatic and venous disease as well as endovenous therapies for venous reflux. At the VA, the fellow will be managing the service with a greater level of responsibility, which is expected to increase during the course of the rotation.

**Duke University Hospital Vascular Service**

Goals for the second year are similar to the first year, however, the VSF 2 will be expected to take a lead role in both formulating a treatment plan and in operative management. The VSF 2 will be expected to do all routine vascular exposures independently, and perform the majority of the reconstruction with attending assistance.

**Patient Care**

While the VSF2 is expected to manage the service, the VSF 2 should be able to independently formulate and direct a treatment plan, and prioritize the patient care, managing the weekly operating room schedule in conjunction with the attending staff.

- Take a lead role in management of the vascular team to facilitate optimal patient care, ensuring timely operative therapy and moving patients through the system as quickly as possible.
- Organize the weekly vascular conference with case presentations and topical lectures.
- Build on the knowledge from the VSF 1 rotation to address more complex vascular surgical issues including complex aortic disease, thoracic outlet syndrome, central venous pathology and re-operative infra-inguinal bypass.
- Perform the following procedures with appropriate supervision:
  - **Endovascular Training**
    - Deployment of branched endografts and use of the “snorkel” technique for renal artery preservation
    - Complex central venous problems including difficult IVC filter extraction, May-Thurner syndrome, and IVC/iliac vein occlusion
    - Retrograde visceral stenting for acute mesenteric ischemia
- Endovascular adjuncts to address endoleak in both TEVAR and EVAR
  - **Open Vascular Surgery**
    - Open thoraco-abdominal aneurysm repair
    - Complex carotid disease including high exposure and re-do surgery.
    - Cervical revascularization in the setting of TEVAR in the aortic arch.
    - Multidisciplinary approaches to thoracic outlet syndrome
    - Operative treatment of visceral and renal lesions.

**Medical Knowledge**

The VSF2 should:

- Demonstrate mastery of all knowledge acquired in prior years (vascular knowledge accrued from the VSF 1 year).
- Demonstrate application of acquired knowledge to the preoperative selection
- Discuss, in detail, the management of:
  - Infected prosthetic grafts
  - Re-do infrainguinal bypass
  - Endovascular and open approaches to ruptured AAA
  - Visceral and renal disorders including debranching for TAAA repair

- Relative strengths and weaknesses of various imaging modalities
- Graft surveillance with duplex ultrasound and revision of the failing graft
- Evaluation and management of patients with iatrogenic vascular injuries in the neck and chest

**Practice Based Learning and Improvement**

The VSF2 should demonstrate the ability to:

- Participate in a program of quality improvement to improve practice on the vascular surgery service.
- Use information presented at morbidity and mortality conference to alter practice and avoid future complications.
- Recognize strengths and weaknesses of team members to assign tasks appropriately.
- Lead academic and clinical discussions at journal club and Friday conference

**Interpersonal and Communications Skill**

The VSF2 should demonstrate the ability to:

- Interact constructively with referring physicians maintain the vascular surgery referral base
- Coordinate with other services to facilitate multidisciplinary care.
- Effectively counsel patients regarding risks inherent in interventional therapies.
• Demonstrate behaviors that reflect an ongoing commitment to continuous professional development, ethical practice, sensitivity to diversity and responsible attitudes.

**Professionalism**

The VSF2 should:

• Demonstrate commitment to the service including effective management of interpersonal conflicts, adjustment to political issues, and flexibility with regard to patient care needs at off-hours
• Recognize and manage competing agendas of attending staff, nursing, and administration
• Actively seek and be receptive to feedback on performance.
• Recognize when care has become futile and effectively initiate end-of-life discussions

**Systems Based Practice**

The VSF2 should:

• Effectively coordinate with consult services and support staff to efficiently move patients through their hospital stay.
• Coordinate with discharge planning to transition quickly to an outpatient environment
• Utilize the existing medical record system in the most efficient manner possible

**Endovascular Therapy**

The VSF 2 should continue to hone basic endovascular skills learned in the first year, while developing the judgment needed to decide which of many possible interventions is most appropriate for the individual patient. In addition, the VSF 2 will be expected to be the primary operator on the most advanced interventions.

**Patient Care**

The VSF2 should already have developed basic endovascular skills including principles of radiation safety, operation of the imaging equipment, and basic catheter and wire manipulation techniques. At this point, they should be focusing on decision-making with regard to the most appropriate technique and device to address the clinical problem.

• Be able to appropriately size balloons and stents, and coordinate the appropriately sized sheath and wire length for the system chosen.
• Decide on the most appropriate device to address individual pathology and discuss the strengths and weaknesses of various approaches.
• Perform the following procedures with appropriate supervision:
Endovascular Training

- Use of microcatheter systems for type II endoleak embolization
- Endovascular treatment of mesenteric occlusive disease.
- Carotid stenting
- During Year 2, the VSF2 will focus on intermediate and advanced skills, and should be independently selecting the appropriate catheter and wire for a given clinical situation
- Venous Disease: Re-canalization with angioplasty and stenting of long-segment venous occlusions including the use of IVUS
- Use of the pre-close technique for large-bore access closure.

Medical Knowledge

- Knowledge of the strengths and weaknesses of various access points (i.e. brachial, radial, femoral)
- Techniques for more complex intervention including microcoil embolization and renal/visceral stenting
- Knowledge of patient selection criteria for carotid stenting
- Making a realistic assessment of possible complications of endovascular intervention
- Learning the basic algorithm for managing AV malformations
- More detailed knowledge of advanced endovascular instruments: reversed curve catheters, CTO wires, cutting balloons, percutaneous mechanical thrombectomy devices, infusion catheters, micro-embolization coils, and use of snares for “body floss.”
- Relative merits of endovascular and surgical management of complications.
- Understand deployment of stent-grafts for trauma

Practice Based Learning

- Develop ability to critically analyze the endovascular literature in order to practice evidence-based medicine.
- Organize with attending input pre-procedure and post-procedure care of patients undergoing endovascular procedures.
- Present and discuss endovascular patient management at preop vascular conference, attending rounds and morbidity and mortality conference.

Interpersonal Relationships and Communication

- Function well in a multidisciplinary environment with cardiology and radiology personnel
- Communicate with the inpatient team to provide seamless transition from outpatient intervention to inpatient care
- Explain complex endovascular intervention to patients in clear, understandable terms.
Systems Based Practice

- Understand the organization of the OR endovascular suite.
- Manage device use to minimize costs of endovascular intervention
- Assess complications of endovascular intervention to minimize the impact on subsequent care

Durham VA Medical Center

The VA rotation offers the opportunity for the VSF2 to function autonomously in preparation for independent practice. The VSF 2 should be taking responsibility for the total care of the vascular patient, with reduced attending input. Specific goals for the VSF 2 during the DVAMC rotation include the following.

Patient Care

- Direct the operative cases including exposure and reconstruction
- Formulate and implement treatment strategies in the vascular clinic
- Manage emergent vascular issues independently with appropriate attending staff supervision
- Consult with other services as appropriate to facilitate multidisciplinary care

Medical Knowledge and Skills

- Discuss the relative merits of endovascular and open reconstruction for patients with AAA and significant comorbidities
- Assess patients for hemodialysis access and implement strategies to maximize utilization of primary arteriovenous fistulas
- Direct the treatment of patients with lower extremity arterial occlusive disease

Practice-based learning

- Presenting VA complications and deaths at the Divisional D&C conference.
- Bringing interesting VA cases up for discussion at the Friday case conference

Systems based practice

- Be aware of metrics used by VA administration to gauge service quality
- Manage the operating room schedule to ensure maximal utilization of operative time
- Understand resource allocation in a closed healthcare enterprise

Interpersonal Relationships and Communication

- Maintain independent decision-making while keeping attending staff apprised of key clinical events
• Delegate effectively to mid-level providers while respecting their limited availability and motivation
• Manage resident staff with varying levels of interest in vascular surgery
Section VII: Department of Surgery Vascular Surgery Fellowship Specific Curriculum:

The following information was adapted to the Vascular Fellowship from the Association of Program Directors in Vascular Surgery Curriculum, 2004.

Please refer to attachments.

In the coming year, the curriculum and goals and objectives will be moving to a learning module based format which includes didactics of disease process, operative and medical interventions, and simulator activity.
BASIC SCIENCE CURRICULUM

1. EMBRYOLOGY OF THE VASCULAR SYSTEM

1. Embryologic development
   - Initial formation of the vascular system (arteries and veins).
   - Development of the aortic arch.
   - Development of the thoracic and abdominal aorta.
   - Development of the arteries to the extremities.
   - Development of the superior vena cava.
   - Development of the inferior vena cava.

2. Embryologic anomalies
   - Aortic arch anomalies (including double aortic arch, right aortic arch, retroesophageal right subclavian artery, absence of the internal carotid artery, patent ductus arteriosus and coarctation of the aorta).
   - Lower extremity arterial anomalies (including persistent sciatic artery, single umbilical artery, and popliteal entrapment syndrome).
   - Superior vena cava anomalies (including double superior vena cava and left-sided superior vena cava).
   - Inferior vena cava anomalies (including transposition or left-sided inferior vena cava, retroaortic left renal vein, circumaortic left renal vein, retrocaval ureter and absent suprarenal inferior vena cava).

References

Against a background of normal embryology, the authors describe the specific pathological conditions and critically review the embryogenesis of a large number of congenital defects, supplying estimates of their frequency, their distribution within the population, and their prognosis. The diagnostic approach and the principles underlying their surgical correction are also discussed.


This paper reviews a 30-year experience of a single center with aortic arch anomalies. Their clinical presentation, diagnosis and treatment are also briefly reviewed.


This article describes the embryologic development and anomalous persistence of the sciatic artery, the pathologic changes that may occur in the persistent sciatic artery and the management of complications related to these pathologic changes.

Popliteal entrapment is an uncommon condition that may cause claudication in young males, some of whom have normal resting pedal pulses. This article reviews popliteal artery entrapment syndrome; it includes the embryology of the arterial supply to the leg and the non-invasive imaging techniques now used in the diagnosis of the condition.


Anomalies of the inferior vena cava are uncommon but important entities to the radiologist and the vascular surgeon. Improper embryogenesis of the inferior vena cava may result in four anatomic anomalies: duplication of the inferior vena cava, transposition or left-sided inferior vena cava, retroaortic left renal vein and circumaortic left renal vein. This paper presents two patients with inferior vena cava anomalies and reviews the embryologic basis and the diagnosis of these rare clinical entities.
2. MOLECULAR BIOLOGY

I. Molecular biology
Cell growth cycle.
Replication, transcription, translation.
DNA and RNA structure and function.
Plasmids, vectors and transfection.
Gene expression, promoters, enhancers.
Basic techniques and assays (Western blot, Northern blot, polymerase chain reaction, in vitro transcription assay, in situ hybridization).
Basic terms in molecular biology.

II. Molecular diagnostics
Aims.
Techniques.
Indications.

III. Molecular biology in the treatment of vascular disease
Gene therapy (systemic and local) including methods and indications.
Cell therapy (ex vivo gene therapy).
Therapy with recombinant protein.
Ethical considerations.

References

This landmark textbook in the field of molecular cell biology summarizes a wide range of knowledge from a basic discussion of cells, genomes and cell chemistry to recent advances in biotechnology, cellular mechanisms, infection and immunity.


The etiology of cardiovascular diseases is known to be multi-factorial. Some of them are causes by environmental factors, some others by specific gene defects, while others result from complex gene-environment interactions. This review article presents molecular diagnostic techniques that have been applied for rapid and reliable detection of specific gene defects. These techniques can provide unequivocal diagnosis beneficial for appropriate drug therapy and genetic counseling.


Gene therapy is emerging as a potential strategy for the treatment of cardiovascular diseases, such as peripheral arterial disease, ischaemic heart disease, restenosis after angioplasty, vascular bypass graft occlusion and transplant coronary vasculopathy, for
which no known effective therapy exists. This review presents recent progress in gene therapy for cardiovascular disease.


This paper discusses the need for development of a new framework for the ethical discussion of genetic interventions into the human genome. It also presents the core arguments for the acceptance of somatic gene therapy and those for the rejection of genetic interventions into germ-line cells.


Gene transfer to the vascular system can be performed both via intravascular and extravascular periadventitial routes. This review describes both of these techniques, their aims and initial results.
3. PHYSIOLOGY AND PATHOPHYSIOLOGY OF BLOOD VESSELS

1. Physiology
   - Circumferential, longitudinal and radial deformation and stresses of blood vessels.
   - Properties of collagen, elastin and glycosaminoglycan ground substance.
   - The length-active stress curve of the vascular muscle.
   - Changes induced by aging.

2. Pathophysiology
   - Aneurysms
   - Histological changes
   - Causes of aneurysms (including the role of proteases, inflammation, autoimmunity and atherosclerosis)
   - Mechanisms preventing instantaneous enlargement of aneurysms.

3. Poststenotic dilatation
   - Histological changes.
   - Mechanisms for poststenotic dilatation.

4. Autogenous vein grafts
   - Histological changes.
   - Mechanisms for the beneficial effects of vein grafts.
   - Distribution and causes of intimal hyperplasia in end-to-side vascular anastomoses.

5. Arteries of hypertensive subjects
   - Histological changes.
   - Behavior and control of the precapillary resistance vessels.
   - Atherosclerotic arteries
   - Histological and mechanical changes.
   - Distribution of vasa vasorum.

References


Results of elastolytic and collagenolytic studies on canine and human arteries are presented. Treatment with elastase caused the vessels to dilate but to remain intact, while all vessels treated with collagenase ruptures. The authors conclude that wall integrity depends on intact collagen rather than elastin.


This paper presents the pathogenetic mechanisms of poststenotic dilatation. According to experimental studies, shear stress and turbulence are the most likely causes. Whatever
the flow disturbance, it must may the wall vibrate to produce poststenotic dilatation. Vibrations are thought to produce alterations in wall elastic and possibly in vascular smooth muscle tone.


This paper presents experimental data showing that intimal hyperplasia is best associated with low flow velocity, a factor correlated with low blood-artery shear stress. By contrast, medial thickening is best associated with increased deformation of the vein wall in the circumferential direction (increased diameter). These findings correlate with clinical responses of vein grafts.


This is a study of the response of artery segments to enlarging atherosclerotic plaques. It shows that coronary arteries enlarge in response to increasing atherosclerotic plaque and that such enlargement can prevent narrowing of the lumen. However, differential segments of the same artery may respond differently. Local differences in the relative rates of plaque growth and artery enlargement may determine progression to stenosis, preservation of normal lumen area, or enlargement.


Exposure of vein grafts to arterial pressure increases the following nine mechanical factors: deformation in the circumferential, longitudinal, and radial directions; stresses in each of these three directions; pulsatile deformations and pulsatile stresses; and flow velocity. The experiments presented in this article demonstrate that intimal thickening is best correlated with low flow velocity, a correlate of low shear stress, whereas medial thickening is best correlated with deformation in the circumferential direction.
4. HEMODYNAMICS AND ATHEROSCLEROSIS

Physiology – Pathophysiology
Types of blood flow (laminar, turbulent) and their determinants (Reynolds number).
Hemodynamic forces (shear stress, tensile stress): definitions and equations.
Vessel wall properties affecting the development of atherosclerosis (thickness, elasticity, number of vasa vasorum).
Shear stress effects on the endothelium.
Causes and effects of turbulence.
The role of hypertension in atherosclerosis.
Hemodynamic changes associated with arterial stenoses.
Hemodynamics associated with anastomoses.

Clinical implications
Plaque localization in the carotid bifurcation (the effects of low shear stress).
Plaque localization in the coronary arteries (the effects of heart rate).
Plaque localization in the aorta (the effects of low flow velocity, the curvature of the abdominal aorta and the aortic bifurcation).

References

This review provides fundamental knowledge on the predominant hemodynamic forces that have been characterized: shear stress and cyclic circumferential strain. The role of hemodynamics in the localization of atherosclerosis is discussed as well as the intracellular events that link hemodynamic stimuli and endothelial cell response.


This article outlines how modern molecular techniques are being utilized in studies of endothelia mechanotransduction associated with controlled shear stress in vitro and hemodynamics in vivo. The value of such techniques as components of an integrated understanding of vascular rheology is emphasized.


This review outlines the mechanisms that link hemodynamic factors to plaque development and rupture and describes in some detail recently developed techniques that, for the first time, make it possible to determine these factors in vivo.

Phenotypic modulation of endothelium to a dysfunctional state contributes to the pathogenesis of cardiovascular diseases such as atherosclerosis. This article reviews the role of the vascular endothelium in the atherosclerotic disease process, the impact of the various types of hemodynamic forces on vessel wall biology and the mechanisms of endothelial gene regulation by biomechanical forces.


This article summarizes the basic concepts of arterial hemodynamics and wall mechanics as they relate to the development of arterial pathology. A few practical mathematical relationships and examples are provided for both illustration and utilization. The use of computer models for the estimation of wall stresses in individual abdominal aortic aneurysms is also discussed.
5. PEPTIDE GROWTH FACTORS

1. General considerations
   Function of growth factors.
   Growth factor receptors and mechanism of action.

2. Specific growth factors
   Platelet-derived growth factor (characteristics, receptor, effects, role in proliferative diseases of the vascular system).
   Fibroblast growth factor (characteristics, receptor, effects, role in proliferative diseases of the vascular system).
   Epidermal growth factor (characteristics, receptor, effects, role in proliferative diseases of the vascular system).
   Transforming growth factors α and β (characteristics, receptor, effects, role in proliferative diseases of the vascular system).
   Insulin-like growth factors (characteristics, receptor, binding protein, effects, role in proliferative diseases of the vascular system).

References

   This paper reviews the role of various growth factors, such as platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), insulin, insulin-like growth factor-I (IGF-I), and transforming growth factors alpha and beta (TGF alpha and beta), in the development of arteriosclerosis and intimal hyperplasia.


   This review describes the vascular biology of PDGF. It particularly focuses on recent findings regarding the intracellular signals activated by PDGF in the context of vascular smooth muscle cell proliferation, migration and contraction.


   This paper presents evidence that hypercholesterolemia and oxidized low-density lipoprotein impair endothelial cell growth by suppressing basic fibroblast growth factor expression. Background studies on the subject are briefly reviewed.

Transforming growth factor beta isoforms have been strongly implicated in a number of pathophysiologic processes including chronic vascular diseases such as atherosclerosis and hypertension. This article reviews the molecular mechanisms by which these factors exert their complex and pleiotropic actions on cells and tissues of the cardiovascular system.


Dysregulated actions of insulin-like growth factors I and II have been found to contribute to coronary atherosclerosis and restenosis. This article first reviews the basic physiology of the IGF axis and then discusses specific autocrine and paracrine actions of IGFs in atherosclerotic plaque progression and the neointimal hyperplasia of restenosis.
6. ENDOTHELIAL CELLS

1. Development of the endothelium

2. Structure of the endothelium
   Extracellular matrix.
   Endothelial cell cytoskeleton.
   Endothelial cell integrins.

3. The endothelium as a metabolic organ

4. Endothelial interactions
   Endothelium and platelets.
   Endothelium and lymphocytes.
   Endothelium and leukocytes.

5. Culture of endothelial cells
   Large vessel endothelial cells (including human umbilical vein endothelial cells and bovine aortic endothelial cells).
   Microvascular endothelial cells.
   Identification of endothelial cells.
   Endothelial cell culture media.

References

This paper reviews the role of endothelium on atherogenesis and emphasizes the profound prognostic and therapeutic implications of endothelial dysfunction.


This article reviews the scientific and clinical evidence showing that changes in endothelial cell physiology are an important component of inflammatory and atherosclerotic vascular disease.


The endothelium is critically involved in the regulation of vascular function through its barrier role, via interaction with circulating cells such as platelets, which then release vasoactive or growth regulating agents, and through production of substances which may modulate vascular tone, smooth muscle cell growth and coagulation. This article reviews
the role of the endothelium in health as well as in various diseases such as hypertension, atherosclerosis, diabetes, heart failure, ischemic heart disease, sepsis, and shock.


The endothelium regulates vascular tone and growth by releasing factors involved in relaxation and contraction, in coagulation and thrombus formation, and in growth inhibition and stimulation. The role of various chemical and physical stimuli, mediators and endothelium-derived factors is summarized.


Endothelial cells may produce and release vasoconstrictor substances in response to a number of agents and physical stimuli. This brief review discusses the mechanisms of endothelium-dependent contractions and speculates about the possible importance of such contractions for venous graft function, development of vasospasm, increased vascular resistance in hypertension, and vascular complications in diabetes.
7. VASCULAR SMOOTH MUSCLE CELLS

Smooth muscle cells in vivo
Histology.
Function.

Culture of smooth muscle cells
Isolation, identification and culturing of smooth muscle cells.
Smooth muscle cell media.
Phenotypic modulation of smooth muscle cells in culture (including contractile protein expression, Na\(^+\) pump activity, receptor changes and extracellular matrix formation).

Smooth muscle cell signal transduction
Signaling pathways (including the role of calcium, protein kinase C, tyrosine kinase and cyclic AMP).
Smooth muscle cell agonists (including ATP, serotonin, angiotensin II, endothelin, \(\alpha_1\)-adrenergic agonists, prolactin, atrial natriuretic polypeptide, heparin, thrombin, nitric oxide and vasopressin).
Growth factors produced by smooth muscle cells (including platelet-derived growth factor, heparin-binding epidermal growth factor, fibroblast growth factor, transforming growth factor \(\beta_1\) and insulin-like growth factor).

References

VSMCs exhibit several growth responses to agonists that regulate their function including proliferation, hypertrophy, endoreduplication, and apoptosis. This review discusses the autocrine and paracrine growth factors important for VSMC growth in culture and in vessels. Four mechanisms by which individual agonists signal are described: direct effects of agonists on their receptors, transactivation of tyrosine kinase-coupled receptors, generation of reactive oxygen species, and induction/secretion of other growth and survival factors. Additional growth effects mediated by changes in cell matrix are discussed.


This article reviews the different factors that are involved in the control of VSMC proliferation, especially in the context of cardiovascular disease. Therapeutic approaches that targeted specific cell-cycle control genes or growth regulatory molecules which effectively inhibited neointimal lesion formation are also discussed.

Apoptosis describes the morphological changes that identify a specific form of regulated cell death. This article reviews the role of apoptosis in the maintenance of vascular homeostasis. Specifically, it addresses the role of vascular smooth muscle cell death, how this may be regulated at the molecular level and whether any of these molecular mediators will provide targets for intervention in diseases such as atherosclerosis.


This review focuses on an emerging field of cardiovascular research in which the direct effects of mechanical strain on VSM cells and isolated blood vessels in organ culture have been characterized, in vitro.


The accumulation of vascular smooth muscle cells plays an important role in the development of atherosclerotic plaques and in the restenotic process occurring after balloon angioplasty. This paper reviews the role of various growth factors and cytokines in vascular smooth muscle cell proliferation and migration.
8. MACROPHAGES

General considerations
Macrophage production.
Macrophage kinetics.
Macrophage function.

The role of macrophages in vascular disease
Chemoattractants.
Monocyte endothelial cell adherence.
Monocyte migration.
Macrophage activation.

Macrophage secretory products
Proteases and local tissue destruction.
Cytokines (including interleukin-1, interleukin-6, interleukin-8, interferon, tumor necrosis factor and colony stimulating factors).
Growth factors (including platelet-derived growth factor, fibroblast growth factor and transforming growth factor-β).

The role of macrophages in lipid metabolism

References

In this chapter the role of the monocyte/macrophage in the genesis of the atherosclerotic plaque is discussed. As it is demonstrated, the pivotal role of the macrophage in atherosclerosis depends not only on its ability to handle lipids but also on its physical and secretory functions and its role as a mediator of inflammation.


This article describes the role of macrophages and oxidized low density lipoproteins in the pathogenesis of atherosclerosis. The clarification of this role offers an interesting possibility to reduce atherosclerosis by antioxidants, enzyme inhibitors and other compounds that protect LDL against oxidative damage and/or reduce the subsequent harmful effects of oxidized LDL on various cellular functions.


This review outlines the complex interactions between macrophages, endothelial cells, and lipoprotein oxidation in the pathogenesis of atherosclerosis. The basic steps of the
pathogenetic pathway including trapping of LDL, oxidation of LDL, monocyte chemotaxis, cell transformation into macrophage-derived foam cells, endothelial cell injury and formation of mural thrombi are summarized.

Aviram M, Fuhrman B. LDL oxidation by arterial wall macrophages depends on the oxidative status in the lipoprotein and in the cells: role of prooxidants vs. antioxidants. Mol Cell Biochem 1998;188:149-159.

All major cells in the arterial wall including endothelial cells, smooth muscle cells and monocyte derived macrophages can oxidize LDL. Oxidized LDL is highly atherogenic as it stimulates macrophage cholesterol accumulation and foam cell formation, it is cytotoxic to cells of the arterial wall and it stimulates inflammatory and thrombotic processes. This review article summarizes the above issues with an emphasis on the authors’ own data.


The interactions of endothelial cells, smooth muscle cells, and monocyte-derived macrophages as well as the role of thrombin, monocyte adhesion proteins and platelet-derived growth factor in atherogenesis has been the focus of great interest over the past decades. The resultant information is summarized in this brief review.
9. PLATELETS

General considerations
Platelet production and structure.
Platelet kinetics and life span.
Platelet membrane glycoproteins.

The role of platelets in vascular disease
Adhesion.
Aggregation.
Secretion, including dense bodies, alpha granules, platelet specific proteins (platelet factor 4, β-thromboglobulin, platelet-derived growth factor and thrombospondin).
Inhibition of secretion.

Biochemistry of platelet activation and inhibition
The importance of thromboxane.
Other agonists of platelet activation (including thrombin, collagen, ADP, epinephrine, platelet activating factor and ristocetin).
Platelet inhibitors (including aspirin, indomethacin and sulindac, phenylbutazone and sulfinpyrazone, nonsteroidal anti-inflammatory drugs, dipyridamole, prostacyclin, thromboxane synthetase inhibitors, thromboxane receptor antagonists, ticlopidine and glycoprotein IIb-IIIa inhibitors).

Platelet interactions with coagulant proteins
Coagulation proteins in platelets (including fibrinogen, factor V, von Willebrand factor, high-molecular weight kininogen, factor XI, factor XIII and plasma protease inhibitors).
Platelet contribution to the coagulation mechanism (including contact activation and factor X).

References

Although anucleated, blood platelets are highly organized cells rich in different types of organelles. Dense granules contain small non-protein molecules that are secreted to recruit other platelets. alpha-Granules contain large adhesive and healing proteins. Lysosomes contain hydrolases able to eliminate the circulating platelet aggregate. Granules’ constituents, secretion and functions as well as typical platelet disorders resulting from a storage granule abnormality are described in this review article.


This review focuses on recent developments in elucidating the mechanisms that regulate platelet aggregation. The role of platelet receptors for collagen, von Willebrand factor, thrombin and adenosine diphosphate is briefly reviewed.

This article reviews recent evidence showing how the interaction between platelets and endothelial cells may play a important role in the pathogenesis of atherosclerosis, suggesting an underappreciated potential locus for pharmacologic intervention.


This review addresses our current understanding of platelet function and how this information has been applied to the discovery of novel platelet inhibitors. Platelet inhibitors include inhibitors of prostaglandin-stimulated platelet activation, inhibitors of ADP-mediated platelet activation, phosphodiesterase inhibitors, inhibitors under development (CD39/ATP diphosphohydrolase and thrombin receptor inhibitors) and antagonists of ligand binding to αIIbβ3. The evidence on the efficacy of each platelet inhibitor is summarized.


This review is divided into three sections. The first section considers the contributions of platelet-derived and plasma-derived reactants to prothrombin activation on platelets. The second section briefly reviews the mechanisms of platelet activation and the critical role of platelet activation in hemostasis. The third section reviews some of the pathological consequences that can arise from inadequate regulation of platelet activation.
10. RESPONSE OF THE ARTERIAL WALL TO INJURY AND INTIMAL HYPERPLASIA

General considerations
Degrees of vascular injury.
General characteristics of intimal hyperplasia (including its differentiation from restenosis).

Types of injury resulting in intimal hyperplasia
Angioplasty, stenting, endarterectomy.
Vein grafting.
Prosthetic grafting.

The three-wave model of intimal hyperplasia
Medial smooth muscle cell proliferation (including the role of platelet-derived growth factor, basic fibroblast growth factor and angiotensin II).
Smooth muscle cell migration (including the role of extracellular matrix, platelets, migratory factors and the endothelium).
Intimal expansion (including the regulation of the extent of intimal hyperplasia).

Prospects for control of intimal hyperplasia
Surgical technique.
Promoting endothelialization.
Pharmacologic control of smooth muscle cell activity (including systemic and local drug delivery techniques).
Brachytherapy.
Strategies for the future (including antibodies to growth factors, antisense nucleotides, gene therapy etc).

References

The underlying causes of intimal hyperplasia are migration and proliferation of vascular smooth muscle cells provoked by injury, inflammation, and stretch. This review discusses, at a molecular level, both the final common pathways leading to smooth muscle migration and proliferation and their (patho)-physiological triggers. It emphasizes the key roles played by growth factors and extracellular matrix-degrading metalloproteinases, which act in concert to remodel the extracellular matrix and permit cell migration and proliferation.


In this article the structure and development of neointimal hyperplasia in vascular grafts, both venous and arterial, are reviewed briefly. The clinical outcomes of various arterial grafts that are now being used, including the radial, the internal mammary and the
gastroepiploic arteries, as well as the underlying cell biology of their adaptation to the grafted environment are also reviewed.


*Intimal hyperplasia may be defined as the abnormal migration and proliferation of vascular smooth muscle cells with associated deposition of extracellular connective tissue matrix. In this article, the pathology of intimal hyperplasia is reviewed with particular attention to its physiology, pharmacology, cell biology and molecular biology.*


*The development of the intimal hyperplasia at the outflow anastomosis of a prosthetic bypass or in autogenous saphenous vein bypass placed in the arterial system is responsible for most bypass failures. This article reviews current knowledge on the pathogenesis of myointimal hyperplasia and addresses possible therapeutic considerations for the future.*


*This article briefly reviews the histological evidence for the genesis of intimal hyperplasia and atherosclerosis in arteries. It concentrates upon the origin, structure, behaviour and interactions of vascular smooth muscle cells in the intimal (subendothelial) layer.*
11. ATHEROSCLEROSIS: THEORIES OF ETIOLOGY AND PATHOGENESIS

1. Atherosclerotic lesions
   - Fatty streaks.
   - Gelatinous plaques.
   - Fibrous plaques.
   - Complicated plaques.

2. Theories of atherogenesis
   - Lipid hypothesis.
   - Thrombogenetic hypothesis.
   - Mesenchymal hypothesis.
   - Monoclonal hypothesis.
   - Response to injury hypothesis.

3. Lesion arrest and regression
   - Risk factor modification.
   - Modification of the plaques.

References

   In this review the microscopic appearance of the normal arterial wall, the definition of atherosclerosis and the five theories of atherogenesis are described (the lipid theory, the hemodynamic theory, the fibrin incrustation theory, the nonspecific mesenchymal hypothesis and the response to injury hypothesis). The classification of the atherosclerotic lesions according to Stary (types I-VI) as well as the epidemiology and the role of various risk factors are presented in detail.


   This review article provides insights into the complex biology of arterial atheroma and the etiologic peculiarities of advanced complicated plaques. This knowledge may serve as a basis for identifying high-risk subjects and for novel vascular prevention strategies with focus on plaque stabilization and antithrombotic/anticoagulant measures.


   Pathogenesis of atherosclerosis is reviewed including genetic factors, environmental factors, pathological stages and cell types involved in the disease process. Therapeutic implications of this knowledge are also briefly reviewed.


This report describes the characteristic components and pathogenic mechanisms of the various advanced atherosclerotic lesions. An attempt is also made to correlate the appearance of lesions noted in clinical imaging studies with histological lesion types and corresponding clinical syndromes.


Endothelial dysfunction, which results from biochemical and hemodynamic stresses associated with cardiovascular risk factors, causes an imbalance in the expression of vasodilating and vasoconstricting substances, as well as excess production of chemoattractant molecules and growth factors. The role of these parameters in the process of atherosclerosis is briefly described.
12. HISTOPATHOLOGIC FEATURES OF NONARTERIOSCLEROTIC DISEASES OF THE AORTA AND ARTERIES

Degenerative changes of the aorta and arteries
Age-related changes in the aorta.
Aortic dissection.
Heritable disorders of connective tissue (including Marfan’s syndrome and Ehlers-Danlos syndrome).

Acquired structural defects
Cystic adventitial disease.

Developmental or acquired structural abnormalities of arteries
Fibromuscular dysplasia

Inflammatory conditions of the aorta and arteries
Vasculitis.
Syphilitic aortitis.
Inflammatory aortic aneurysms and retroperitoneal fibrosis.
Rheumatoid panaortitis.
Seronegative spondyloarthritides.
Takayasu’s aortitis.
Giant cell aortitis.
Infectious aortitis.
Collagen vascular diseases.
Scleroderma.
Giant cell arteritis.
Polyarteritis nodosa.
Allergic granulomatosis and angiitis.
Polyangiitis overlap syndrome.
Thromboangiitis obliterans.
Ergot-alkaloid associated arterial disease.

References

This chapter briefly reviews the historical evolution of this heritable disorder of connective tissue and related conditions, discusses the important developments of the past few years, and suggests where progress is most needed in the immediate future.


This is a comprehensive review of fibromuscular dysplasia including its pathology, pathogenesis, natural history, clinical presentation, diagnosis and treatment.
Takayasu arteritis is a well known yet rare form of large vessel vasculitis. This review details the history, clinical features, differential diagnoses, classification, and immunology of the disorder. Current evidence-based treatments are also presented and discussed.


This review paper summarizes myriad basic science studies on the pathogenesis of giant cell arteritis. It also reviews the expanding knowledge of the epidemiology, clinical presentation, diagnosis and treatment of this systemic vasculitis.


Thromboangiitis obliterans (Buerger's disease) is a nonatherosclerotic segmental inflammatory obliterative disease that most commonly affects the small- and medium-sized arteries and veins in both upper and lower extremities. The outline of Buerger’s disease is described in this article.
13. REGULATION OF VASOMOTOR TONE AND VASOSPASM

General considerations
Regulation of vasomotor tone (including the role of mechanical forces, blood elements, vasoactive substances and ions).
Definition of vasospasm and its role in atherogenesis.
Experimental models for the study of blood vessel contractile responses (including the muscle bath, ex vivo perfusion devices, angiography, cell culture and fresh vascular smooth muscle cells).

Vasoconstriction
Molecular mechanisms of vasoconstriction.
Mediators of vascular smooth muscle contraction.

Vasorelaxation
Molecular mechanisms of vasorelaxation.
Mediators of vascular smooth muscle relaxation.

References

This review describes the physiology and biochemistry of NO as it relates to the control of vasomotor tone. Methods of measuring NO in basic science and clinical settings are outlined, and the derangements of endothelial NO production and release (endothelial dysfunction) in pathophysiologic and disease states are discussed. Potential therapies aimed at preserving endothelial function and augmenting NO production are also reviewed.


This review article discusses several of the mechanisms which may undergo change and substantiate the endothelial dysfunction which accompanies experimental arterial hypertension and arterial hypertension in humans. The synthesis and effect of nitric oxide and endothelin as well as the production of free radicals and vasoconstrictor peroxides is also analyzed.


Based on early work by the author and a selective review of the literature, evidence is presented to show how a common cardiovascular event, vasospasm, may be one of the factors responsible for vessel wall injury, producing a substantial arteriopathy in the very vessel in which it occurs.

The endothelium makes a significant contribution to the regulation of vascular tone through the release of potent vasodilator agents such as nitric oxide (NO) and prostacyclin (PGI2) as well as vasoconstrictor compounds such as endothelin. This article describes the mechanisms underlying these functions and their impact on vascular disease.


The contractility of vascular smooth muscle cells is controlled in a complex manner by both extracellular and intracellular messages. This review describes briefly each step of these signalling pathways which are possible sites for potential therapeutic interventions.
14. VENOUS SYSTEM OF THE LOWER EXTREMITIES: PHYSIOLOGY AND PATHOPHYSIOLOGY

1. Anatomy
   The superficial system of veins.
   The deep system of veins.
   The perforating veins.
   Structure of venous wall.

2. Physiology
   Venous hemodynamics.
   Venous pressures.
   The calf muscle pump.
   The thoracoabdominal muscle pump.
   Venous tone.
   Venous endothelium.

3. Clinical tests of physiologic function of the venous system
   Ambulatory venous pressure.
   Arm-foot pressure differential.
   Ascending and descending venography.
   Air-plethysmography.
   Photoplethysmography.
   Duplex ultrasound.

4. Pathophysiology
   Varicose veins (epidemiology, pathology, pathophysiology).
   Chronic venous insufficiency (epidemiology, pathology, pathogenesis).

References

This consensus document provides an up-to-date account of the various methods available for the investigation of chronic venous insufficiency of the lower limbs, with an outline of their history, usefulness, limitations and indications of which patients should be subjected to the tests and when and of what clinical decisions can be made.


This paper is aimed at providing basic anatomic information of the lower extremity venous system. It also highlights the new research areas and the changing concepts on the pathophysiology of varicose veins.
Numerous noninvasive tests including venous duplex ultrasound, photoplethysmography and air plethysmography have been described for assistance in the diagnosis and treatment of patients with chronic venous insufficiency. These tests are reviewed including the typical information obtained, the usefulness of this information, and the relevance for clinical management of patients with CVI. Based on the clinical class, recommendations for a noninvasive testing protocol are outlined.


In this review, the anatomy and physiology of the venous system and its pathophysiology are described. Theories regarding the possible causes of disturbances in venous microangiopathy as well as the recently discovered pattern of perfusion in microcirculation within and around venous ulcers are also discussed.


Chronic venous insufficiency is inseparably linked to elevated venous pressure and is accompanied by vascular, dermal, and subcutaneous tissue damage and restructuring. Among several possible mechanisms (hypoxia, humoral stimulation), a shift in fluid shear stress from normal physiological levels and endothelial distension under the influence of elevated venous pressure may serve as trigger mechanisms for inflammation. The key role of inflammation in chronic venous insufficiency including its trigger mechanisms and consequences is summarized in this review.
15. STRUCTURE AND FUNCTION OF THE LYMPHATIC SYSTEM

Embryology

Structure and function
Peripheral lymphatic system.
Lymph nodes.
Central lymphatic system.
Cellular components of the lymphatic system.

Physiology
Lymph formation.
Lymph flow.

Lymph visualization
Lymphangiography.
Lymphoscintigraphy.

References

This paper presents an overview of the anatomy, physiology, and biology of the lymphatic system specifically relevant to lymphatic drug delivery. It briefly reviews the classic fluid and solute transport literature, and also examines the current research in lymphatic endothelial cell biology and tumor metastasis in the lymphatics because of the increasing potential for targeted delivery of immunomodulators, chemotherapeutics, and genetic material to specific lymph nodes.


This article reviews current knowledge of lymph node cellular architecture as well as of the structure and course of lymphatic vessels. The function of the lymphatic system is also summarized along with its response to pathologic processes.


Recent developments in diagnostic and interventional imaging of lymphatic disorders are reviewed. Several imaging techniques are presented, including lymphangiography, magnetic resonance imaging, ultrasonography, fluorescent microangiography, and intradermal brominated fluorocarbon. The use of these techniques in the treatment of certain lymphatic disorders is also discussed.

The authors review the anatomy and physiology of the lymphatic system. The current understanding of the pathophysiology of lymphedema is also discussed, including its congenital and acquired causes, pathophysiologic consequences and clinical results.


The development of lymphoscintigraphy for surgical research and practice is reviewed. The characteristics of the radiopharmaceutical used, the technique of injection and imaging as well as the most common applications of these nuclear medicine techniques is discussed.
16. DIABETIC VASCULAR DISEASE

1. Insulin
   - Structure.
   - Synthesis.
   - Mechanisms of action.
   - Insulin as a growth factor.

Diabetes mellitus and peripheral vascular disease
Macrovascular disease.
Microvascular disease.
The diabetic foot.

References

This section provides the reader with some basic knowledge on the structure of human insulin and the conversion sequence of proinsulin to insulin. The significance of this knowledge for the clinical practice is also discussed.


The high prevalence of macrovascular disease in non-insulin-dependent diabetes appears to be related to insulin levels and to the degree of hyperinsulinemia as measured in the blood of these patients. The various components of the Metabolic syndrome or Syndrome X are presented in this review along with the suggested therapeutic strategies in these patients.


This article reviews possible pathophysiological mechanisms for diabetic angiopathy in type 2 diabetes. The key role of oxidative stress, endothelial function, and insulin resistance in this process is emphasized.


In this review potential mechanisms for the high prevalence and altered distribution of peripheral vascular disease in patients with type 2 diabetes are explored. It is hypothesized that the metabolic abnormalities in the prediabetic phase predispose to a more distal and aggressive atherosclerosis. Once diabetes has developed this process is accelerated due to chronic hyperglycaemia. Furthermore, endothelial damage, non-
enzymatic glycosylation and polyneuropathy could lead to impaired vascular remodelling and collateral formation.


Diabetic peripheral neuropathy and peripheral vascular disease have been recognized as the most important etiologic factors of diabetic foot problems. The complex interplay between these abnormalities and a number of other contributory factors, such as altered foot pressures, limited joint mobility, glycemic control, ethnic background, and cardiovascular parameters is presented in this brief review article.


This summarizes the complex interaction of neuropathy and vascular disease, including an explanation of the neuroinflammatory response and its role in the pathogenesis of ulceration.
17. PLASMA LIPOPROTEINS AND VASCULAR DISEASE

PHYSIOLOGY
Structure of lipoproteins.
Categories of lipoproteins.
Lipoprotein metabolism.

PATHOPHYSIOLOGY
Abnormal chylomicron metabolism.
Abnormal VLDL metabolism.
Abnormal LDL metabolism.
Abnormal HDL metabolism.

THERAPEUTIC INTERVENTIONS
Diet.
Drug therapy.
Surgical therapy.

THE RELATIONSHIP OF LIPOPROTEINS TO ATHEROGENESIS AND CLINICAL VASCULAR DISEASE
The role of LDL in atherogenesis.
The role of VLDL and chylomicron remnants in atherogenesis.
The role of HDL in atherogenesis.
The relationship of lipoproteins to clinical vascular disease.

References

This review article describes in detail the composition and transportation of lipoproteins and apolipoproteins, including a discussion of cellular receptors and the enzymes relevant to lipoprotein metabolism.

Batiste MC, Schaefer EJ. Diagnosis and management of lipoprotein abnormalities. Nutr Clin Care 2002;5:115-123.

This article provides the reader with an update on the current status of the diagnosis and management of lipid disorders. The guidelines of the National Cholesterol Education Program Adult Treatment Panel III are presented including the current recommendations for treatment by diet and drugs.


This article reviews the pathways of cholesterol entry and removal, the metabolism, and the physical changes of cholesterol in the vessel wall. How these processes are believed to contribute to cholesterol buildup in atherosclerotic plaques is discussed.
Mouse models have greatly advanced our understanding of the pathology associated with altered lipoprotein levels, including cellular uptake, intracellular metabolism, cellular efflux mechanisms and transcriptional regulation. This review article describes progress in all of these areas and shows that animal models are likely to remain important to our view of gene function in the context of the whole organism.


This review discusses the genetic basis of the principal lipoprotein abnormalities associated with coronary heart disease susceptibility in the general population. Individual sections discuss genes regulating LDL cholesterol, HDL cholesterol, and triglyceride levels. A section is included on the effects of the common apo E genetic variation on lipoprotein levels, as well as sections on the genetic regulation of lipoprotein(a) levels, genes regulating the inverse relationship between triglyceride-rich lipoproteins and HDL cholesterol levels, and our current understanding of the genetic basis of familial combined hyperlipidemia.
18. CIGARETTE SMOKING AND VASCULAR DISEASE

Smoking and the development of atherosclerosis
The effects of smoking on endothelial cells.
The effects of smoking on plasma lipoproteins.
The effects of smoking on platelet function.
The effects of smoking on white blood cells.

Pharmacology of nicotine
Pharmacokinetics.
Mechanisms of action.

References

This review concentrates on new evidence regarding the precise components of cigarette smoke responsible for the relationship between cigarette smoking and cardiovascular disease as well as the mechanisms by which they exert their effect.


The relationships between smoking and cardiovascular disease result from multiple mechanisms that interact to contribute to atherosclerosis, vascular injury, thrombosis, and vascular dysfunction. This article reviews our current understanding of how smoking contributes to the genesis and progression of these disorders.


This article reviews the mechanisms of arterial wall damage caused by smoking. Several products of tobacco combustion, including nicotine, free radicals and aromatic compounds, have been shown to cause release of catecholamines, endothelial injury, oxidation of LDL, increase of plasma fibrinogen and alteration of platelet activity. All these proatherogenic effects of smoking are summarized in this paper.


This article review current evidence showing that nicotine contributes, via its hemodynamic effects, to acute cardiovascular events. However, the effects of nicotine are much less important than are the prothrombotic effects of other products of tobacco combustion. Another issue that is emphasized is that the dose response for cardiovascular events of nicotine appears to be flat, suggesting that if nicotine is involved, adverse effects might be seen with relatively low-level cigarette exposures.
Nicotine and carbon monoxide produce acute cardiovascular consequences, including altered myocardial performance, tachycardia, hypertension, and vasoconstriction. Smoking injures blood vessel walls by damaging endothelial cells, thus increasing permeability to lipids and other blood components. Among metabolic and biochemical changes induced by smoking are a tendency for increased serum cholesterol, reduced high density lipoprotein, elevated plasma free fatty acids, elevated vasopressin, and a thrombogenic balance of prostacyclin and thromboxane A2. In addition to rheologic and hematologic changes from increased erythrocytes, leukocytes, and fibrinogen, smokers have alterations in platelet aggregation and survival that produce thrombosis. All of these interactive mechanisms by which smoking exerts its deleterious effects are summarized in this article.
19. COAGULATION AND DISORDERS OF HEMOSTASIS

1. Physiology
   Basic mechanisms of coagulation (extrinsic/intrinsic pathways of coagulation, the role of platelets).
   Natural anticoagulant mechanisms (antithrombin III, proteins C and S, heparin cofactor II).
   Fibrinolysis.

2. Hypercoagulable states
   Heparin-associated thrombocytopenia.
   Antithrombin III deficiency.
   Protein C and S deficiency.
   Factor V Leiden mutation.
   Lupus anticoagulant/antiphospholipid syndrome.
   Abnormalities of fibrinolysis.
   Abnormal platelet aggregation.
   Disseminated intravascular coagulation.

3. Bleeding disorders
   Hemophilia A.
   Hemophilia B.
   von Willebrand disease.
   Factor XI deficiency.
   Factor V deficiency.
   Factor VII deficiency.
   Deficiencies of fibrinogen.
   Platelet disorders.

4. Pharmacologic – nonpharmacologic interventions
   Anticoagulant agents (structure, mechanism of action, complications, monitoring of the anticoagulant effect).
   Heparin (unfractionated and low molecular weight).
   Heparinoids (danaparoid).
   Warfarin.
   Hirudin.
   Ancrod.
   Antiplatelet agents (mechanism of action, complications).
   Aspirin.
   Dipyridamole
   Ticlopidine.
   Clopidogrel.
   Abciximab.
   Fibrinolytic agents (source, mechanism of action, complications, monitoring of the fibrinolytic effect).
First-generation agents (streptokinase and urokinase).
Second-generation agents (recombinant tissue plasminogen activator and antistreplase).
Third-generation agents (reteplase).
Nonpharmacologic interventions.
Mechanical measures (early ambulation, elastic stockings, electrical calf muscle stimulation, external pneumatic compression).
Vena cava filters.
Pulmonary embolectomy.

References

This article is abroad review presenting in detail the roles of coagulation testing in the management of bleeding and thrombotic disorders. Limitations of coagulation testing in defining the hemostatic state, interpretation of abnormal coagulation test results and the possible relationship to excessive bleeding and thrombosis are thoroughly discussed.


The focus of this article is understanding mechanisms in the hypercoagulable state that enhance and maintain the production of thrombin in circulating blood while preventing its progression to thrombosis. These mechanisms include reactions that produce thrombin from prothrombin, feedback loop mechanisms that affect the rate of thrombin production from prothrombin and the inactivation of thrombin in blood.


This report describes the protein C/protein S pathway, the significance of activated protein C resistance and the factor V Leiden mutation, and the clinical testing used to detect activated protein C resistance and the factor V Leiden mutation. A proposed laboratory testing algorithm is also provided.


This review considers laboratory tests used to evaluate coagulation, including prothrombin time, activated partial thromboplastin time, thrombin time, and platelet count. It discusses hereditary disorders of platelets and/or coagulation proteins that lead to clinical bleeding as well as acquired disorders, including disseminated intravascular coagulation and acquired circulating anticoagulants.

This review discusses pharmacologic therapy of cardiovascular disorders including antiplatelet agents, anticoagulants, thrombolytics, and claudication-alleviating drugs. Each drug category is introduced with a brief review of the current "gold standard" medication, with emphasis on the limitations and weaknesses that the newer agents have been designed to overcome.
20. BLOOD RHEOLOGY AND THE MICROCIRCULATION

Blood rheology
Definitions of rheologic parameters (shear stress, shear rate, viscosity, Newtonian fluid, non-Newtonian fluid, yield stress, Hagen-Poiseuille law, Reynold’s number).
Measurement of viscosity (rotating cylindrical viscometers and the cone on plate viscometers).
Viscosity of plasma.
Viscoelastic properties of erythrocytes and leukocytes.
Viscosity of blood.
Effects of vessel diameter on the viscosity of blood.

Microcirculation
Topographic features.
Measurement of microvascular hemodynamics (microvessel pressures, microvessel blood flow).
Pressure-flow relations in the microcirculation.
Microvessel hematocrit and apparent viscosity.
Blood flow through bifurcations or branch points.
Leukocyte-endothelial cell adhesion.
Capillary blood flow.
Effect of red cell concentration on oxygen transport.
Regulation of blood flow in the microcirculation (local regulation, nervous regulation, humoral and biochemical regulation).
Capillary-lymphatic dynamics, transport and exchange.

References
This article, written by experts in microcirculation, analyses the significance of microcirculation in clinical and molecular medicine. The physiology and pathophysiology of this system are reviewed and future directions in the study of microcirculation are outlined.

In this article, recent instrumentation developments for the study of hemorheology and molecular biology are reviewed. New viscometers for blood viscometry, improved intravital microscope, fluorescence microscopy, digitized video microscopic techniques as well as laser confocal microscopy are represented in terms of recent developments and applications.

This article reviews the involvement of hemorheological and hemostatic mechanisms in thromboatherogenesis and explores the interactions between these factors and the traditional risk factors for atherosclerosis. Elucidation of these mechanisms might lead to new preventive strategies as well as to therapeutic procedures in the management of atherosclerosis and associated thrombotic events.


Blood behaves like a non-Newtonian fluid exhibiting specific features with the probable existence of a plasticity threshold, a viscosity that varies as a function of shear rate and a non-homogeneous nature of the medium during flow. This paper discusses factors affecting blood viscosity and reviews hyperviscosity syndromes ensuing from disorders of these factors.


This article reviews current methods for clinical investigation of the cutaneous microcirculation. These methods are based mainly on laser Doppler and capillary microscopy and, combined with systems for digital image analysis, they allow quantification of the structure of the microvascular bed (quantitative appraisal of microangiopathies) and function (capillary haemodynamics and exchange).
21. DRUGS IN VASCULAR DISEASE

Anticoagulants (mechanism of action, clinical use, complications, monitoring of the anticoagulant effect).
Heparin (unfractionated and low molecular weight).
Heparinoids (danaparoid).
Warfarin.
Hirudin.
Ancrod.

Thrombolytic agents (source, mechanism of action, complications, monitoring of the fibrinolytic effect).
First-generation agents (streptokinase and urokinase).
Second-generation agents (recombinant tissue plasminogen activator and antistreplase).
Third-generation agents (reteplase).

Antiplatelet medications (mechanism of action, complications).
Aspirin.
Dipyridamole
Ticlopidine.
Clopidogrel.
Abciximab.

IV Claudication drugs (mechanism of action, clinical use, complications).
Pentoxifylline.
Dextran.
L-carnitine, L-propionylcarnitine.
Cilostazol.

Serotonergic agents (mechanism of action, clinical use, complications).

Vasodilator drugs (mechanism of action, clinical use, complications).
Direct acting drugs (papaverine, isoxuprine, cyclandelate).
α-adrenergic blockers (guanethidine, phenoxybenzamine, prazosin, tolazoline, phentolamine).
Prostaglandins (PGE, PGI).
β-stimulating drugs (nylidrin).
Calcium channel blockers (nifedipine, verapamil, diltiazem, amlodipine, felodipine, isradipine, nicardipine, nimodipine).
Nitrates (nitroprusside).

References
This review discusses pharmacologic therapy of cardiovascular disorders including antiplatelet agents, anticoagulants, thrombolytics, and claudication-alleviating drugs. Each drug category is introduced with a brief review of the current "gold standard" medication, with emphasis on the limitations and weaknesses that the newer agents have been designed to overcome.


A broad spectrum of issues related to anticoagulation therapy is presented in this article, including initiation and control of anticoagulation therapy, a comparison between unfractionated and low molecular weight heparin, and the management of the "problem patient" who requires anticoagulants.


Several drugs are currently used for patients with intermittent claudication: pentoxifylline, cilostazol, naftidrofuryl, inhibitors of platelet aggregation (including nitric oxide from L-arginine or glyceryl trinitrate), anticoagulants (low molecular weight heparin, defibrotide) and intravenous or oral prostaglandins. The evidence supporting the use of these drugs is summarized by the author and new approaches to the treatment of intermittent claudication, including propionyl-L-carnitine and basic fibroblast growth factor, are outlined.


An overview of pharmacotherapy for peripheral arterial disease is provided by the author of this article. The properties of pentoxifylline and cilostazol are reviewed and new therapeutic opportunities offered by angiogenic growth factors are presented.


This review focuses on risk-factor modification and antiplatelet therapies, as well as strategies for symptomatic relief in patients with peripheral arterial disease. Evaluation of patients with suspected peripheral arterial disease as well as evaluation and treatment of patients with proven peripheral arterial disease are also summarized.
22. SCIENTIFIC BASIS FOR BALLOON EMBOLECTOMY.

Mechanics of balloon embolectomy
Lateral wall pressure.
Balloon pressure versus lateral wall pressure.
Balloon-artery shear forces.
Histologic effects of embolectomy.

Determinants of lateral wall pressure and shear forces
Catheter size.
Brands of catheters.
Balloon eccentricity.
Fluid-filled versus gas-filled balloons.
Syringe size.
Velocity of catheter motion.
Blood in the vessel lumen.
Inflating balloons at rest and during catheter motion.

Balloon embolectomy induced injuries

References

This article reviews the spectrum of clinical injuries produced by balloon embolectomy. The concepts of lateral wall pressure and balloon-artery shear force are presented, and the histologic reactions to passage of embolectomy catheters are described. On the basis of the results of experimental investigations, technical recommendations are made regarding the performance of embolectomy in patients.


The aim of this experimental study was to evaluate the character and time course of arterial injury caused by balloon embolectomy catheters. Shear forces of up to 30 gm caused no injury, while shear forces of 60-120 gm caused stripping of the endothelium which was completely repaired by myointimal proliferation within 6 months. Two hundred gram initial force caused intimal injury and fracturing of the internal elastic lamina, with the latter injury persisting even after 6 months.


This study was undertaken to compare balloon eccentricity in air with that which occurs in arteries, to determine the influence of balloon eccentricity on shear force, and to estimate the injury potential of eccentric balloons. The presented data suggest that
balloon eccentricity in air is an accurate indicator of balloon eccentricity within arteries, that moderately eccentric balloons are acceptable for clinical use, but that extremely eccentric balloons may cause severe injury and should not be used in the operating room.


_Balloon embolectomy catheters were studied in canine common carotid arteries (2 to 3 mm) in vitro to evaluate a technique of preventing excessive shear forces and to examine the effect of blood within the lumen. From these studies it is recommended that during embolectomy in patients the balloons be distended during the first half centimeter or centimeter of catheter withdrawal to prevent excessive shear forces and that residual blood in the vessel lumen proximal to the point of embolic obstruction be accepted without concern, provided adequate heparinization has been achieved._


_This article presents data from balloon embolectomy experiments in 2 to 3 mm canine arteries in vitro. These data suggest that every effort should be made to achieve the low LWP since this strongly influences shear force; the smallest effective catheter should be used and that negligible benefit may be gained if catheters are withdrawn at moderate velocities._
23. BASIC PRINCIPLES UNDERLYING THE FUNCTION OF ENDOVASCULAR DEVICES.

Transluminal balloon angioplasty
Forces of transluminal vascular dilatation.
Mechanism of transluminal dilatation.
Characteristics of balloon construction (catheter pushability, trackability, crossability, balloon compliance).
Characteristics of catheter design.
Pathophysiology of the complications of transluminal dilation.

Endoluminal stents
Characteristics of balloon expandable stents.
Characteristics of self-expanding stents.
Coated stents.
Drug-eluting stents.
Parameters of proper stent deployment.
Complications of stent deployment.

Atherectomy
Characteristics of the Kensey catheter.
Characteristics of the Auth rotoblator.
Characteristics of the Simpson atherectomy catheter.
Characteristics of the transluminal extraction catheter.

Laser angioplasty
Laser physics.
Principles of dosimetry.
Characteristics of optical fibers.
Laser effects on target tissue (photothermal, photochemical, photoacoustic, nonlinear).
Specific laser systems (Nd:YAG laser, argon laser, excimer laser, CO\textsubscript{2} laser).

Intravascular ultrasound
Basic properties of sound.
Instrumentation of intravascular ultrasound.
Practical application of ultrasound.

Angioscopy
Principles of light transmission.
The angioscopic system.

References
Basic principles underlying the function of endovascular devices are discussed in these sections, including guidewires, catheters and sheaths, balloon angioplasty, peripheral atherectomy, vascular stents, thrombectomy catheters, endovascular grafting, angioscopy and intravascular ultrasonography.


An overview of endovascular interventions for the treatment of lower-extremity atherosclerotic disease is presented in this article. The indications, and results of balloon angioplasty, endoluminal stenting and transluminal atherectomy are discussed.


This article presents the unique features of four atherectomy devices: Simpson AtheroCath, Transluminal Extraction Catheter (TEC), Trac-Wright Catheter, and Auth Rotablator. The results, complications, and limitations reported by clinical investigators are discussed critically and realistically.


This review describes the rationale, technique, and interpretation of IVUS imaging in diagnostic and therapeutic applications. Special emphasis is placed on the impact of ultrasound in understanding atherosclerotic coronary disease and its management.


The diagnostic and therapeutic applications (angioscopically guided luminal intervention) of angioscopy are reviewed in this article, including advantages, indications and technical considerations.
24. VASCULAR GRAFTS

Vascular graft interfacial histology
Protein absorption.
Platelet adhesion.
Neutrophil infiltration.
Monocyte recruitment.
Endothelial cell and smooth muscle cell ingrowth.

Mechanisms of vascular graft healing
The role of platelets.
The role of macrophages.
The role of endothelial cells and smooth muscle cells.

Characteristics of grafts
Composition.
Porosity.
Durability.
Flexibility.
Compliance.

Modes of graft failure
Thrombogenicity.
Anastomotic pseudointimal hyperplasia.

Current vascular grafts
Aortic grafts (knitted Dacron grafts coated with albumin, gelatin or collagen, ePTFE grafts: characteristics, advantages, disadvantages).
Femoral-popliteal/tibial grafts (autogenous saphenous vein, ePTFE grafts, Dacron grafts, glutaraldehyde-stabilized human umbilical vein graft, homologous vein: characteristics, advantages, disadvantages).

Experimental biohybrid prostheses
Synthetic materials impregnated with antimicrobial agents.
Anticoagulant substances affixed to synthetic graft surfaces.
Synthetic grafts impregnated with growth factors.

Bioresorbable synthetic grafts
Bioresorbable grafts (polyglycolic acid, polygactin 910, polydioxanone grafts).
Grafts of compound yarns containing both resorbable (polyglycolic acid, polygactin 910, polydioxanone) and a nonresorbable (Dacron or polypropylene) material.

References
The healing of prosthetic arterial grafts in animals and in humans is described in this paper. New strategies and approaches, such as endothelial cell seeding, that have recently been attempted to improve the patency of synthetic vascular grafts are also outlined.


The importance of mechanical and hemodynamic factors for the development of intimal hyperplasia at the graft anastomotic site is analyzed in this article. Disturbed flow at the anastomosis leading to fluctuations in shear stress at the endothelium (a known cause of intimal hyperplasia in normal arteries), injury due to suturing and stress concentration at the anastomosis are explained in detail with equations, graphs and schematic representations.


In this overview article, the strategies used to improve the patency of these small-diameter grafts, the current status in clinical trials, and further perspectives in the field of artificial vascular graft development are reviewed. It is concluded that, in view of recent developments in tissue engineering approaches, the future of small-diameter vascular prostheses looks promising.


Biomechanical engineering approaches can be used to reduce tensile stress and strain due to exposure to arterial blood pressure and to prevent eddy blood flow in vein grafts. In this article, the background, principles, clinical potentials, as well as the limitations of vascular biomechanical engineering are discussed.


Tissue engineering, using either polymer or biological based scaffolds, represents the newest approach to overcoming limitations of small diameter prosthetic vascular grafts. This current review represents an overview about previous and contemporary studies in the field of artificial vascular conduits development regarding arterial and venous autografts, allografts, xenografts, alloplastic prostheses, and tissue engineering.
25. STATISTICS FOR THE VASCULAR SURGEON

Fundamental concepts
Sample versus population.
Random sampling.
Descriptive versus inferential.

Descriptive statistics
Data collection (nominal scale, ordinal scale, interval scale, ratio scale).
Frequency distribution, histogram, frequency polygon.
Location: measures of central tendency (arithmetic mean, median, mode), quartiles, deciles and percentiles.
Measures of variability and spread (minimum and maximum and range, variance, standard deviation).
Distribution curves (normal distribution, bimodal distribution), Kurtosis, outlier, skewness.

Inferential statistics
Standard error of mean, confidence intervals.
Significance tests, hypothesis testing, error of hypothesis testing, statistical power and sample size, p value, one-tail versus two-tail test).
Inference on means (student’s t-test, comparison of means in paired and unpaired samples).
Inference on proportions (chi-square test, Fisher’s exact test)

Regression and correlation
Nonparametric methods or distribution-free methods
Sighed rank test (Wilcoxon).
Wilcoxon rank sum test.
Mann-Whitney U test.
Kruskal-Wallis test.

Life-table analysis
Kaplan-Meier curve.
Log rank test or Mantel-Haenszel test.

Meta-analysis (goals, pitfalls).

Evaluation of a new diagnostic test
Reliability.
Sensitivity, specificity, positive predictive value, negative predictive value, overall accuracy.
Receiver operating characteristic curve.

References
26. ANEURYSMAL DISEASE OF THE ABDOMINAL AORTA

Histological changes
Extracellular matrix (collagen, elastin).
Cellular components – inflammatory infiltrates.

Genetics

Experimental models
Spontaneous animal models.
Pharmacologic models.
Dietary models.
Surgical models.

Causes of aneurysms
The role of proteases (elastases, collagenases, plasmin, matrix metalloproteinases) and proteases inhibitors (α-1 antitrypsin, tissue inhibitor of metalloproteinases).
The role of inflammation.
The role of autoimmunity.
The role of atherosclerosis.
The role of hemodynamics.

V. Potential for intervention based on pathophysiology.

References

This article gives an overview of research data on the genetic background of AAA. Based on the familial clustering of the AAA, reported in 11-19% of AAA patients, a gene mutation in one of the structural proteins of the connective tissue is expected. However, no specific genetic factor responsible for familial AAA has been identified yet.


This article discusses animal models and experimental techniques that have been described in the investigation of the pathophysiology of AAA and in the development of improved endovascular surgical and pharmacological therapies. The advantages of these models and some of the problems encountered in extrapolating experimental findings to the human condition are also discussed.

Recent progress in our understanding of the pathogenesis of aneurysmal disease is summarized in this article. The role of immunology, biochemistry, cell biology, and genetic issues is reviewed with special emphasis on the role of the local inflammatory infiltrates and the destructive proteolytic enzymes.


This article reviews current knowledge on the pathogenesis of abdominal aortic aneurysm. The role of atherosclerosis, inflammation, matrix changes and proteolysis is specifically addressed.


The finding that elastolytic MMPs, particularly MMP-9 and MMP-2, are expressed and produced in increased amounts in human aneurysm tissue, has led to the possibility that these enzymes might serve as rational targets for pharmacotherapy in this disease. The role of matrix metalloproteinases in abdominal aortic aneurysm disease as well as the therapeutic implications of this role are outlined in this review.
27. CEREBRAL BLOOD FLOW

Regulation of cerebral blood flow
Mechanical effects on cerebral blood flow.
Neurogenic coupling mechanisms in cerebral blood flow.
Local effects on cerebral blood flow.

Methods for evaluating cerebral blood flow
Experimental methods (pial artery diameter, hydrogen diffusion, radioactive microspheres, laser Doppler).
Clinical methods (Duplex ultrasound, transcranial ultrasound, xenon clearance, single photon emission cerebral tomography, magnetic resonance angiography, magnetic resonance spectroscopy, perfusion/diffusion MRI, positron emission tomography).

Clinical research areas in cerebral blood flow
Pharmacologic effects on cerebral blood flow.
Effects of anesthesia on autoregulation.
Neonatal cerebral blood flow.
Cerebral blood flow in altitude sickness.
Cerebral blood flow following cardiac arrest.
Head trauma and cerebral blood flow.
Hemodynamics of atherosclerotic cerebrovascular disease.

References

Cerebral blood flow is largely independent of perfusion pressure when autoregulation is intact. The mechanisms of cerebral autoregulation are reviewed in this paper, including the role of local-chemical factors, endothelial factors, autacoids, and transmitters from perivascular nerves.


This article reviews the concepts of cerebral blood flow for the clinician involved in the management of patients with carotid stenosis and/or ischaemic stroke. Methods of assessing cerebral blood flow in vivo using nuclear medicine, magnetic resonance and X-ray computed tomography are described. Applications of magnetic resonance and X-ray computed tomographic methods are reviewed and illustrated by examples from the authors' radiological practice.

The effects of isoflurane or halothane on cerebral blood flow (CBF) reactivity to changes in arterial carbon dioxide tension during carotid endarterectomy were compared using the intravenous method of 133Xe-CBF determination. It is concluded that there is no significant difference between halothane and isoflurane in their effects on CO2 reactivity in the mildly hypocapnic to normocapnic range.


Although vascular damage is a key event, it remains a somewhat neglected component to the underlying degenerative processes that evolve following injury to the brain. The present review integrates the current knowledge of the vascular events proceeding injury to the brain, with an emphasis on how this impacts the control of vascular function and thus cerebral blood flow.


This article reviews the responses of the cerebral vasculature to reduced perfusion pressure, examines their association with stroke risk, presents various methods of hemodynamic assessment and discusses their clinical applications.
28. BASIC SCIENCE OF RENOVASCULAR HYPERTENSION

1. Anatomy of the renal vasculature
   Arterial anatomy.
   Venous anatomy.

2. Physiology of the renin-angiotensin system
   Angiotensin peptides and the nephron.
   Angiotensin peptides and the cardiovascular system.
   Angiotensin peptides and the central nervous system.
   Angiotensin peptides and the adrenals.

3. Mechanisms of renal autoregulation
   The myogenic mechanism and angiotensin peptides.
   Tubuloglomerular feedback and angiotensin peptides.
   Vascular endothelial substances.
   Renal nerves and function regulation.

4. Pathologic considerations
   Atherosclerosis (etiology, pathology, pathophysiology).
   Fibromuscular dysplasia (etiology, pathology, pathophysiology).
   Developmental lesions (etiology, pathology).

5. Diagnostic studies (performance, diagnostic criteria, application and limitations, accuracy).
   Screening studies for renovascular occlusive disease (rapid sequence excretory pyelogram, peripheral plasma renin assays, renal Duplex ultrasonography, renal arteriography).
   Functional studies (isotope renography, renal vein renin assays, split renal function studies).

References

This article reviews the pathophysiology of renin release in renovascular hypertension, including stimuli and mechanisms of release as well as factors modifying renin release.


Autoregulation of the renal vasculature provides a mechanism by which renal function is maintained relatively constant despite variations in systemic blood pressure. Alterations in the autoregulatory response can have clinical consequences such as hypertension, hypertension-induced renal injury and increase in the serum creatinine concentration. The consequences of impaired renal autoregulation are discussed in this review.

Fibromuscular dysplasia is an important cause of renovascular hypertension in young, predominantly female patients. This article presents the main characteristics of fibromuscular dysplasia, including pathologic classification, etiology, clinical manifestations, differential diagnosis and treatment.


This paper presents diagnostic imaging modalities of renovascular hypertension including captopril scintigraphy, angiography, Doppler sonography and MR angiography. The indications, advantages and disadvantages of each technique are discussed.


Available tests for the diagnosis of renovascular hypertension can be divided into those that identify the functional consequences of a renal artery obstruction (angiotensin-converting enzyme inhibitor-augmented renography) and those that identify the anatomic presence of stenosis (duplex ultrasonography, magnetic resonance angiography, and contrast tomography angiography). After reviewing current evidence regarding the use of these techniques, the authors present a potential treatment algorithm.
29. BASIC MECHANISMS IN MESENTERIC ISCHEMIA

Anatomy
Normal arterial and venous anatomy of the mesenteric circulation.
Collateral circulation.
More frequently encountered anatomic variations.

Regulation of mesenteric blood flow
Intrinsic control of the mesenteric circulation (metabolic theory, myogenic theory).
Reactive and postprandial hyperemia.
Extrinsic control of the mesenteric circulation (neural mechanisms, hormonal mechanisms).

Intestinal ischemia
Histologic injury.
Biochemical – metabolic events.

Reperfusion injury
Formation of reactive oxygen species (redox reactions, enzyme-substrate reactions, activation/degranulation of inflammatory cells).
Mechanisms of reactive oxygen species cell and tissue injury.
Polymorphonuclear leukocytes in reperfusion injury.

V. New approaches to diagnosis and treatment of mesenteric ischemia/reperfusion.
1. Recent laboratory efforts in quantifying the effects of mesenteric ischemia-reperfusion (ICAM-1, ELAM-1, alkaline phosphatase).
2. New approaches to treatment of mesenteric ischemia/reperfusion (changes in the nature of the reperfusate, alterations in the adherence or activation of polymorphonuclear cells and the administration of pharmacologic scavengers of reactive oxygen species).

References


This article reviews the angiographic appearance of the major visceral arteries, the more common variants, their embryologic origins, and some of the most common sources of collateral flow. A brief review of the physiology of the mesenteric circulation is also provided, including a discussion of the intrinsic and extrinsic mechanisms of splanchnic blood flow control.


This review summarizes the current understanding regarding the regulatory mechanisms of intestinal blood flow in fasted and fed conditions and during pathological stress. The role of absorbed nutrients, enteric nervous system effects and reflexes, gastrointestinal
hormones and peptides and local nonmetabolic and metabolic vasoactive mediators is discussed. Alterations of intestinal blood flow in pathologic conditions, including septic shock, hemorrhagic shock, cardiogenic shock and portal hypertension are also described.


Intestinal ischemia can result from a host of pathophysiologic disturbances and, in turn, may produce a variety of adverse local and systemic consequences. Mechanisms of ischemic injury and the central role of vasoconstriction are discussed.


In this review the physiology of the intestinal circulation is briefly outlined, followed by a discussion of nonocclusive intestinal ischemia and reperfusion injury. The clinical causes, diagnostic process and therapeutic options of intestinal ischemia are also outlined.


The various diagnostic tests for intestinal ischemia are presented in this study. These include serum biochemical markers, peritoneal fluid analysis, tonometry, radionuclide imaging, laparoscopy, and endoscopic techniques. Newer techniques, including radionuclide-labeled antibodies, tonometry, and reflectance spectrophotometry, are also discussed.
30. HEMODYNAMIC BASIS OF PORTAL HYPERTENSION

Anatomy
Anatomy of the liver.
Anatomy of the portal circulation.
Anatomy of the hepatic arterial circulation.
Anatomy of the collateral circulation.

Regulation of hepatic blood flow
Regulation of the portal circulation.
Regulation of the hepatic arterial blood flow (intrinsic, extrinsic).
Hepatic artery:portal vein interactions.

Pathophysiology of portal hypertension
Increased intrahepatic resistance (pathology, pathophysiologic sequel). 
Hyperdynamic circulation (definition, mechanisms, pathophysiologic sequel). 
the role of increased circulating vasodilators (glucagon, prostacyclin, NO). 
the role of reduced response to endogenous vasoconstrictors. 
The role of the sympathetic nervous system. 
The role of plasma volume.

Extrahepatic responses to portal hypertension
Cardiac and systemic hemodynamics. 
Gastrointestinal effects.

References

The liver microvascular architecture is described in detail in this paper. The implications of the microvascular structure for hepatic hemodynamics and portal hypertension are also discussed.


The structural, cellular, and humoral factors involved in the regulation of sinusoidal blood flow in normal and injured liver are reviewed in this article. The role of the stellate cells and the modulation of their function by the endothelin and NO systems is discussed. These systems represent potential targets for gene therapy.

This article presents an overview of the hyperdynamic circulation in cirrhosis. Based on the available data, the authors propose a two-phase pathogenesis of the hyperdynamic circulation of cirrhosis. Initially, passive vascular relaxation, and the resulting hyperdynamics, appear to be secondary to blood volume expansion. In the second, much more complex phase, active vasodilatation, associated with hyporesponsiveness to vasoconstrictors, especially in the splanchnic bed, increased portosystemic shunting, and the development of new vessels by angiogenesis likely are part of the explanation.


An overview of portal hypertensive gastropathy is provided in this article, including its pathogenesis, diagnosis, clinical presentation and treatment.


Patients with cirrhosis and portal hypertension exhibit characteristic hemodynamic changes with hyperkinetic systemic circulation, abnormal distribution of blood volume and neurohumoral dysregulation. The pathophysiologic aspects of these disorders are discussed in this article.
31. ANATOMY AND PHYSIOLOGY OF NORMAL ERECTION

Anatomy
Arterial anatomy.
Venous drainage.
Penile innervation.

Hemodynamics of normal erection

Causes of impotence
Cavernosal malfunction.
Venous or cavernosal leakage.
Arteriogenic impotence.

Diagnostic methods
Noninvasive sequence (penile brachial blood pressure index, penile plethysmographic pulse recording, pudental-evoked potentials, bulbo-cavernosal reflex time).
Artificial erection.
Dynamic infusion cavernosometry and cavernosography.
Ultrasonography.
Nocturnal penile tumescence.

Treatment of vasculogenic impotence
Drug therapy.
Small vessel reconstruction.
Venous interruption.
Prosthetics.

References

The anatomy of the penile vasculature and the physiology of erection are reviewed in this paper. Risk factors for vasculogenic erectile dysfunction are also briefly discussed.


At present, there are two major views regarding the pathophysiology of erectile dysfunction. In the first hypothesis, the oxygen tension-dependent changes in the penis during erection are proposed to impact corpus cavernosum structure by altering smooth muscle metabolism and connective tissue synthesis. The alternate hypothesis proposes that ED is the result of a metabolic imbalance between relaxatory and contractile processes within the trabecular smooth muscle such that contractile processes
predominate. In this review of the pathophysiology of ED, each hypothesis is examined and a synthesis devised incorporating both views.


This article focuses on the main biochemical events leading to penile erection and detumescence as well as on the potential manipulation of these events for therapeutic purposes. The role of nitric oxide, cGMP, cAMP and phosphodiesterases is analyzed and potential pharmacologic interventions including papaverine, sildenafil and yohimbine are discussed.


The most commonly utilized diagnostic tests for erectile dysfunction are outlined in this monograph. These tests include nocturnal penile tumescence studies, somatosensory evoked potentials, bulbocavernosus reflex latency, corporal cavernosal smooth muscle electrical activity, penile plethysmography, penile blood pressures, penile brachial index, selective internal pudendal pharmacoangiography, Doppler sonography, dynamic infusion cavernosometry/cavernosography, nuclear washout radiography, and color duplex Doppler ultrasound.


This review considers current and past results of vascular surgery in men with impotence failing to respond to medical treatment. Guidelines for case selection for vascular interventions as well as reporting criteria are suggested.
SKELETAL MUSCLE ISCHEMIA AND REPERFUSION: MECHANISMS OF INJURY AND INTERVENTION

Skeletal muscle energy metabolism
Normal skeletal muscle energy metabolism.
Skeletal muscle energy metabolism during ischemia.
Skeletal muscle energy metabolism during reperfusion.

Tissue injury in skeletal muscle ischemia/reperfusion
Histology.
Methods of assessment.

Pathophysiology of skeletal muscle ischemia/reperfusion
Endogenous free radical production by postischemic endothelial cells.
Recruitment and activation of neutrophils wielding free radicals and lytic enzymes (including the role of complement, arachidonic acid metabolites, platelet activating factor, cytokines, cell adhesion molecules).
Occlusion of microvascular beds secondary to endothelial cell swelling, perivascular tissue edema, failed endothelium-dependent vessel relaxation, adherence of activated neutrophils and microvascular thrombosis).

Interventional outline
Metabolic salvage.
Inhibition of free radical production during reperfusion.
Graded reoxygenation during reperfusion.
Leukopenic reperfusion.
Hypothermia during reperfusion.
Fibrinolysis, anticoagulation.
References

This article analyzes the local and systemic consequences of severe ischemia and reperfusion of the skeletal muscle. The mechanisms responsible for reperfusion injury as well as methods of prevention and treatment are also discussed.


This article reviews the pivotal role of endothelium-leukocyte interactions and of cytokines in the genesis of postischemic damage in muscle. Clinical considerations and future directions based on research and practice are presented.

This article describes the cytologic and biochemical responses of skeletal muscle to ischemia and reperfusion injury. It is also discussed how an endogenous protective mechanism, ischemic preconditioning, may be exploited to limit postischemic skeletal muscle injury.


This article reviews the mechanisms involved in the pathogenesis of skeletal muscle ischemia-reperfusion injury including oxidant generation, elaboration of proinflammatory mediators, infiltration of leukocytes, Ca\textsuperscript{2+} overload, phospholipid peroxidation and depletion, impaired nitric oxide metabolism, and reduced ATP production. Based on these mechanisms, rational intervention strategies may be proposed and implemented as potential treatments for skeletal muscle dysfunction associated with ischemia-reperfusion.


This article provides a concise review on the potential causes of ischemia-induced reperfusion injury and pharmacologic intervention in the skeletal muscle. The mechanism of ischemic preconditioning and its clinical applications for augmentation of skeletal muscle tolerance to prolonged ischemic insult are also discussed.
33. **SPINAL CORD ISCHEMIA ASSOCIATED WITH HIGH AORTIC CLAMPING: METHODS OF PROTECTION**

1. Anatomy of the blood supply to the spinal cord

2. **Pathophysiology of spinal cord ischemia and reperfusion**
   - Hemodynamic changes.
   - Ischemic injury.
   - Reperfusion injury.
   - Delayed onset paraplegia.

3. **Methods of protection**
   - Experimental results of spinal cord protection (including the role of cerebrospinal fluid drainage, systemic hypothermia, hypothermic perfusion, regional cooling, barbiturates, superoxide dismutase, calcium channel blockers, prostaglandins, papaverine, MK-801, monoclonal antibodies, fluosol-DA, opiate antagonists, aminosteroids).
   - Clinical results of spinal cord protection (including the role of aortic cross-clamp time, reimplantation of intercostals arteries, bypass or shunt, evoked potentials, spinal fluid drainage, hypothermia).

**References**

This review discusses the significance and management of delayed-onset neurologic deficit. The pathophysiology of delayed-onset neurologic deficit after thoracoabdominal aortic aneurysm repair, the various factors known to increase the risk of spinal cord ischemia, as well as the different intraoperative adjuncts to improve spinal cord protection are presented.


The authors present a method for providing regional cord hypothermia with epidural cooling during TAA repair. Technical considerations with epidural cooling and the clinical results obtained in their experience are discussed.


The purpose of this randomized clinical trial was to evaluate the impact of cerebrospinal fluid drainage (CSFD) on the incidence of spinal cord injury after extensive thoracoabdominal aortic aneurysm (TAAA) repair. Overall, CSFD resulted in an 80% reduction in the relative risk of postoperative deficits. The authors conclude that perioperative CSFD reduces the rate of paraplegia after repair of extent I and II TAAAs.
In this article, the literature on pharmacological neuroprotection in experimental SCI is systematically reviewed to assess the neuroprotective efficacy of the various agents. The results suggest that numerous agents may protect the spinal cord from transient ischemia. However, poor temperature management and lack of statistical power severely weakened the evidence. The authors conclude that clinical evaluation of pharmacological neuroprotection in surgical procedures that carry a risk of ischemic spinal cord damage is not justified on the basis of this analysis.


After a brief discussion of the etiology of spinal cord ischemia, the authors present several intraoperative interventions and strategies, which address the multifactorial nature of cord injury. The role of adequate distal aortic perfusion, cerebrospinal fluid drainage, pharmacological agents such as papaverine and steroids, as well as the role of circulatory arrest and profound hypothermia are analyzed.
34. ARTERIOVENOUS HEMODIALYSIS ACCESS

Anatomy
Snuff-box fistula.
Brescia-Cimino fistula.
Radial artery to antecubital vein (straight graft).
Radial artery to basilic vein-above elbow (straight graft).
Brachial artery-below elbow to antecubital vein (loop graft).
Brachial artery-below elbow to basilic vein-above elbow (loop graft).
Brachial artery-above elbow to axillary vein (C-shaped graft).
Basilic transposition.

Physiology
Resistance.
Velocity and volume.
Flow patterns.
Energy changes.

Pathophysiology
Local hemodynamics/pathophysiology (hemodynamic and structural changes in the proximal artery, the distal artery, the proximal vein and the distal vein).
Systemic hemodynamics/pathophysiology (determinants and consequences of the drop in total peripheral resistance).

Pathogenesis of complications
Thrombosis.
Infection.
Steal syndrome.
Pseudoaneurysm formation.
Venous hypertension.

References

Venous stenosis and thrombosis as a result of venous neointimal hyperplasia are the major causes of hemodialysis vascular access dysfunction. This review describes the lesion of venous neointimal hyperplasia in human samples and in a pig model and suggests possible future directions for the development of effective local therapies for this condition.

Potential fistula-related problems which may impact on patient survival include high fistula flow with hyperkinetic circulation and cardiac failure, low fistula flow with the risks of underdialysis and fistula thrombosis, vascular access infection with local or systemic manifestations, and possibly induction and maintenance of a microinflammatory state. All of these problems are briefly reviewed in this article.


The effects of diameters of canine femoral arteriovenous fistulas upon regional and central hemodynamics were determined to correlate fistula size with fistula flow, as well as changes in cardiac output, reversal of distal arterial flow, and distal venous hypertension. The study shows that there is a direct correlation between fistula size and cardiac output, a direct correlation between fistula size and venous hypertension and an inverse relation between fistula size and distal femoral artery flow and pressure.


This article describes the means of accessing the circulation for hemodialysis, the pathogenesis of access failure through progressive stenosis followed by thrombosis, methods of detecting access dysfunction before thrombosis, and therapeutic options. Although angiographic or surgical intervention remain the mainstays of management, medical treatments to decrease stenosis and delay thrombosis are currently under investigation.


Physiologic and hemodynamic issues related to the construction of a vascular access for hemodialysis are reviewed in this section. These issues provide the basis for the understanding, prevention and treatment of vascular access complications.
35. ARTERIAL AND VASCULAR GRAFT INFECTION

Etiology
Bacterial contamination at the time of graft implantation.
Hematogenic contamination.
Lymphogenic contamination.

Microbiology and immunology
Most commonly involved organisms.
The effect of graft material.
The effect of systemic and local immunosuppression.

Clinical presentation
Depending on the anatomic location.
Depending on the type of graft.
Depending on the infecting organism.

Diagnostic evaluation
Duplex ultrasonography.
Computed tomography.
Magnetic resonance imaging.
Nuclear medicine techniques.
Arteriography.
Endoscopy.

Prevention
Asepsis – antisepsis.
The role of prophylactic antibiotics.
Antibiotics bonded to the vascular graft.

Management
Aortic graft infection.
Femoropopliteal graft infection.

References

Hemorrhage, early thrombosis of a graft or vessel nerve injury, graft infection, and renal failure are frustrating problems for vascular surgeons. All frequently arise from technical complications. Methods of avoiding these problems are discussed.


This article addresses the problem of methicillin-resistant Staphylococcus aureus (MRSA) which is now the commonest cause of serious vascular wound and graft
infection. Preoperative, intraoperative and postoperative measures against MRSA infections are briefly reviewed.

Valentine RJ. Diagnosis and management of aortic graft infection. Semin Vasc Surg 2001;14:292-301.

This report summarizes the currently available methods of diagnosing and treating aortic graft infections. Available imaging techniques and complementary tests are presented, followed by a discussion of the therapeutic options which include graft excision and extra-anatomic revascularization or in situ replacement with autogenous vein, allograft or rifampin-bonded prosthesis.


This review discusses diagnosis and treatment of patients with prosthetic vascular graft infection, emphasizing the basic principles necessary for successful management of this complex problem. Basic information about the incidence, etiology, and bacteriology of prosthetic vascular graft infections is also be briefly reviewed.


This article summarizes the indications and efficacy of antibiotic prophylaxis and treatment in vascular surgery. Well-established indications, as well as new applications for antibiotics in vascular surgery are discussed.
36. NEUROPATHIC AND BIOMECHANICAL ETIOLOGY OF FOOT ULCERATION IN DIABETICS

1. Classification of diabetic neuropathy
   Mononeuropathies (isolated and multiple, cranial mononeuropathies).
   Polyneuropathies (diabetic sensory polyneuropathy, proximal motor polyneuropathy, autonomic neuropathies).

2. Pathogenesis of diabetic neuropathy
   Sorbitol accumulation.
   The activated polyol pathway theory.
   Changes in perineural and endoneurial vasculature and resultant ischemia of the nerve.
   Hyperglycemia-related nonenzymatic glycosylation of the vasoneurosum and the endoneural matrix.
   The role of insulin.

3. Foot changes as they relate to neuropathy
   Consequences of sensory dysfunction.
   Consequences of motor dysfunction.
   Consequences of autonomic dysfunction.

4. Foot biomechanics
   Normal gait (stance and swing phases).
   Abnormal foot biomechanics.
   Charcot deformity.
   Biomechanical changes caused by segmental amputations of the foot (hallux amputation, toe amputation, transmetatarsal ray amputations, Lisfranc and Chopart amputations).

5. Pathophysiology of diabetic foot ulceration
   The role of the loss of protective sensation.
   The role of the structural deformity of the foot.

References

The pathogenesis of diabetic neuropathy is multifactorial. There is increasing evidence to link abnormalities in the polyol pathway to the pathogenesis of diabetic neuropathy. In addition, there appear to be abnormalities of nerve regeneration and of sodium and calcium channels. Aldose reductase inhibitors, neurotrophic factors and vascular endothelial growth factor have shown promise for reversing neuropathy. Lamotrigine and bupropion represent new treatments for neuropathic pain. All this new information about the pathogenesis and treatment of diabetic neuropathy is summarized in this article.

This article addresses the many synergistic factors that cause both ulceration and neuroarthropathy. These include dramatic alterations in all components of the peripheral nerves, the mechanical characteristics of bones and soft tissues, gait kinematics, the vasculature at both a microscopic and a macroscopic level, the immune system, and the fundamental processes of wound healing.


Biomechanical issues are now widely recognized as being important in the treatment of diabetic foot disease. This article summarizes recent advances in the understanding of the association between foot deformity and plantar pressure, the measurement of shear stress and the importance of neuropathy and callus in the pathogenesis of ulceration. Recent data on the biomechanical evaluation of surgery as well as the efficacy of unloading devices is also presented.


The underlying pathophysiology and treatment of diabetic foot ulcers, infections, and the diabetic Charcot foot are thoroughly reviewed. Based on currently available evidence, the authors present a Clinical Practice Guideline for diabetic foot disorders.


Neuropathy and ischemia, two common complications of diabetes mellitus, are the primary underlying risk factors for the development of foot ulcers and their complications. However, an initiating factor, such as physical or mechanical stress, is also required for an ulcer to develop. In addition to increasing the risk of ulceration, diabetes mellitus also increases the risk of infection by impairing the body's ability to eliminate bacteria. The complex processes by which diabetic ulcers develop are reviewed in this article.
CLINICAL CURRICULUM AND EDUCATIONAL OBJECTIVES
FOR VASCULAR SURGERY

Developed by the Association of Program Directors in Vascular Surgery

James M. Seeger, M.D.,
Chairman of the Clinical Curriculum Committee

General:
Each of the categories in the Clinical Curriculum is assumed to include the diagnosis and management of the problem for all etiologies to include atherosclerosis, trauma, infection, etc. where appropriate. A general understanding of each topic in the Clinical Curriculum is expected at the completion of vascular surgery training. In addition, the trainee is expected to know the natural history of the various diseases. Knowledge of additional/non-core topics will be encouraged but not required.

Educational objectives have also been developed for each section of the Clinical Curriculum. It is expected that these objectives will be achieved by each trainee at the completion of training. Included are selected references for each set of objectives that are suggested as minimal background reading for each section.
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Clinical Curriculum for Vascular Surgery

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1. Diagnosis and Management of Aneurysmal Disease

Includes:

- Aortic and Iliac Artery Aneurysms
- Peripheral Artery Aneurysms
- Extra-cranial Carotid Aneurysms
- Subclavian/Axillary Artery Aneurysms
- Femoral/Popliteal Artery Aneurysms
- Splanchnic and Renal Artery Aneurysms
- Thoracoabdominal Aortic Aneurysms
- Thoracic Aortic Aneurysms
- Thoracic/Abdominal Aortic Dissection

2. Diagnosis and Management of Extremity Arterial Occlusive Disease

Includes:

- Aortoiliac Occlusive Disease
- Femoral-Popliteal-Tibial Occlusive Disease
- Upper Extremity Occlusive Disease
- Combined Aortoiliac and Infrainguinal Occlusive Disease
- Arterial Bypass Graft Surveillance
- Failing Arterial Bypass Graft
- Ischemic Foot Lesions

3. Diagnosis and Management of Renal Artery Occlusive Disease

Includes:

- Renovascular Hypertension
- Ischemic Nephropathy
- Renal Artery Surgery
- Renal Angioplasty
- Diagnostic Studies to Detect Functionally Significant Renal Artery Stenosis

Additional Important/None-Core Curriculum Topics:

- Renal Arteriovenous Fistulae

4. Diagnosis and Management of Visceral Ischemia

Includes:

- Chronic Visceral Ischemia
Acute Visceral Ischemia  
Non-Occlusive Mesenteric Ischemia  
Mesenteric Venous Occlusive Disease

Additional Important/Non-Core Curriculum Topics:

Celiac/SMA Compression

5. **Diagnosis and Management of Carotid Artery Occlusive Disease**

Includes:

- Atherosclerotic Carotis Artery Disease
- Carotid Artery Fibromuscular Dysplasia
- Carotid Artery Coils and Kinks
- Carotid Artery Radiation Injury
- Carotid Body Tumor
- Overall Management of Stroke
- Spontaneous Carotid Artery Dissection
- Atherosclerotic Aortic Arch Disease Leading to Proximal Carotid Artery Stenosis

6. **Diagnosis and Management of Innominate, Subclavian and Vertebrobasilar Arterial Disease**

Includes:

- Stenotic and Embolic Innominate Artery Disease
- Stenotic and Embolic Vertebral Artery Disease
- Stenotic and Embolic Subclavian Artery Disease
- Subclavian Steal Syndrome

Additional Important/Non-Core Curriculum Topics:

- Vertebral Arteriovenous Fistulae

7. **Diagnosis and Management of Thoracic Outlet Syndrome**

Includes:

- Cervical Rib/Abnormal First Rib
- Arterial Complications
- Venous Complications
- Neurogenic Complications

8. **Diagnosis and Management of Acute Arterial Occlusion**

Includes:

- Acute Thrombotic Disease
9. **Diagnosis and Management of Diabetic Foot Problems**

Includes:

- Pathophysiology of Ischemia, Neuropathy and Infection
- Antibiotic Treatment
- Amputation Types
- Wound Management
- Foot Care

Additional Important/Non-Core Curriculum Topics:

- Orthotic Management

10. **Diagnosis and Management of Complications of Vascular Therapy**

Includes:

- Pseudoaneurysms
- Aortoenteric Fistulae/Erosions
- Vascular Graft Infections
- Colon Ischemia after Aortic Surgery
- Chronic Perigraft Seromas
- Occluded Prosthetic Grafts
- Prosthetic Graft Dilation

11. **Diagnosis and Management of Vascular Trauma**

Includes:

- Aortic Trauma
- Carotid Trauma
- Brachiocephalic Trauma
- Visceral Arterial Trauma
- Extremity Trauma
- Venous Trauma
- Diagnosis of Vascular Trauma - Arteriography/Duplex
- Nonoperative Therapy
- Traumatic A-V Fistulas
- Iatrogenic Vascular Trauma

Additional Important/Non-Core Curriculum Topics:

- Associated Neural Injury
12. Diagnosis and Management of Venous Thromboembolic Disease

Includes:

- Deep Venous Thrombosis
- Deep Venous Thrombosis Prophylaxis
- Pulmonary Emboli
- Caval Interruption
- Subclavian/Axillary Thrombosis
- Venous Thrombectomy/Thrombolytic Therapy
- Anticoagulation

Additional Important/Non-Core Curriculum Topics:

- Acute Caval Thrombosis Syndrome
- Pulmonary Embolectomy (open & catheter based)
- Renal Vein Thrombosis
- Budd-Chiari Syndrome

13. Diagnosis and Management of Chronic Venous Insufficiency

Includes:

- Noninvasive Diagnosis
- Medical Treatment
- Sclerotherapy
- Surgical Reconstruction including Subfascial Ligation of Perforators, Valvular
- Congenital Causes

14. Diagnosis and Management of Lymphedema

15. Indications and Techniques for Extremity Amputation

Includes:

- Determination of Amputation Level

Additional Important/Non-Core Curriculum Topics:

- Post-Amputation Care
- Prosthetic Management
- Rehabilitation
- Phantom Pain Symptoms

16. Techniques for the Diagnosis of Peripheral Vascular Disease

Includes:

- Hemodynamic Assessment of Arterial and Venous Disease
Duplex Evaluation of Carotid, Venous, Mesenteric, Renal and Extremity Vascular Disease
Arteriography
Computerized Tomography
MRI/MRA
Intraoperative Duplex Evaluation

Additional Important/Non-Core Curriculum Topics:

Intravascular Ultrasound

17. Use of Endovascular Therapy in the Management of Peripheral Vascular Disease

Includes:

Lytic Therapy
Balloon Angioplasty
Endoluminal Stents
Stent Grafts
Angioscopy
Endoluminal Ultrasound
Embolization

18. Risk Stratification in Patients with Peripheral Vascular Disease

Includes:

Cardiac Risk Evaluation
Pulmonary Risk Evaluation
Atherosclerotic Risk Factor Assessment
Lipid Disorder Evaluation and Management

19. Diagnosis and Management of Coagulation Disorders in Patients with Peripheral Vascular Disease

Includes:

Bleeding Disorders/Intraoperative Bleeding
Heparin Associated Thrombocytopenia
Hypercoagulable States
Low Molecular Weight Heparin
Antiplatelet Agents Including Ticlopidine

20. Diagnosis and Management of Miscellaneous Vasculogenic Problems

Includes:

Vasospastic Diseases
Neurogenic Thoracic Outlet Syndrome
Causalgia/Reflex Sympathetic Dystrophy
Additional Important/Non-Core Curriculum Topics:

Vasculogenic Impotence
Pediatric Vascular Disorders
Frostbite

21. **Diagnosis and Management of Non-Atherosclerotic Vascular Diseases**

Includes:

Systemic Vasculitis
  - Giant Cell Arteritis
  - Takayasu’s Disease
Radiation Induced Arterial Disease
Arterial Infections
Adventitial Cystic Disease
Popliteal Entrapment Syndrome
Buerger’s Disease
Congenital Problems
  - Coarctation
  - Persistent Sciatic Artery
  - Aberrant Subclavian Artery
Arteriopathies
  - Marfan’s Syndrome
  - Ehlers-Danlos Syndrome
  - Arterial Magna Syndrome
  - Cystic Medical Necrosis
  - Behcet’s Disease
Homocystinuria
Intra-Arterial Drug Induced Injury

22. **Diagnosis and Management of Arterial Venous Malformations**

Includes:

Surgical, Catheter and Nonoperative Management of Angiodysplasias

23. **Indications for and Techniques of Vascular Access**

Includes:

Vascular Access for Hemodialysis
  - Ischemic Hand After Vascular Access
  - Peripheral Dialysis Access

24. **Indications for and Results of Sympathectomy in Patients with Peripheral Vascular Disease**

25. **Diagnosis and Management of Portal Hypertension**
EDUCATIONAL OBJECTIVES

1. Aneurysmal Disease
William H. Pearce, M.D., Christopher Zarins, M.D., John W. Hallett, M.D.

I. Basic Science
1. To describe aortic architecture and functions.
2. To describe hemodynamic changes at major bifurcation and Laplace’s Law.
3. To describe the role of aging and atherosclerosis in aortic enlargement.
4. To describe the role of inflammation and proteases in aneurysm formation.
5. To describe the differences in Marfan’s disease and Ehlers Danlos syndrome.

II. Diagnostic Evaluation
1. To understand the incidence and prevalence of aneurysmal disease according to age.
2. To understand the natural history of abdominal aortic aneurysms.
3. To understand the genetic distribution of the disease.
4. To understand the roles of ultrasound, angiography, CT and MRI/MRA in screening and in planning surgery.

III. Treatment
1. To understand the indications for surgical repair and the factors which contribute to surgical decision making.
2. To understand the technical aspects of aortic aneurysm repair and surgical options and alternatives.
3. To describe the surgical management of complex aortic aneurysms (including horseshoe kidneys, aortocaval and aortoduodenal fistulae, mycotic, inflammatory).
4. To have knowledge of both the immediate and long-term outcomes of surgery for aortic aneurysmal disease (including symptomatic, asymptomatic, thoracoabdominal, juxtarenal, infrarenal and recurrent).
5. To describe the management and prevention of surgical complications including spinal cord ischemia, distal embolization, myocardial infarction, graft infection.

References
3. Dobrin PD. Mechanical properties of arteries. Physiol Rev. 1978;58:397-460
30. Katz KA, Cronenwett JL. The cost-effectiveness of early surgery versus watchful waiting in the


2. **Peripheral Vascular Occlusive Disease**  
Anthony D. Whittmore, M.D., James M. Seeger, M.D., Jon R. Cohen, M.D.

I. Anatomy & Pathophysiology  
1. To define the normal arterial anatomy of the peripheral vascular system including commonly encountered anatomic variations.  
2. To recognize the physiologic and pathophysiologic collateral circulatory routes which commonly develop in response to occlusive disease.  
3. To understand the neural, humoral and pharmacologic mechanisms which affect peripheral vascular reactivity and auto-regulatory function.  
4. To appreciate the multiple etiologies of acute peripheral vascular ischemia including embolism, thrombosis, dissection, venous occlusion, trauma.  
5. To appreciate the multiple etiologies of chronic peripheral vascular ischemia including atherosclerosis, aneurysm, entrapment syndromes, trauma, and a variety of non-atherosclerotic occlusive entities.  
6. To understand the mechanism of early and late graft failure, fibro-intimal hyperplasia and progression of disease.

II. Diagnostic Evaluation  
Acute Peripheral Ischemia  
1. To understand the signs and symptoms characteristic of acute arterial ischemia and the differential diagnosis.  
2. To understand the importance of assessing the degree of acute ischemia.  
3. To appreciate the significance of the duration of acute ischemia.  
4. To recognize the importance of antecedent clinical entities which may predispose to acute peripheral ischemia including atrial fibrillation, prior myocardial infarction, aortic dissection and hypercoagulopathies.  
5. To appreciate the significance of initial electrolyte, acid base and other laboratory parameters useful in assessing the magnitude of ischemia to define the indications for appropriate therapy.  
6. To understand the relative indications for immediate diagnostic angiography versus urgent surgical exploration.  
7. To understand the arteriographic findings characteristic of different etiologies and to appreciate the diagnostic imaging options available in addition to arteriography (MRA, CT, duplex imaging).

Chronic Peripheral Vascular Ischemia  
1. To understand the characteristic signs and symptoms of chronic peripheral vascular ischemia relative to the patient’s history and physical examination.  
2. To understand the importance of appropriate imaging studies prior to formulating a therapeutic management plan.  
3. To understand the importance of hemodynamic testing in the formulation of a therapeutic management plan.  
4. To appreciate the characteristic angiographic findings in patients with common patterns of peripheral vascular occlusion as well as the importance of assessing available collaterals.

III. Treatment  
Acute Peripheral Vascular Ischemia  
1. To appreciate the relative indications for immediate angiography, thrombolytic therapy, or urgent surgical exploration relative to the duration of symptoms and magnitude of ischemia.  
2. To have a comprehensive understanding of the variety of surgical exposures of the peripheral vasculature.  
3. To understand the relative indications for the major surgical options available for peripheral occlusive disease including endarterectomy, patch angioplasty and bypass graft (autogenous versus prosthetic).  
4. To understand the role of intra-operative thrombolytic agents, dosage and mechanisms of action.
5. To appreciate the sequela of reperfusion following acute ischemia in terms of systemic effects as well as local effects warranting fasciotomy including the anatomy and physiology of fasciotomy.
6. To be familiar with endovascular options for the treatment of occlusive disease including atherectomy, laser, balloon angioplasty, stent graft, as well as the role of angioscopy.
7. To understand the importance of completion imaging studies following peripheral arterial reconstruction.

**Chronic Peripheral Vascular Ischemia**

1. To have a comprehensive understanding of all standard surgical approaches for surgical revascularization including endarterectomy, patch angioplasty and bypass (in-situ and reversed vein grafts, prosthetic grafts).
2. To understand the difference in application of options relative to the degree of ischemia (claudication versus critical ischemia, with or without tissue necrosis).
3. To understand indications for primary amputation.
4. To have an understanding of the role of endovascular approaches including laser, atherectomy, thrombectomy, balloon dilatation with or without stent, and angioscopy.
5. To have a comprehensive knowledge of popliteal entrapment and advential cystic disease and their treatment.
6. To understand the necessity for post revascularization non-invasive hemodynamic assessment and criteria for reintervention for a failing of failed bypass.

**References**

3. Renal Artery Disease
Kimberly J. Hansen, M.D., Robert G. Atnip, Jr., M.D., Gregorio A. Sicard, M.D.

I. Anatomy and Pathophysiology
1. To define normal renal artery anatomy and collateral pathways important in renal artery disease.
2. To understand the etiology, pathology and natural history of these renal artery lesions:
   a. Renal artery atherosclerosis
   b. Renal artery fibromuscular dysplasia
   c. Renal artery aneurysm
   d. Renal arteriovenous malformation
   e. Takayasu’s arteritis
   f. Middle aortic syndrome/congenital hypoplasia
   g. Atheroembolic disease
   h. Renal artery trauma
   i. Embolic occlusion
   j. Renal artery dissection
3. To define common co-existing extrarenal diseases associated with the various renal artery lesions.
4. To understand the exocrine and endocrine function of the kidney, and relate these to the structure and function of the nephron unit.
5. To understand the renin-angiotensin axis in the absence and presence of renal artery disease.
6. To describe the mechanisms of renovascular hypertension and renovascular insufficiency (i.e., ischemic nephropathy) and to understand how these differ for unilateral and bilateral renal artery disease.

II. Diagnostic Evaluation
Screening and Imaging
1. To describe the clinical features of renovascular hypertension and renovascular insufficiency, and to contrast these with essential hypertension and parenchymal renal failure.
2. To describe the performance and diagnostic criteria for these screening/imaging studies:
   a. Captopril renin test
   b. Captopril renography
   c. Intravenous urography
   d. Ultrasonography
      1. Duplex sonography
      2. Intravascular sonography
   e. Spiral computerized tomography
   f. Magnetic resonance imaging
   g. Angiography
      1. Digital subtraction angiography
         a. Intravenous
         b. Intra-arterial
      2. Cut-film angiography
      3. CO₂ angiography
3. To define the applications and limitations of available screening/imaging studies.

Tests of Functional Significance
1. To distinguish between functionally significant and clinically silent renal artery disease.
2. To define the selection and patient preparation for these studies of functional significance:
   a. Split renal function test
   b. Selective renal vein renin determination
   c. Peripheral plasma renin determination
d. Captopril renin test
e. Captopril renography
3. To describe the diagnostic criteria, predictive value and limitations of each study of physiologic significance.

III. Treatment

1. To describe the strategies, options and anticipated results of medical management for the various renal artery lesions.
2. To appreciate the limitations and complications associated with medical management of renovascular hypertension and renovascular insufficiency.
3. To understand the indications, anticipated anatomic results and clinical response associated with catheter-based intervention for the various renal artery lesions:
   a. PTA ± intravascular stenting
   b. Atherectomy
c. Fibrinolytic therapy
4. To understand the indications for surgical renal artery reconstruction as they relate to the various renal artery lesions.
5. To define the techniques of surgical exposure for renal artery lesions.
6. To understand the selection and performance of direct and indirect reconstruction for the different renal artery lesions:
   a. Direct reconstruction
      1. Aortorenal bypass
      2. Endarterectomy
         a. Transaortic
         b. Transrenal
      3. Reimplantation
      4. Ex vivo reconstruction
   b. Indirect reconstruction
      1. Splanchnorenal bypass
         a. Splenorenal
         b. Hepatorenal
c. Nephrectomy
5. To define the techniques of surgical exposure for renal artery lesions.
7. To describe the anticipated results of reconstruction and nephrectomy as they relate to hypertension response, renal function response, subsequent cardiovascular events and patient survival.
8. To define the management of silent and functionally significant renal artery lesions combined with occlusive or aneurysmal aortic disease.
9. To recognize and develop a plan of management for complications associated with surgical management of renal artery disease and understand how these complications relate to co-existing renal and extrarenal disease.

References
4. Visceral Ischemia
William R. Flinn, M.D., Bruce L. Gewertz, M.D., Leonard P. Krajewski, M.D.

I. Anatomy and Pathophysiology
1. To define the normal arterial and venous anatomy of the mesenteric circulation and to be familiar with the more frequently encountered anatomic variations.
2. To recognize the physiologic and pathophysiologic collateral circulation to the gastrointestinal tract that may develop in response to occlusive disease of the main mesenteric vessels.
3. To understand the high flow, low resistance physiology of normal mesenteric blood flow, recognize the neural, humoral (hormonal) and enteric (intraluminal) mechanisms of autoregulation, and understand the high degree of vasoreactivity of this arterial bed.
4. To understand the multiple etiologies of acute mesenteric ischemia including embolism, thrombosis, dissection, venous occlusion, trauma, and gut ischemia following aortic reconstruction.
5. To understand the multiple possible etiologies of syndromes of chronic mesenteric ischemia including atherosclerosis, aneurysm, extrinsic compression syndromes, and other nonatherosclerotic arteriopathies.
6. To understand the clinical correlation of multiple visceral vessel involvement with the development of symptoms of chronic intestinal ischemia based upon an understanding of the compensatory collateral perfusion of the gut.

II. Diagnostic Evaluation
Acute Mesenteric Ischemia
1. To understand the characteristic initial signs and symptoms suggestive of acute mesenteric ischemia and how symptoms and physical findings may differ from other causes of the acute abdomen.
2. To define preexistent clinical conditions that may predispose to, or support the clinical diagnosis of acute mesenteric ischemia, e.g. atrial fibrillation, previous myocardial infarction (mesenteric embolism), severe cardiopulmonary dysfunction (non-occlusive ischemia), history of post-prandial pain and weight loss, known aortic dissection (mesenteric thrombosis), hypercoaguable states (mesenteric venous thrombosis).
3. To understand the parameters of initial serologic testing that characterize or may support the clinical diagnosis of acute mesenteric ischemia.
4. To define the indications for mesenteric arteriography (or other forms of visceral arterial imaging) in patients with suspected acute mesenteric ischemia and understand the technical aspects of the conduct of arteriography necessary to make an accurate diagnosis.
5. To define the characteristic arteriographic findings diagnostic of the major causes of acute mesenteric arterial ischemia; mesenteric thrombosis, mesenteric embolism, and non-occlusive mesenteric ischemia.
6. To define the appropriate diagnostic evaluation for suspected intestinal ischemia following aortic surgery.
7. To understand the usefulness of alternative imaging techniques (CT, MRI) for the diagnosis of acute mesenteric venous thrombosis.

Chronic Mesenteric Ischemia
1. To understand the characteristic signs and symptoms of chronic mesenteric ischemia and how other aspects of patients’ history (e.g. previous aortic surgery) or physical examination (e.g. aortoiliac occlusive disease) may suggest the presence of associated visceral arterial occlusive disease.
2. To understand the limitations of standard gastrointestinal diagnostic testing modalities (e.g. GI endoscopy, contrast studies, CT, etc.) for diagnosis of chronic mesenteric ischemia.
3. To understand the usefulness of porto-mesenteric duplex ultrasound scanning for elective noninvasive evaluation of the major visceral vessels.
4. To define the indications for arteriography (or alternative vascular imaging studies) in patients with suspected chronic mesenteric ischemia and understand the arteriographic findings that are considered diagnostic of this condition.
5. To recognize the characteristic arteriographic findings in atypical causes of mesenteric arterial compromise.
III. Treatment

Acute Mesenteric Ischemia
1. To be familiar with techniques for surgical exposure of the main mesenteric vessels, to understand standard surgical options for revascularization following acute mesenteric embolism or acute mesenteric arterial thrombosis, and to understand surgical options for the management of intestinal necrosis when this has occurred.
2. To recognize the relationship of different anatomic patterns of gut infarction to the different causes of acute mesenteric ischemia when intestinal infarction is encountered unexpectedly at the time of laparotomy.
3. To understand the critical relationships between the extent of viable bowel (before and/or after successful revascularization) and the extent of resection of nonviable intestine, and the impact of these observations upon both the short and long-term prognosis for the patient.
4. To understand the relative usefulness of intraoperative techniques available for the assessment of intestinal viability at the time of surgical treatment for acute mesenteric ischemia.
5. To understand the pathophysiologic effects of intestinal reperfusion after surgical treatment of acute mesenteric ischemia and the impact of these effects on postoperative patient care.
6. To understand the role of early empiric re-exploration following surgical treatment of acute mesenteric ischemia.
7. To understand standard and alternative treatments for mesenteric venous thrombosis including the role of surgical treatment in the management of this disorder.
8. To understand the management of suspected acute gut ischemia occurring after aortic surgery.
9. To understand the therapeutic role of interventional non-surgical treatments in the management of all forms acute mesenteric ischemia, particularly in non-occlusive mesenteric ischemia.

Chronic Mesenteric Ischemia
1. To be familiar with all standard surgical techniques for direct, elective visceral revascularization and understand the importance of comprehensive revascularization in the surgical treatment of chronic intestinal ischemia.
2. To be aware of surgical alternatives for treatment of atypical or non-atherosclerotic visceral arterial occlusive lesions.
3. To understand the possible application of interventional, nonsurgical treatments for chronic visceral arterial occlusive lesions.
4. To understand the usefulness of noninvasive vascular testing for the follow-up of patients having visceral revascularization procedures.

References
5. **Carotid Artery Disease**  
Alan M. Graham, M.D., Wesley S. Moore, M.D., William Baker, M.D.

I. Anatomy and Pathophysiology  
1. To describe the anatomy of the arch, great vessels, and intracranial arteries.  
2. To describe the embryology of the above and relate the common anomalies to the embryology.  
3. Discuss the collateral arterial communications of the extracranial and intracranial arteries.  
4. To discuss the diagnosis of anomalies and collateral circulation utilizing diagnostic modalities including CT scan, MRI, SPECT, and transcranial doppler.  
5. To understand the different etiologies of carotid artery disease.  
   a. Atherosclerosis  
      i. Define the systemic risk factors for atherosclerosis.  
      ii. Define the systemic effects of atherosclerosis and how these effects impact the diagnosis and treatment of the patient with carotid stenosis.  
   b. Kinking and tortuosity  
   c. Fibromuscular dysplasia  
   d. Compression  
   e. Traumatic occlusion  
   f. Acute Dissection  
   g. Inflammatory arteriopathies  
6. To describe the gross pathologic and histologic characteristics of each etiology above.  
7. To discuss how each etiology produces cerebral events in terms of occlusion and/or embolism.  
8. To discuss the normal flow patterns at the carotid bifurcation, and how they are affected by the atherosclerotic process.

II. Diagnostic Evaluation  

**History and Physical Examination**  
1. To define hemispheric, non-hemispheric, and non-specific symptoms.  
2. To differentiate among transient ischemic attack (TIA), reversible ischemic neurologic deficit (RIND), stroke in evolution and completed stroke.  
3. To describe the arterial and neurologic examination and their importance in caring for patients with carotid artery disease.  
4. To describe the relationship between carotid artery atherosclerosis and the clinical syndrome of vertibrobasilar insufficiency.  
5. To describe and defend the appropriate evaluation for patients with each of the above clinical presentations.

**Carotid Duplex Examination**  
1. To be able to explain the principles of doppler ultrasound.  
2. To describe the normal doppler signals in the internal, external, and common carotid arteries.  
3. To discuss the sensitivity and specificity of duplex scanning in detecting carotid artery stenosis.  
4. To discuss the risks and benefits of relying on duplex ultrasound and eliminating angiography.  
5. To understand the basics of P.V. Lab Accreditation.

**Angiography and MRA**  
1. Angiography: to be able to discuss the technique, its limitations and complications.  
2. MRA: to be able to discuss the technique. limitations and complications.  
3. To discuss and compare the different methods of measuring stenosis.
Diagnostic Brain Scanning
1. For each of the following modalities, explain the principles, indications, complications, and its influence upon the indications for carotid endarterectomy.
   a. CT scan
   b. MRI
   c. SPECT
   d. Transcranial doppler

III. Treatment
Treatment of Neurologic Syndromes in Patients with Carotid Stenosis
1. To discuss the non-surgical and surgical treatment of acute ischemic syndromes including stroke.
2. To discuss the role of thrombolytic therapy in the treatment of stroke syndrome.
3. To be able to construct a diagnostic and treatment algorithm for various stroke syndromes.
4. To be able to discuss the potential role of endovascular treatment.

Surgical Treatment
1. To discuss the intrathoracic and extrathoracic treatment of atherosclerotic stenosis or occlusion of the great vessels.
2. To describe the standard approach to carotid endarterectomy including intraoperative shunting, patching, anesthetic techniques, tacking sutures and methods of completion evaluation.
3. To describe the surgical treatment of fibromuscular dysplasia, kinking, radiation arteritis, tumors involving the carotid artery, other arteritides, and recurrent carotid stenosis.
4. To recognize the carotid sinus syndrome and discuss its treatment.
5. To discuss EC-IC bypass.
6. To discuss the indication and performance of proximal and distal vertebral artery reconstruction.

Complication of Carotid Endarterectomy
1. To describe the etiology and management of:
   a. Wound hematoma
   b. Wound infection
   c. Post-operative hyper and hypotension
   d. Peripheral nerve palsies
   e. Transient ischemic attack and stroke
   f. Asymptomatic thrombosis
   g. Intracranial hemorrhage
   h. Post-operative seizure
   i. Extracarotid (cardiac) events

References


6. **Innominate, Subclavian and Vertebrobasilar Arterial Disease**
Ramon Berguer, M.D., Michael DaValle, M.D., Francis Robicsek, M.D.

**I. Etiology, Pathophysiology and History**
1. Pathophysiology of atherosclerosis, trauma, dissection, arteritis and radiation as it applies to the innominate, subclavian and vertebrobasilar arteries.
2. Concomitant diseases and associated risk factors commonly associated with stenosis, occlusion, dissection, ulcerated atheroma, arteriovenous fistula and false aneurysm of these arteries.
3. Define the most appropriate diagnostic steps for the evaluation and for the choice of treatment of these conditions.
4. Abnormal and alternative flow patterns that may develop as a consequence of lesions of the innominate, subclavian and vertebrobasilar arteries.
5. Best diagnostic methods available to assess end-organ effects in the brain and upper extremities of the lesions mentioned above.
6. Natural history of these conditions and how this natural history is affected by treatment methods when the latter are successful and when they fail.

**II. Diagnosis**
1. Symptoms and signs of brain ischemia in its various manifestations, localized and global, progressive and sudden.
2. Symptoms of ischemia of the upper extremity.
3. Signs of ischemia of the brain or upper extremities elicited by provocative maneuvers.
4. Understand the differential diagnosis of conditions that may present with similar signs or symptoms.
5. Understand how noninvasive tests may suggest or deny the presence of lesions of the innominate, subclavian and vertebrobasilar arteries and how these tests may preclude or indicate arteriography.
6. Understand the anatomy of these arteries and their lesions as defined by arteriography, the timing of films and the best projections to display them.
7. Know the risks involved in arteriography relative to the contrast agents used and their amount, the approach used and the pharmacologic and technical maneuvers employed.
8. Value and shortcomings of CT and MRA/MRI imaging techniques in the diagnosis of these entities.

**III. Treatment**
1. Options for (a) medical treatment (antiplatelet, anticoagulant, steroids, antiinflammatory drugs), (b) surgical repair whether direct (endarterectomy, transposition, ligation) indirect (bypass, decompression) or (c) endovascular (angioplasty, stenting, covered stents).
2. Indications for combined treatment and their timing.
3. Possible complications of each of the above treatments and their management.
4. Long-term results with the different treatment options.

**References**
7. Thoracic Outlet Syndrome
Jonathan B. Towne, M.D., John Corson, M.D., Irving Kron, M.D.

I. Anatomy and Pathophysiology
1. To understand the anatomy of the thoracic outlet to include anatomic variations in bones, muscles, and cervical ribs.
2. To recognize the origin of insertion of the musculoskeletal structures which surround the nerves and blood vessels that supply the arm.
3. To recognize the location of the costovertebral ligaments and the boundaries of the scalene triangle and the costoclavicular space.
4. To recognize the location and incidence of anatomic variations of the insertion of the cervical rib.
5. To recognize insertions of the anterior scalene and its relationship to the neurovascular structures.
6. To recognize the origin and insertion of the subclavius muscle and the possibility of encroaching the neurovascular structures in the costoclavicular triangle.
7. To recognize and define skeletal abnormalities, e.g. elongated C7 transverse process, callous formation from a fractured clavicle or first rib, hypoplastic first rib, the anatomy of cervical nerves C5, C6, C7, C8, and T1, and their relationships to the thoracic outlet.

II. Diagnostic Evaluation
1. To understand that pain is a principal symptom of neurologic type of thoracic outlet and that the distribution of pain which arises from the upper three nerves of the brachial plexus, C5, C6, and C7, as distinct from the pattern of pain emanating from the lower nerves of the plexus, C8 and T1.
2. To recognize the arterial symptoms (embolization to hand and forearm, post stenotic dilatation, and subclavian artery occlusion) and venous symptoms (subclavian vein thrombosis for clinical diagnosis).
3. To understand this may present as spontaneous, related to injury (hyperextension, flexion injuries of the neck, blunt trauma), or that symptoms may occur with hyperadduction of the shoulder or arm exertion.
4. To define differential diagnoses of thoracic outlet to include cervical disc syndrome, carpal tunnel syndrome, orthopedic shoulder problems (shoulder sprain, rotator cuff injuries, tendonitis, cervical spondylitis, ulnar nerve compression at the elbow), Multiple Sclerosis, spinal cord tumor disease, angina pectoris, and Pancoast’s tumor.
5. To understand the importance of obtaining blood pressure in both arms, clinical examinations of the hand, examination for muscle atrophy, and evaluation for muscle strain and percussion of the supra clavicular fossa.
6. To understand and have knowledge of tests used to evaluate thoracic outlet, i.e. Adson’s test, hyperabduction test, and costoclavicular test.
7. To understand the role of vascular lab in the diagnosis using duplex evaluation to detect thrombosis of the subclavian vein and arterial studies of the upper extremity.
8. To define the physical findings of embolization to the digital vessels and occurrence of palpable aneurysm in the supraclavicular fossa.
9. To recognize the angiographic findings related to this syndrome including false aneurysm, post stenotic dilatation, and subclavian artery occlusion.

III. Treatment
1. To be familiar with surgical techniques and anatomy for first rib resection (transaxillary, supraclavicular, total anterior scalenotomy).
2. To define specific complications related to the surgical approach (traction injuries to the brachial plexus, pneumothorax, injury of the subclavian artery, injury to the subclavian vein, air embolus as a result of subclavian vein injury, nervous system injury, i.e. long thoracic nerve, intercostobrachial nerve, musculocutaneous nerve).
3. To be aware of the symptoms and incidence of these complications and nerve injuries.
4. To be familiar with the management of subclavian artery aneurysms including the use of graft materials and treatment of distal emboli.

5. To be familiar with thrombolytic therapy in the management of subclavian vein thrombosis.

6. To define the timing of a 1st rib resection with regard to subclavian vein thrombosis.

7. To be aware of the incidence of recurrence of thoracic outlet syndrome.

8. To be aware of the incidence of litigation pertaining to the diagnosis and treatment of thoracic outlet syndrome.

9. To have an understanding of the treatment options to include conservative approaches such as physical therapy and treatment of muscle spasm.

References


4. Sanders RJ, Cooper MA. Surgical management of subclavian vein obstruction, including six cases of subclavian vein bypass. Surgery 1995;118:856-863


8. Acute Arterial Occlusion
Keith Calligaro, M.D., David Drezner, M.D., Frank Veith, M.D.

I. Anatomy and Pathophysiology
1. To understand the various causes of acute arterial thrombosis including chronic atherosclerosis, hypercoaguable conditions, catheters and medical devices, and drug injections.
2. To understand various sources of peripheral arterial emboli including the heart (and underlying factors including myocardial infarcts, valve disease, atrial fibrillation, intracardiac tumors), arterial aneurysms and ulcerative plaques.
3. To define the variable interval of acute arterial ischemia before irreversible changes of the muscle and peripheral nerves begin to occur.
4. To understand the reasons for the high morbidity and mortality associated with acute arterial occlusion even when treatable by simple, straightforward operations.
5. To understand impaired reflow phenomenon including cellular edema, vascular lumen narrowing, capillary occlusion, and oxygen derived radicals.
6. To understand ischemia-reperfusion syndrome and its complications, including compartment syndrome, hyperkalemia, metabolic acidosis, myoglobinuria and renal insufficiency, and pulmonary insufficiency.
7. To understand the etiology and clinical presentation of "blue-toe syndrome".
8. To understand how the degree of arterial collateralization, in particular chronicity of underlying arterial disease and site of arterial occlusion in reference to major collaterals, affects severity and course of symptoms.

II. Diagnostic Evaluation
1. To understand the classic signs and symptoms of acute arterial insufficiency (pallor, decreased temperature, pulselessness, paraesthesias, paresis, pain) along with other more subtle findings such as poor venous filling.
2. To be able to recognize features of the viable, threatened and irreversibly ischemic extremity.
3. To correlate other systemic clinical findings with the likely cause of acute arterial occlusion including atrial fibrillation, claudication or a past history of unexplained previous arterial or venous clotting.
4. To understand the utility of doppler studies of peripheral arteries.
5. To understand the indications for preoperative arteriography in the setting of acute arterial occlusion.
6. To understand arteriographic findings suggestive of embolus or thrombus due to underlying arterial stenosis.

III. Treatment
1. To understand the role of heparin to prevent propagation of thrombus and protect the distal arterial tree.
2. To understand the benefits of mannitol for patients with advanced acute arterial occlusion.
3. To understand the importance of hydration and correcting electrolyte imbalances.
4. To understand the role of thrombolysis as the initial treatment of acute arterial occlusion and its role intraoperatively.
5. To understand the value of full preoperative arteriography in localizing the level of occlusion, the presence of other occlusions and stenoses, and suitable vessels for a bypass should it be needed.
6. To understand the importance of appropriate prepping and draping of the patient to gain access for possible venous conduits and appropriate inflow and outflow arteries.
7. To make correct decisions concerning the proper locations and type of arteriotomy depending on whether an embolus is the likely source of acute arterial occlusion or thrombus secondary to underlying chronic arterial stenosis.
8. To understand the proper technique when using thromboembolectomy catheters.
9. To understand the importance of completion arteriography.
10. To understand the indications for and technique of fasciotomy.
References
9. **Diabetic Foot Problems**  
Frank W. LoGerfo, M.D., Jennifer Doyle, M.A.

I. **Anatomy and Pathophysiology**  
1. To define the normal arterial and venous anatomy of the circulation of the foot.  
2. To demonstrate an understanding of the etiology of three pathogenic mechanisms underlying problems of the diabetic foot:  
   a. ischemia.  
   b. neuropathy  
   c. infection (polymicrobial nature)  
3. To outline factors that can affect blood glucose levels in the peri- and postoperative period

II. **Evaluation and Diagnosis**  
1. To demonstrate an understanding of the presenting signs and symptoms of three pathogenic mechanisms underlying problems of the diabetic foot:  
   a. ischemia: microvascular abnormalities, atherosclerosis, pattern of atherosclerosis, tibial vessel disease, mediocalcification.  
   b. neuropathy: motor, foot deformities, charcot foot, sensory neuropathy, neuroinflammatory response, manifestations of autonomic neuropathy  
   c. infection: altered clinical picture, metabolic consequences, polymicrobial nature  
2. To understand the limitations of various non-invasive tests in the diagnosis of ischemia, the effect of calcified vessels, the role PVR, toe pressures  
3. To understand the role of angiography  
   a. susceptibility to contrast induced ARF  
   b. role and techniques of hydration  
   c. need for visualization of foot arteries  
4. To evaluate ulcer for ischemia, infection, neuropathy  
   a. use of sterile probe  
   b. role of foot films and interpretation, appearance of charcot changes  
5. To accurately interpret clinical laboratory results, pathology reports, and radiographic studies  
6. To synthesize historical findings, physical examination and laboratory data for diagnosis;  
7. To identify inflow and outflow vessels on an arteriogram  
8. To assess patient’s ability to maintain level of activity (walk, drive motor vehicle, work, exercise, sexual activity)

III. **Treatment**  
1. To understand priorities of management in diabetic patients with foot problems:  
   a. timing and methods of debridement in drainage for sepsis  
   b. metabolic control  
   c. evaluation of ulcer, depth, sepsis, involvement of bone, tendon  
   d. options for conservative management, role of foot gear, weight bearing  
   e. when to evaluate for ischemia  
   f. options in the management of the non-ischemic, purely neuropathic ulcer  
2. To understand the role of distal bypass  
   a. role of dorsalis pedis bypass  
   b. alternative inflow sights  
   c. outcome as a function of inflow and outflow site  
3. To understand the principles and techniques of wound care, dressing changes, debridement  
4. To understand the timing and methods of soft tissue closure  
5. To understand the long term importance of glycemic control, weight
6. To recognize the need for careful follow-up and patient education for diabetic patients with foot problems
7. To specify proper dressings and foot care for prevention of problems in diabetic patients, e.g., the role of orthotics, foot gear, nail care
8. To categorize the prevention and management of operative and postoperative complications, including graft infections, graft thrombosis and extremity ischemia
9. To develop familiarity with all techniques of arterial reconstruction including dorsalis pedis bypass and describe the specific role these operations have in management of the diabetic foot
10. To outline the indications for and illustrate the techniques of distal reconstruction, major and minor amputations
11. To outline indications for, and illustrate techniques of:
   - debridement and drainage;
   - arterial reconstruction;
   - vascular bypass grafting;
   - amputation
12. To maintain appropriate control of diabetes peri-operatively, in:
   - NIDDM patient
   - IDDM patient
13. To present an appropriate management plan for the severely septic foot
14. To describe the general outcomes of the diabetes control and complications trial (DCCT) for the purpose of counseling patients
15. To develop appropriate plans for management
16. To manage postoperative surgery and anesthesia complications
17. To delineate and select appropriate postoperative care of patients with diabetes
18. To communicate to patients instructions and expectations for follow-up, such as:
   - pain level and location
   - possible side-effects of medications
   - level of activity and return to work
   - wound care and potential problems
   - timing of follow-up appointment
19. To arrange for home health and other outpatient services using institutional and community resources
20. To understand the role of the surgeon in taking the lead in management of the diabetic foot problem
21. To understand that care of the diabetic foot must necessarily go beyond the vascular reconstruction
22. To appreciate the importance of the team to provide maximum benefit for the patient
23. To demonstrate an understanding of, and sensitivity to, patient socioeconomic concerns regarding such issues as insurance and the ability to pay for physician services, hospitalization, and prescribed medications: loss of work time and wages
24. To demonstrate sensitivity and appropriate flexibility regarding patient fears and concerns, including:
   a. preoperatively - anxiety about pain
   b. postoperatively - ability to care for self, drugs, level of function, prognosis

References
10. **Complications of Vascular Therapy**
Dennis F. Bandyk, M.D., Jeffrey L. Ballard, M.D., Calvin B. Ernst, M.D.

I. Anatomy and Pathophysiology
1. To recognize the factors involved in loss of arterial wall and anastomotic tensile strength resulting in the development of pseudoaneurysms.
2. To define the incidence and mechanisms which led to the development of secondary aortoenteric fistulae and erosions.
3. To understand the multiple etiologic factors associated with increased risk of infection following arterial surgery, including biomaterial implantation, host immune factors, concomitant medical conditions, nature and magnitude of bacterial contamination, and wound healing complications.
4. To understand virulence factors of gram-positive and gram-negative microorganisms involved in vascular graft infections.
5. To understand mechanisms involved in bacterial contact, adherence, and colonization of prosthetic graft material.
6. To understand the etiologies causing absence of graft incorporation and perigraft fluid collections including infection, seroma, hematoma, and lymphocele.
7. To define the normal arterial circulation of the colon and anatomic variations produced by abdominal aneurysm repair and prior colectomy.
8. To understand the etiologies causing failure of graft incorporation and perigraft fluid collections including infection, seroma, hematoma, and lymphocele.
9. To recognize anatomic and hemodynamic conditions which can result in graft occlusion including myointimal hyperplasia, atherosclerotic disease progression, anastomotic false aneurysm formation, graft entrapment, low flow, thromboembolism, hypercoagulable states, and infections.
10. To understand the etiologic differences between immediate and late graft occlusions.
11. To define the incidence and mechanisms of prosthetic graft dilatation.
12. To understand the expected incidence and etiologies of wound healing complications including hematoma, infection, and lymphocele.
13. To recognize non-vascular complications associated with arterial therapy including cardiac ischemia, renal failure, and neurologic deficits.
14. To understand the normal arterial circulation of the spinal chord and the pathophysiology of paraplegia caused by spinal chord ischemia.

II. Diagnostic Evaluation
**Pseudoaneurysms**
1. To recognize the clinical manifestations of pseudoaneurysm following arteriography, percutaneous transluminal angioplasty, and bypass grafting.
2. To define the appropriate diagnostic evaluation of pseudoaneurysm including the use of duplex ultrasound, computed tomography, magnetic resonance imaging, and arteriography.
3. To recognize the operative findings of infected anastomotic false aneurysm.

**Aortoenteric Fistulae/Erosions**
1. To understand characteristic symptoms and signs of secondary aortoenteric fistula/erosion including prior aortic graft implantation, herald gastrointestinal bleeding, fever, and concomitant anastomotic false aneurysm.
2. To define the appropriate diagnostic evaluation of suspected secondary aorto- or graft-enteric fistula including upper endoscopy, anatomic imaging techniques (CT, MRI, arteriography), and radionucleide functional scans.
3. To understand the role of operative graft exploration in patients with recurrent GI bleeding and normal GI endoscopy.
Vascular Graft Infections
1. To define the epidemiology of prosthetic graft infection.
2. To understand the characteristic signs and temporal presentation of acute versus late-appearing graft infections including sepsis, GI or perigraft bleeding, fever, malaise, false aneurysm, abdominal, back, or groin pain.
3. To understand the usefulness of various microbiologic culture (agar media, broth media) and recovery (swap culture, biomaterial culture, CT-directed aspiration) techniques in the diagnosis or confirmation of graft infection.
4. To understand the parameters of serologic testing that support the clinical diagnosis of vascular graft infection.
5. To define the appropriate diagnostic evaluation of suspected graft infection including microorganism recovery techniques, functional and anatomic graft imaging, and arteriography.

Colon Ischemia after Aortic Surgery
1. To understand the characteristic initial signs and symptoms suggestive of colon ischemia
2. To define pre- post-operative clinical conditions that may predispose to colon ischemia after after surgery including visceral occlusive disease (meandering mesenteric artery), prior colorectal surgery, ligation of inferior mesenteric artery, ruptured abdominal aortic aneurysm, postoperative shock.
3. To understand the usefulness of Doppler ultrasound and photoplethysmography in the operative diagnosis of colon ischemia.
4. To define the appropriate diagnostic evaluation for suspected colon ischemia following aortic surgery including the use of rigid and flexible sigmoidoscopy, colonoscopy, and operative exploration.
5. To describe the endoscopic features of the severe and mild (reversible) forms colon ischemia after aortic surgery.

Occluded Prosthetic Grafts
1. To recognize the symptoms and signs of limb ischemia associated with graft thrombosis.
2. To understand the role and the interpretation of noninvasive vascular testing techniques used for the diagnosis of graft thrombosis including Doppler-derived limb blood pressure measurements, velocity waveform analysis, pulse volume recordings, and duplex scanning.
3. To define the appropriate diagnostic evaluation of graft occlusion based on severity of limb ischemia.
4. To describe the angioGraphic features of graft occlusion which indicate embolic versus thrombotic occlusion, and the potential for catheter-directed thrombolysis as a treatment option.

Perigraft Seroma and Graft Dilatation
1. To define the clinical presentation of perigraft seroma and graft dilatation including symptoms, signs, and postoperative appearance time.
2. To understand the usefulness imaging techniques (ultrasound, CT, MRI, arteriography) in the diagnosis of etiologic factors associated with failure or graft incorporation.
3. To describe the features of aspirated perigraft fluid which distinguish between chronic perigraft seroma and low-grade graft infection caused by S. epidermidis.
4. To understand the graft types associated with dilatation.

Wound Complications
1. To define the incidence and clinical manifestations associated with wound hematoma, infection, lymphocele, and dermal necrosis following arterial surgery.
2. To define the classification of wound infection following arterial bypass grafting.
3. To define the essentials of diagnosis to distinguish infectious from non-infectious wound complications.

Non-Vascular Complications
1. To understand the clinical symptoms and signs, and ECG features of cardiac ischemic.
2. To define the parameters of serologic and urine testing that characterize acute renal failure.
3. To understand the clinical symptoms and signs of neurologic deficit associated with spinal chord ischemia, injury to peripheral nerves, and cauda equina syndromes.
4. To define the appropriate evaluation of paraplegia following aortic surgery.

III. Treatment

Pseudoaneurysm
1. To define the anatomic features of false aneurysms which should be repaired.
2. To understand techniques for surgical exposure and proximal control of aortic, femoral, and other peripheral artery false aneurysms, including the use of balloon-tipped catheters to prevent backbleeding.
3. To define the role of duplex-guided ultrasound for the treatment of common femoral artery pseudoaneurysms following diagnostic arteriography or percutaneous endovascular procedures.
4. To understand the role of interposition grafting to normal artery wall in the treatment anastomotic false aneurysm.

Aortoenteric Fistulæ/Erosions
1. To understand the role of staged-remote versus immediate-sequential bypass in treatment of aorto-enteric fistula based on severity of GI bleeding and degree of systemic sepsis.
2. To be familiar with surgical techniques involved in ex-situ bypass, total and partial graft excision, restoration of GI tract continuity, and in situ graft replacement using autologous venous graft, endarterectomized arteries, allograft, and antibiotic-bonded vascular prostheses.
3. To be familiar with surgical techniques of aortic ligation including treatment or aortic sepsis involving the renal or visceral arteries.
4. To define the nature and duration of antibiotic therapy associated with treatment of secondary graft-enteric fistulæ/erosions.
5. To define the follow-up of patients treated or aortoenteric fistula/erosion.

Vascular Graft Infections
1. To understand the role local treatment and other graft preservation techniques, including muscle flap coverage, in the treatment of exposed arterial grafts and graft infections without anastomotic involvement.
2. To understand the usefulness of in situ graft replacement techniques using autologous, allograft, and vascular prosthetic grafts in selected patients with vascular graft infections, including selection of treatment method based on clinical manifestations, microbiology, and operative findings.
3. To be familiar with antibiotic therapy based on susceptibility testing in the treatment of arterial graft infections.
4. To be familiar with surgical techniques for excision and ex-situ bypass of infected aortic, peripheral, and carotid arterial reconstructions/bypass grafts.
5. To understand the role of graft excision and arterial ligation in patients with graft infection and adequate collateral circulation.
6. To be familiar with surgical techniques for the treatment of aortic stump sepsis or disruption.
7. To define the expected outcome of patients treated for aortic, infrainguinal, or carotid graft infections.

Colon Ischemia after Aortic Surgery
1. To be familiar with criteria for IMA re-implatation during aortic surgery.
2. To understand the role and technique of colon resection in treatment of severe ischemia.
3. To define the treatment and follow-up of mild colon ischemia following aortic surgery.

Occluded Prosthetic Grafts
1. To be familiar with surgical techniques useful in the treatment of immediate versus late graft occlusions.
2. To define the role of thrombolysis versus surgical intervention for graft occlusion/thrombosis.
3. To define the indications for graft thrombectomy and revision versus graft replacement.
4. To define the role of endovascular techniques (angioscopy, PTA, stent placement) as adjuncts to graft revision procedures.
5. To be familiar with extra-anatomic bypass grafting techniques in treatment of aortofemoral graft limb occlusion.
6. To understand the role of anti-thrombotic therapy in treatment of graft thrombosis.

**Perigraft Seroma and Graft Dilatation**
1. To be familiar with graft replacement techniques as treatment for perigraft seroma and graft structural failure.
2. To understand the importance of microbiologic recovery techniques, including broth culture of graft material, to exclude a biofilm infection.
3. To define the surveillance of prosthetic grafts following implantation to diagnose dilatation, failure of graft incorporation/healing, and anastomotic false aneurysm.

**Wound Complications**
1. To understand the role of prophylactic antibiotics in the prevention wound and graft infections.
2. To understand the standard surgical principles used to treat wound necrosis, hematoma, and infection.
3. To be familiar with non-surgical and surgical techniques useful in the treatment of lymph fistula, lymphocele, and postoperative lymphoedema.

**Nonvascular Complications (Cardiac, Renal, Neurologic)**
1. To understand the role of pre-operative testing, intra-operative monitoring, and post-operative measures to prevent cardiac ischemia.
2. To be familiar with renal preservation techniques associated with aortic and renal surgery.
3. To be familiar with techniques to improve spinal chord perfusion during aortic surgery.

**References**
11. Management of Vascular Trauma
David Rosenthal, M.D., Robert Batson, M.D., Joseph Mills, M.D.

I. Etiology and Pathophysiology
1. To understand the mechanism of vascular injury to the upper extremity, thoracic aorta, abdominal aorta and its branches, and lower extremities.
2. To recognize the clinical importance of penetrating vascular trauma (penetrating objects), significance of different gunshot wounds (high/low velocity) and the blunt or crush injury to the vascular system.
3. To define how vascular reconstructive procedures and the failure of these procedures affect the circulatory system.
4. To understand the mechanism of iatrogenic vascular injury and its prevention.

II. Diagnostic Evaluation
1. To understand the characteristic signs and symptoms of acute vascular compromise.
2. To demonstrate an understanding of the wounding mechanism, assessment of the wound and characteristic findings of the affected extremity distal to the wound and associated injuries.
3. To understand the usefulness of alternative imaging techniques (ie two plane x-ray, Doppler/duplex color flow ultrasonography, venography, angiography, MRI and CT scans) in the management of vascular trauma.
4. To define the characteristic diagnostic finding of imaging techniques in vascular trauma.

III. Acute Arterial Injuries
1. To understand the characteristic signs and symptoms of acute arterial injury.
2. To define the clinical features of major arterial injury.
3. To understand the indications for noninvasive (Doppler or duplex color flow ultrasonography CT, MRI) and invasive (arteriography, venography) diagnostic studies.
4. To define the preoperative assessment and management of the patient with a major arterial injury.
5. To understand the operative management of acute arterial injury and the management of concomitant venous or visceral injuries.
6. To define the operative approach for specific arterial injuries (ie left and right subclavian).
7. To understand the management of postoperative complications and the management of associated injuries.

IV. Venous Injuries
1. To understand the characteristic signs and symptoms of acute venous injury.
2. To define the clinical features of major venous injury.
3. To understand the indications for noninvasive (Doppler or duplex color flow ultrasonography CT, MRI) and invasive (venography) diagnostic studies.
4. To define the preoperative assessment and management of the patient with a major venous injury.
5. To understand the operative management of combined arterial and venous injuries, technical management of venous injuries (ie ligation, lateral suture repair, end-to-end anastomosis, venous patch graft or venous replacement graft).
6. To define operative approach and appropriate management of specific major venous injuries (ie management of retro hepatic IVC, subclavian vein).
7. To understand the management of postoperative complications, and associated injuries.

V. Arteriovenous Fistulas (AVF)
1. To understand the characteristic signs and symptoms of AVFs.
2. To understand the mechanism of injury associated with traumatic AVFs.
3. To define the pathophysiology of AVFs (ie peripheral vascular resistance, heart rate, stroke volume, cardiac output and blood pressures).
4. To understand the indications for noninvasive and invasive diagnostic studies.
5. To define and understand treatment options (ie invasive radiologic procedures, endovascular procedures, and operative techniques).

VI. Iatrogenic Injuries
1. To define the mechanism of the iatrogenic injury.
2. To understand the clinical features associated with the iatrogenic injury.
3. To understand the indications for noninvasive and invasive diagnostic studies suspected iatrogenic injury.
4. To define the indications for nonoperative vs. operative treatment of iatrogenic injury.
5. To understand the management and potential complications associated with an iatrogenic injury.

VII. Concomitant Fracture and Neurologic Injuries
1. To understand the characteristic signs and symptoms of associated fractures and neurologic injuries with vascular trauma.
2. To understand the anatomic relations with fractures, neurologic injury and the vascular system.
3. To define the mechanism of injury from fracture, dislocation, or subluxation.
4. To understand the influence of penetrating, blunt and crush injuries on the vascular system.
5. To define the noninvasive and invasive diagnostic tests associated with fracture and neurologic injury and the vascular system.
6. To define associated reconstructive procedures associated with fracture, neurologic injury and the vascular system.
7. To understand the postoperative management of the patient with combined vascular, fracture, or neurologic injury.

VIII. Nonoperative Management of Vascular Injuries
1. To define the clinical criteria and indications for nonoperative versus operative management of patients with vascular injuries.
2. To define the clinical pathology and mechanism of injury (penetrating, crush, blunt) associated with combined vascular and visceral injuries.
3. To define and understand the surgical anatomy in relationships of the abdominal aorta and its major branches to the abdominal organs.
4. To define the role of the surgical technique (ie x-rays, peritoneal lavage, laparoscopic assessment, systoscopy, proctosigmoidoscopy, IVP, arteriography, etc) with suspected vascular and visceral injury.
5. To define the operative management of the patient with combined vascular and visceral injury.
6. To demonstrate an understanding of postoperative care for critically ill patients with combined vascular and visceral injuries, potential complications and their appropriate management.

References
Thoracic/Mediastinal

Carotid/Subclavian/Vertebral
3. Hiatt JR, Busuttil RW, Wilson SE. Impact of routine arteriography on management of penetrating neck

Renal

Combined Orthopedic/Vascular Injuries

Extremities

Venous Injury

Diagnostic Studies
2. Johansen K, Lynch K, Paun M, Copass M. Noninvasive vascular tests reliably exclude occult arterial
12. **Venous Thromboembolic Disease**  
Anthony J. Comerota, M.D., H. Edward Garrett, Jr., M.D., Richard Welling, M.D.

I. Etiology, Risk Factors, Epidemiology and Pathophysiology  
1. To understand that Rudolf Virchow, the Father of cellular pathology, wrote the classic triad of stasis, hypercoagulable state and vein wall damage leading to venous thrombosis.  
2. To understand that all three elements can be involved in patients undergoing elective operations, causing postop DVT remote from the operative wound.  
3. To understand that risk factors are quantitative and that increasing the number of risk factors increases the likelihood of venous thromboembolic complications.  
4. To understand that not all risk factors are created equal. Malignancy, older age, obesity, long bone fractures, joint replacement, pelvic operations and a previous history of DVT/PE carry more weight (and higher risk) than other considerations.  
5. To be familiar with the known hypercoagulable states, and to understand the relative frequency, mechanism of action and treatment of each. (These include: anticardiolipin/antiphospholipid antibodies, lupus anticoagulant, protein C and protein S deficiency, antithrombin III deficiency, hyperfibrinogenemia, plasminogen deficiency, factor V Leiden mutation (activated protein C resistance), heparin induced thrombocytopenia, Coumadin (warfarin) induced skin necrosis.  
6. To realize that pulmonary embolism is the most common preventable cause of death in hospitalized patients.  
7. To understand that many patients, and perhaps a majority, receive inadequate DVT prophylaxis.  
8. To understand the alterations of coagulation which occur during pregnancy.

II. Spectrum of Venous Thrombosis  
1. To understand that asymptomatic DVT can be dangerous (80% of fatal pulmonary emboli occur in patients who do not carry the diagnosis of acute DVT).  
2. To understand that 20-30% of isolated calf DVT will extend if untreated.  
3. To understand that the superficial femoral vein is actually the main deep vein of the thigh.  
4. To appreciate that as an increasing number of segments are involved with venous thrombosis, the clinical picture of acute DVT is increasingly severe and the more problematic the post-thrombotic sequelae.  
5. To understand that phlegmasia alba and phlegmasia cerulea dolens refer to the clinical findings resulting from iliofemoral DVT, not that the underlying etiology is different.  
6. To appreciate that venous gangrene is not equivalent to phlegmasia cerulea dolens.  
7. To appreciate the difference that extensive greater saphenous thrombosis is best treated by ligation and stripping whereas, extensive greater saphenous thrombophlebitis is best treated by ligation of the saphenofemoral junction, warm soaks, compression and nonsteroidal anti-inflammatory agents.  
8. To appreciate that short segment distal superficial disease is best treated symptomatically.

III. Diagnosis  
Goals:  
1. To understand that the absence of clinical findings does not exclude deep venous thrombosis.  
2. To appreciate that if the thigh is swollen, the common femoral vein and/or iliac veins, or the superficial femoral and profunda must be involved with the thrombotic process, assuming acute DVT is the etiology of swelling.  
3. To understand that physiologic tests, including a venous doppler, impedance plethysmography (IPG), phleborheography (PRG), air plethysmography (APG), or any maximal outflow technique cannot detect nonocclusive venous thrombosis.  
4. To appreciate that physiologic tests are inadequate for use as screening tests and inadequate as endpoint testing for efficacy of DVT prophylaxis.  
5. To appreciate that ascending phlebography is the traditional gold standard for diagnosis of DVT, which is
being replaced by venous duplex imaging.
6. To understand that the primary diagnostic criterion for DVT with ascending phlebography is visualized thrombus. Nonfilling of a deep vein is a secondary criterion.
7. To appreciate that with ascending phlebography artifacts are due to flow voids from nonopacified blood draining from tributaries, external compression, and laminar flow.
8. To know the appropriate indications for evaluation for a hypercoagulable state.
9. To appreciate that in patients with DVT/PE, blood samples drawn for a hypercoagulable evaluation should be obtained before anticoagulation is initiated.
10. To appreciate that a common finding of a patient with lupus anticoagulant (a misnomer) is prolongation of the PTT on a routine screening blood test.
11. To appreciate that the function of activated factor C is to reduce the activity of factor Va and VIIIa.
12. To appreciate that protein C is vitamin K dependent, has a short half-life, and plasma levels are rapidly reduced by warfarin compounds.
13. To appreciate that antithrombin III is also known as heparin cofactor and is lowered by therapeutic levels of heparin.
14. To appreciate that antithrombin IIIa is significantly increased (approximately 750x) with low dose subcutaneous heparin, which is the basis for low dose heparin prophylaxis.
15. To understand that if DVT is suspected in the pregnant woman and the noninvasive test results are equivocal, ascending phlebography should be performed.

IV. Treatment

Goals:
1. To understand the multiple actions of heparin, the reasons for heparin resistance and the complications of heparin.
2. To understand that platelet counts must be monitored during heparin therapy regardless of the dose or route of administration.
3. To appreciate that the PTT value does not correlate with bleeding complications in patients receiving therapeutic anticoagulation who do not have identifiable comorbidities.
4. To appreciate that early inadequate anticoagulation (sub-therapeutic PTT) increases the risk of recurrent venous thrombosis 15x.
5. To appreciate that warfarin compounds can be started immediately after the heparin is therapeutic.
6. To appreciate that continuous IV heparin is associated with a better therapeutic outcome and fewer bleeding complications than bolus IV or high dose subcutaneous injection.
7. To appreciate that heparin induced thrombocytopenia (HIT) occurs in 4-6% of patients given unfractionated heparin, and is not dose related.
8. To understand that there are two types of HIT, immediate onset and delayed onset (5-10 days), with platelet counts falling to 40% of baseline or less, or absolute platelet counts < 150,000.
9. To understand that HIT is a result of an IgG-Ab to the platelet membrane causing platelet aggregation.
10. To understand that thrombocytopenia in the presence of heparin induced antibodies is associated with an extremely high risk of thrombotic complications, whereas a patient who is antibody positive but does not drop their platelet count does not have an increased risk of thrombotic complications.
11. Once HIT is diagnosed (or suspected), all heparin must be avoided.
12. To understand that HIT occurs significantly less in patients receiving low molecular weight heparin compared to unfractionated heparin.
13. To appreciate that warfarin compounds can produce skin necrosis when given to patients who have a heterozygote protein C deficiency.
14. To understand that in patients with warfarin induced skin necrosis, warfarin compounds cause protein C to drop thereby increasing activity of factors Va and VIIIa, thereby causing increased coagulation.
15. To appreciate that at least six months of oral anticoagulation is required for first time proximal DVT to adequately avoid recurrences.
16. To understand that low molecular weight heparin, given in weight adjusted dose subcutaneously once daily, is at least as effective and possibly more effective as IV unfractionated heparin for acute DVT.
17. To appreciate that the action of low molecular weight heparin is reduction of factor Xa activity, and it does not affect the PTT.
18. To understand the mechanism of action and the relative merits/risks of fibrinolytic therapy for acute deep venous thrombosis.
19. To appreciate that systemic fibrinolytic therapy for iliofemoral DVT is likely to fail, and that catheter-directed intra-thrombus thrombolysis is preferred.
20. To appreciate that venous thrombectomy is an effective option for patients with acute iliofemoral DVT.
21. To appreciate that the current operative technique of venous thrombectomy has improved compared to the early procedures.
22. To understand that a complete preop evaluation of the contralateral iliofemoral system and vena cava is important prior to venous thrombectomy.
23. To appreciate that an on-table completion phlebogram and correction of an underlying iliac vein stenosis is crucial to successful venous thrombectomy.
24. To appreciate that vena caval filters do not "treat acute DVT", they prevent large pulmonary emboli from occurring.
25. To appreciate that a Bird's nest filter is indicated for patients with a large vena cava.
26. To know reasons why warfarin should be avoided during pregnancy.
27. To understand that the indications for vena caval filters during pregnancy are the same as the non-pregnant patients, however, the filter should be placed in the supra-renal position.
28. To understand that those pregnant patients requiring heparin prophylaxis increase their heparin requirements during the second and third trimester.

V. Pulmonary Embolism
Goals:
1. To appreciate that pulmonary emboli occur without clinical warning in the majority of the patients.
2. To appreciate that the majority of the deaths from pulmonary emboli occur within 1-2 hours of the embolic event, and that untreated pulmonary embolism is associated with a 30% mortality.
3. To understand the typical signs/symptoms and the usual chest x-ray, blood gas and EKG findings in patients with large pulmonary emboli.
4. To appreciate the proper use of ventilation perfusion lung scan, and understand the valuable integration of predictive values based upon clinical suspicion of PE. (PIOPED data)
5. To appreciate that thrombolysis of pulmonary emboli results in better cardiopulmonary hemodynamic parameters than standard anticoagulation.
6. To understand the indications for operative pulmonary embolectomy and to appreciate that patients considered for a pulmonary embolectomy should be offered high dose fibrinolytic therapy first (if there are no contraindications).

References
13. **Chronic Venous Insufficiency**
John F. Eidt, M.D., Dhiraj M. Shah, M.D., James N. Thomas, M.D.

I. Anatomy & Pathophysiology
1. To review normal venous anatomy: superficial, deep and perforating veins, greater saphenous vein (GSV), lesser saphenous vein (LSV), femoral, popliteal & tibial vessels.
2. To describe the major venous anatomic variants of clinical importance including left sided inferior vena cava, retroaortic and circumaortic left renal vein.
3. To understand normal venous hemodynamics and the derangements associated with chronic venous insufficiency.
4. To review the epidemiology of chronic venous insufficiency.
5. To understand the function of normal venous endothelium and its alteration in chronic venous insufficiency (e.g. production of prostacyclin, plasminogen activator, heparans and thrombomodulin).
6. To outline the major risk factors for venous thrombosis including acquired and hereditary hypercoagulable conditions.
7. To review the postulated consequences of venous thrombosis on normal venous patency and valve function.
8. To explain the relationship between acute deep vein thrombosis and the eventual development of chronic venous insufficiency.
9. To define:
   - Chronic venous insufficiency
   - Varicose veins
   - Perforating veins
   - Telangiectasia
   - Sclerotherapy
   - Lipodermatosclerosis
   - Venous claudication
   - Phlelegmasia cerulea dolens
10. To review the postulated chain of events that leads to lipodermatosclerosis and venous ulceration.
11. To understand that chronic venous disease is defined as an abnormally functioning venous system caused by venous valvular incompetence with or without venous outflow obstruction which may affect the superficial venous system, the deep venous system or both.
12. To understand that the term post-thrombotic may be used if the patient has experienced an objectively documented episode of DVT. The term postphlebitic syndrome should not be used because this implies the presence of an inflammatory component that is infrequently confirmed.
13. To review the role of inflammatory cells in the development of venous stasis ulcers.
14. To understand that chronic venous insufficiency can lead to significant morbidity and may be disabling.
15. To differentiate congenital from acquired forms of venous insufficiency.

II. Diagnostic Evaluation
1. To review the “CEAP” classification system of chronic venous insufficiency: Clinical condition, Etiology, Anatomic distribution and Pathophysiology.
2. To understand and differentiate the three etiologic categories of venous dysfunction: congenital, primary (acquired, undetermined cause) and secondary (acquired, e.g. post-thrombotic or post traumatic).
3. To differentiate the clinical features of superficial venous insufficiency from deep vein (or combined) insufficiency.
4. To review the noninvasive and invasive evaluation of the venous system including ascending & descending venography, photoplethysmography, air plethysmography, and duplex scanning.
5. To describe the characteristics of venous stasis ulcers and differentiate from other types of ulcers including arterial, neuropathic, malignant, infectious and inflammatory (vasculitis).
6. To differentiate stasis dermatitis from other causes of dermatitis in the lower leg.
III. Treatment
1. To describe the types of available therapy for superficial venous insufficiency (varicose veins) including elastic stockings, elevation, sclerotherapy, laser treatment, stab evulsion, stripping.
2. To review the strengths and drawbacks of agents used in sclerotherapy including hypertonic saline, sodium tetradecyl sulfate, polidocanol etc.
3. To recognize the relative risks and benefits associated with treatment of varicose veins including DVT, infection, skin slough, etc.
4. To define the principles of non-operative management of lower extremity chronic venous insufficiency: ambulation, elevation, elastic support.
5. To review the technique of ambulatory phlebectomy (microstab evulsion) for varicose veins including the use of tumescent (large volume, low strength) local anesthesia.
6. To review the indications for surgery and surgical options in the treatment of chronic venous insufficiency, varicose veins, venous obstruction and stasis ulceration.
7. To describe the procedures for treatment of valve reflux including valvuloplasty, vein valve autotransplantation and vein segment transposition.
8. To discuss the relative risks and merits of procedures designed to decrease the degree of valve reflux.
9. To describe the procedures designed to treat venous outflow obstruction including autogenous or prosthetic bypass and venous disobliteration.
10. To describe the non-operative management of venous stasis ulcers including UNNA Boot, etc.
11. To review the operative procedures for venous ulceration including subfascial ligation.
12. To outline the technical features of 1) endoscopic subfascial ligation of incompetent ankle perforating veins in the treatment of chronic venous insufficiency and 2) endoscopic excision of varicose veins.
13. To describe the proposed pharmaceutical treatment of venous stasis ulcers: pentoxyphyllines, prostaglandins, antibiotics, growth factors, etc.

References
14. **Lymphedema**
Louis M. Messina, M.D., Robert B. Smith, M.D.

**I. Anatomy**
1. To know the anatomy of the adult lymphatic system from the level of the terminal lymphatics to the cisterna chyli
2. To know the microscopic anatomy of the lymphatic capillaries and conducting lymph vessels and specifically how they differ from veins and arteries.
3. To understand the physiological determinants of lymph flow, including intrinsic contractility of lymph vessels, increased interstitial pressure, muscular activity, arterial pressure, respiratory pressure, and gravity.
4. To know the major differences that distinguish the physiology of the lymphatic system from the venous system.
5. To know the major purposes of the lymphatic system, including transport of interstitial fluid and macromolecular proteins lost from capillaries, bacterial and fungal infections, foreign material.
6. To know the classification of causes of lymphedema, including:
   A. Primary lymphedema, Congenital (onset before one year of age)
      1. Non-familial
      2. Familial (Milroy’s Disease)
   B. Primary lymphedema, Praecox (onset 1 to 35 years of age)
      1. Non-familial
      2. Familial (Meige Disease)
   C. Primary lymphedema, Tarda (onset after 35 years of age)
   D. Secondary lymphedema, including filariasis, lymph node excision and radiation, tumor invasion, infection, and trauma
      1. To understand the functional classification of lymphedema based on the underlying lymphatic anatomy as determined by lymphangiography.
      2. To understand how lymphedema develops the compensatory mechanisms that develop in response to increased interstitial pressure, and the tissue effects of chronic lymphatic obstruction including impaired immune cell trafficking, lymphatic obstruction, and chronic intestinal inflammation.
      3. To understand the secondary consequences of long-standing lymphedema: infection, fibrosis, and neoplasia.
      4. To understand the functional and anatomical abnormalities that cause chylous disorders.
      5. To understand the consequences of the loss of chyle into body cavities or through a chylocutaneous fistula.

**II. Diagnosis of Lymphedema**
1. To understand classic clinical classifications of lymphedema based on etiology (primary vs secondary), genetics (familial vs sporadic), and time of onset.
2. To understand the history and physical findings which enable the clinician to identify the cause and site of lymphatic obstruction.
3. To understand pattern of pain, edema, and skin changes that distinguish lymphedema from other causes of extremity edema.
4. To understand the clinical presentation of complications of chronic lymphedema including infection (fungal and bacterial) and malignancy.
5. To understand the nutritional and immunological consequences of chronic lymphangiecitesia with protein-losing nephropathy, chylous ascites, or chylothorax.
6. To understand the accuracy and limitations of the most frequent noninvasive imaging modalities used to evaluate lymphatic disease: lymphoscintigraphy, computed tomography, and magnetic resonance imaging.
7. To understand the technique of lymphoscintigraphy, the features of a normal lymphoscintogram and the
IV. Lymphedema

8. To understand the indications, techniques, interpretation and complications of lymphangiograms.

III. Management of Chronic Lymphedema

1. To understand the techniques of non-operative management of primary and secondary lymphedema.
2. To know the mechanisms of action and effectiveness/ineffectiveness of pharmacologic agents such as diuretics, benzopyrones, and steroids in the treatment of lymphedema.
3. To understand the mechanical techniques to reduce a limb swelling
   A. To understand the technique of limb elevation.
   B. To understand the technique, advantages, and disadvantages of manual lymphatic drainage.
   C. To understand the technique of intermittent pneumatic compression, including pressure, ratio of compression/decompression, duration of therapy.
   D. To understand the technique of intermittent, non-pneumatic high pressure compression
   E. To know the role of antibiotics in the treatment and prophylaxis of recurrent cellulitis in patients with chronic lymphedema.
   1. To know the techniques for maintenance of limb size including elastic and non-elastic support.
   2. To know the indications for surgical management of chronic lymphedema
   3. To understand the technique, complication rate, and effectiveness of excisional procedures including the Charles procedure, Thompson’s buried dermal flap, suction curettage, and Sistrunk procedures.
   4. To know the indications, technique, complications rate, and outcome of direct lymphatic reconstruction such as lymphovenous anastomosis including lymphnodal-venous and lymphvenous procedures.
   5. To know the indications, technique, complication rate, and outcome of lymphatic flooding.
   6. To know indication, technique, complications rate of indirect lymphatic reconstructions such as the mesenteric bridge operation, omental flap, and autotransplantation of free lymphatic flap.
      A. To know the indications, technique, complications, and outcome of procedures for primary chylous disorders.

References
15. **Extremity Amputation**  
Roger T. Gregory, M.D., G. Patrick Clagett, M.D., H. Fabio Giron, M.D.

I. **Anatomy and Pathophysiology**  
1. To learn the normal anatomy of the extremities including all muscles, nerves, vessels, and bones.  
2. To understand the various pathophysiologic conditions which leads to the need for an extremity amputation.

II. **Diagnostic Evaluation**  
**Clinical Indications for Amputation**  
1. To understand when acute ischemia is irretrievable.  
2. To understand when chronic ischemia is unacceptable.  
3. To define when amputation offers improved quality of life.  
4. To be able to recognize when an ischemic limb is a threat to survival.  
5. To understand when diabetic foot infections may necessitate amputation despite adequate circulation - the concept of “life threatening infection.”  
6. To define the role of osteomyelitis in determining the need and type of amputation.

**Determining the Level of Amputation**  
1. To understand the importance of proper amputation level selection.  
2. To define the methods of determining amputation level by clinical criteria.  
3. To define methods of determining amputation level by noninvasive methods.  
4. To understand the limits of angiography.

III. **Treatment**  
**Lower Extremity Amputation Techniques**  
1. To understand the basic techniques for toe amputation, ray amputation, transmetatarsal amputation, below knee amputation, above knee amputation and upper extremity amputation.  
2. To understand situations when “unusual” amputations may be appropriate, such as Choparts, Lisfranc, Symes, through-knee, hip disarticulation, hemipelvectomy, and, even, hemicorporectomy.  
3. To understand the causes of stump failure, including technical problems, inadequate skin and muscle perfusion, hematoma, inadequate flaps, pressure necrosis from transected bone, and infection.

**Postamputation Care, Prosthetic Management and Rehabilitation**  
1. To define the differences offered by soft versus rigid dressings.  
2. To understand the importance of early mobilization.  
3. To achieve a basic understanding of lower extremity prosthetic devices, specifically the pros and cons of immediate versus delayed prosthesis.  
4. To understand how amputation technique can impact upon prosthetic application and subsequent rehabilitation.  
5. To define goals of rehabilitation with individual capabilities.  
6. To understand the importance of communication and participation of a multiple disciplined team approach to the amputee and the special problems presented.  
7. To understand the consequences of flexion contracture following amputation.  
8. To be able to recognize and manage phantom pain syndromes.

**References** (*excellent references)*  
**Texts**  

**Journal Articles**
16. Diagnostic Techniques
David S. Sumner, M.D., John Blebea, M.D.

Goals
I. History
1. To understand the essential components of a comprehensive vascular history.
2. To recognize symptoms relevant to vascular disease, identify salient points and understand their significance.
3. To use the information to formulate an initial diagnosis and to evaluate the severity of the likely disease process.
4. To identify confounding symptoms of similar nature produced by non-vascular diseases.
5. To obtain historical information pertinent to the evaluation of patients for operation or information that would militate against operative intervention or dictate the choice of therapy.

II. Physical Examination
1. To understand the significance of observational signs, such as skin color and texture, swelling, gangrene, and ulcers.
2. To detect and evaluate peripheral pulses, bruits, thrills, skin temperature, edema, tissue turgor, and vascular dimensions.
3. To develop the skills necessary to palpate the abdomen, neck, and extremities in order to localize sites of tenderness and to recognize the presence of masses and abnormal pulsations.
4. To be capable of performing basic neurological evaluations.
5. To interpret physical findings, understand how they contribute to the diagnosis, recognize their limitations, and be aware of other diseases that might mimic the findings.

III. Noninvasive Tests
1. To be familiar with commonly used noninvasive instruments and modalities, such as Doppler ultrasound, duplex and color-flow scanning, B-mode imaging, plethysmography (air, mercury, and impedance), magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and computerized X-ray tomography (CT), and to understand the basic principles involved in their design and operation.
2. To be familiar with noninvasive pressure measurements (including ankle/brachial indices, segmental pressures, digital pressures), arterial and venous velocity tracings, Doppler frequency spectral analysis, segmental and digital plethysmography, transcutaneous oxygen tension measurements (TcPO$_2$), venous outflow plethysmography, calf venous air-plethysmography (Nicolaides method) and to understand the hemodynamic principles underlying exercise testing (treadmill walking and claudication times, post-exercise ankle pressure) and reactive hyperemia.
3. To understand the physiologic basis of these tests and their limitations, know when to order noninvasive tests, which to select and how to interpret the results.
4. To perform simple noninvasive assessments (such as Doppler venous and arterial surveys and measurement of ABIs) and be able to interpret duplex scans, MRIs, MRAs, and CT scans.

IV. Invasive Diagnostic Methods
1. To be highly skilled in the interpretation of angiograms of all arterial and venous segments.
2. To understand the limitations and inherent risks of angiography, be aware of sources of error, and know how to minimize complications.
3. To be adept at obtaining and interpreting intraoperative arteriograms and, whenever possible, to acquire the skills necessary to perform percutaneous arteriography, including catheter manipulation techniques required for selective visualization of visceral and brachiocephalic vessels.
4. To be familiar with the intraoperative use of Doppler and duplex surveys in order to answer specific questions (location and patency of vessels, stenotic sites) and to detect technical errors at the completion of
the reconstruction (residual valves, arteriovenous fistulas, thrombi, anastomotic problems).
5. To be familiar with the intraoperative applications of the angioscope.
6. To perform intraoperative and preoperative percutaneous arterial and venous pressure measurements involving the use of pressure transducers.
7. To have some knowledge of other less frequently performed tests, such as intravascular ultrasound, isotope clearance studies and uptake tests, and scintillation scans.

Site Specific Goals

I. Lower Extremity Arterial Disease
1. To identify the symptoms of intermittent claudication and differentiate them from those of orthopedic or neurological conditions.
2. To recognize symptoms of severe ischemia, (such as rest pain, tissue loss, ulcers, and gangrene); differentiate these symptoms from those of diabetic neuropathy, neurologic, venous, infectious, and other problems; and determine the relative importance of several etiologies when more than one is present.
3. To recognize and differentiate the symptoms and signs of acute arterial occlusion (pain, pallor, numbness, and motor dysfunction) from those of chronic arterial occlusive disease; to assess the urgency of the condition and the threat to limb loss; and to distinguish findings suggestive of embolic occlusion from those of arterial thrombosis.
4. To understand the contribution of noninvasive tests (ABI, plethysmography, duplex surveys, treadmill exercise, and reactive hyperemia) to the diagnosis and know when arteriography, MRA, or other more complex tests are required.
5. Based on the history and physical examination, together with the results of invasive and noninvasive tests, to formulate an accurate diagnosis of arterial disease, identify the location and extent of the obstructive process, assess its severity, and determine the need for and the urgency of interventional therapy.

II. Extracranial Cerebrovascular Disease
1. To recognize and evaluate the symptoms and signs of transient hemispheric and nonhemispheric neurologic events and to differentiate them from the symptoms and signs of permanent neurologic damage (stroke) or peripheral neuropathy.
2. To decide, based on their natural history and pathophysiologic behavior, which events require immediate attention.
3. To understand the indications for common noninvasive tests (such as duplex or color-flow scanning), how they may contribute, what their limitations are, and how they are to be interpreted; and to know when to obtain and how to interpret less commonly performed tests, such as transcranial Doppler studies or oculopneumoplethysmography.
4. To know when to order (or not to order) cerebral arteriography, how to read extracranial and intracranial views, how to measure the degree of stenosis, and how to use the findings to select the proper therapeutic approach.
5. To know when to select MRA as an alternative diagnostic method and what its comparative accuracy is compared to arteriography or duplex scanning.
6. To be able to read and interpret CT and MRI scans of the brain, know when to order these studies and how the results influence diagnosis and the need for therapeutic intervention.
7. In asymptomatic patients, to assess cervical bruits, understand their significance, and know which patients without specific signs have a high propensity for extracranial cerebrovascular disease and are likely to benefit from noninvasive diagnostic screening and possible therapeutic intervention.
8. To understand the role of duplex scanning in the follow-up of nonoperated or operated patients with known cerebrovascular disease (to detect recurrent disease or disease progression).

III. Brachiocephalic and Upper Extremity Arterial Disease
1. To recognize the signs and symptoms of brachiocephalic disease, including those of hemispheric ischemia,
vertebrobasilar ischemia, and arm claudication and ischemia.
2. To understand the role that brachial, segmental, and digital pressures play in screening for disease and the roles that duplex scanning, arteriography, and MRA play in establishing the diagnosis.

IV. Aneurysmal Disease
1. To recognize and interpret the signs and symptoms of abdominal aortic, iliac, femoral, popliteal, visceral, thoracic, carotid, and brachiocephalic aneurysms.
2. To be skilled in the palpation of the abdomen, extremities, and neck in order to recognize pulsatile masses, assess their dimensions, and differentiate those likely to be aneurysms from arterial tortuosity, tumors, or other nonvascular masses.
3. To recognize signs of impending or actual rupture including tenderness, ecchymoses, shock, or other evidence of acute blood loss.
4. To determine the urgency of operative intervention, and decide when ultrasonic, CT, or MRI confirmation is necessary.
5. To be acutely aware of the signs of complications, such as aortic-enteric fistula and high-output cardiac failure due to aorto-caval fistulae.
6. To know the indications for arteriography (or MRA) and how to interpret these studies.
7. To be alert to the indirect signs of aneurysms, such as unexplained embolic phenomena (blue toes or fingers) or sudden ischemia due to acute thrombosis or dissections.

V. Visceral Arterial Disease
1. To be familiar with the symptoms of acute visceral arterial occlusion and with the post-prandial pain patterns and weight loss associated with chronic visceral ischemia.
2. To be alert to conditions (such as atrial fibrillation, recent myocardial infarction, arterial dissections) that might lead to acute occlusion of mesenteric arteries.
3. To recognize conditions (such as congestive heart failure) that predispose to nonocclusive mesenteric ischemia.
4. To interpret visceral angiograms and know when these are needed.
5. To understand the role and limitations of duplex scanning in the diagnosis of visceral arterial stenosis.

VI. Renal Arterial Disease
1. To recognize the signs and symptoms of renal arterial occlusive disease, as manifested by the onset and severity of hypertension, and be able to determine which patients require further workup.
2. To be familiar with the diagnostic roles of selective renal vein renins, isotope clearance tests, IVP, duplex scanning, and arteriography and know the limitations and predictive value of these tests.

VII. Arteriovenous Fistula
1. To be cognizant of the systemic manifestations of large arteriovenous fistulas, including tachycardia, Branham's sign, and high-output cardiac failure and be able to differentiate between acquired and congenital fistulas.
2. To understand the diagnostic significance of a history of penetrating trauma, fractures, back surgery, and vascular catheterization and know the significance of signs, such as birthmarks, limb hypertrophy, unilateral varicose veins, vascular malformations, bruits, and thrills.
3. To be aware of the role that noninvasive pressure measurements and duplex scanning have in establishing a diagnosis, know when to order CT scans, MRI, MRA, or arteriography, and be able to interpret the results.
4. To distinguish between congenital arteriovenous fistulas and primary venous malformations.

VIII. Vasospastic Disease
1. To recognize and evaluate the symptoms of episodic digital ischemia provoked by cold exposure (Raynaud's phenomenon) and to be aware of the manifestations of vasospasm, such as changes in skin color
and temperature.
2. To identify signs of underlying autoimmune disease, such as digital atrophy, ulceration, or gangrene and other skin changes.
3. To be aware of the role that noninvasive tests (Doppler surveys, duplex scans, digital pressure measurements, plethysmographic studies, and skin temperature recordings) play in distinguishing purely vasospastic disease from vasospasm superimposed on fixed digital arterial stenoses or occlusions.
4. To know when arteriography is indicated and how to interpret the findings.

IX. Acute Venous Thrombosis
1. To recognize the signs and symptoms of acute deep venous thrombosis (DVT) and differentiate them from the signs and symptoms of cellulitis, muscle tears, superficial venous thrombosis, arterial obstruction, and a host of other causes of unilateral limb swelling, edema, pain, and cyanosis.
2. To be aware of the significance of factors predisposing to DVT, such as recent trauma, orthopedic or major abdominal surgery, malignancy or chronic illness, pregnancy, airplane or bus trips, and hypercoagulability.
3. To understand the limitations of the history and physical examination and be aware of the critical role that noninvasive testing (primarily duplex scanning and to a declining extent, hand-held Doppler and impedance plethysmography) now plays in the diagnosis of this disease.
4. To know when phlebograms, magnetic resonance studies, or CT scans are indicated and how to interpret the results.
5. To be aware of the indications for screening asymptomatic high-risk patients for occult DVT and know the limitations of the noninvasive methods used for this purpose.

X. Chronic Venous Insufficiency
1. To know the symptoms and signs of varicose veins, chronic venous obstruction, and deep venous incompetence and be able to differentiate these diseases from lymphedema, acute DVT, arteriovenous malformations, and arterial disease.
2. To recognize and evaluate the cutaneous manifestations of chronic venous insufficiency, including lipodermatosclerosis, pigmentation, dermatitis, and ulceration.
3. To know when objective testing is required to establish the diagnosis and understand how duplex scanning may contribute to the anatomic assessment by identifying the sites and distribution of chronic venous obstruction and incompetent venous valves; how air plethysmographic, photoplethysmographic, and other physiologic tests (such as ambulatory venous pressure measurements) may assist in the evaluation and assessment of the severity of physiologic aberrations; and when to order and how to evaluate ascending and descending phlebograms.

XI. Lymphedema
1. To be familiar with the historical aspects of lymphedema, noting the time of onset and the presence of previous or coexisting infections, injuries, radiation, or malignancy.
2. To be aware of the significance of the location of swelling, the type of edema (pitting or woody), the presence of cutaneous lichenification, and associated cellulitis.
3. To understand the diagnostic roles of lymphangiography and scintillation scans and when to order and how to interpret these studies.
4. To differentiate between primary and secondary lymphedema and distinguish the various forms of lymphedema from swelling due to chronic venous insufficiency.

XII. Trauma
1. To understand the importance of obtaining a history of the injury (whether it was due to blunt or penetrating trauma, gun-shot of knife); of an expeditious physical examination noting the location of the injury (entry and exit points, multiple sites or localized), the presence of external hemorrhage, hematoma,
ecchymoses, or shock, of assessing peripheral pulses, neurologic status, and respiratory compromise, and of identifying associated skeletal or visceral injuries.

2. To know when to obtain Doppler studies, peripheral pressure measurements, duplex scans, transesophageal echo studies, compartmental pressures, CT scans, X-rays, and arteriography.

XIII. Amputations
1. To recognize the need for amputation and to predict the optimum level based on a history of previous revascularization attempts, etiology of vascular obstruction, the presence of infection, diabetes, or coagulation disorders, location and severity of pain, extent of ulcers or gangrene, presence or absence of pulses, the appearance and temperature of the skin, capillary refill, and overall medical status.
2. To understand the limitations and advantages of using objective tests such as TcPO$_2$ measurement, isotope clearance, and ankle, segmental, digital, and skin pressures to select the site of amputation.

References

General

Peripheral Arterial

Arteriography, MRI

Carotid

**Abdominal, Visceral**


**Venous**

Emerging Technologies
Thomas F. Panetta, M.D., Teruo Matsumoto, M.D., Rodney A. White, M.D.

I. General
1. To understand the basic principles of emerging technologies in vascular and endovascular surgery.
2. To develop a working knowledge of the equipment, techniques, technical problems, troubleshooting and recovery techniques.
3. To understand the physical properties of devices including but not limited to wires, catheters, balloons, coils, stents, stent-grafts, filters and delivery systems. To understand the physical properties, basic engineering and evolution of devices as they relate to their clinical applications, implantation, biocompatibility, tissue reactions and interactions, graft-metallurgical interactions, wound healing, limitations and overall use in the treatment of vascular disease.
4. To understand the indications, applications, complications, management and results of imaging modalities, basic techniques, newly developed techniques and implantable devices used to treat vascular disease.

II. Imaging Modalities
1. To understand radiation physics, safety, risks, cellular effects, somatic effects, dose responses, monitoring, shielding and variations in x-ray equipment as they relate to both patients and personnel including preventative measures for safety.
2. To understand basic principles and equipment used for fluoroscopy and arteriography. To obtain a working knowledge of contrast media, road-mapping, imaging techniques, measurement techniques, parallax, hand and power injection techniques and film sequencing.
3. To understand the basic principles of intravascular ultrasonography (IVUS). To obtain a working knowledge of B-mode imaging, transducers and catheters.
4. To understand the basic principles of angioscopy. To obtain a working knowledge of endoscopes and fiberoptic technology, imaging and irrigating equipment, and channel instrumentation.
5. To understand the techniques used for preoperative, intraoperative and postoperative imaging, measurements and evaluation of endovascular techniques including ultrasonography, magnetic resonance imaging, computerized axial tomography including helical techniques with 3 dimensional reconstructions and angiography.
6. To understand the accuracy, utility, limitations and clinical importance of each modality.

III. Basic Techniques
1. To obtain a working knowledge of basic endovascular techniques. To understand individual techniques and obtain a knowledge base for standard and emerging technologies.
2. To understand the proper use of needle, catheter, guidewire, dilator and introducer techniques used to gain access to the vascular system and perform vascular interventions.
3. To understand the techniques and mechanisms of angioplasty and atherectomy.
4. To obtain a working knowledge of pharmacological and mechanical methods of thrombolysis.

IV. Emerging Technologies
1. To obtain a working knowledge of self expanding and balloon expandable intravascular stents. To understand delivery techniques, rationale for use and retrieval/recovery techniques.
2. To understand the various types and uses of occlusion techniques including sclerosing agents and occlusion devices. To obtain a working knowledge of coils, temporary and permanent occlusion balloons, and the variety of covered stent occluding devices.
3. To understand and obtain a working knowledge of endovascular grafts, covered stents and stent-grafts for the treatment of vascular disease. This includes the variety of delivery systems, attachment devices, covered stents, and stent-graft combinations and devices. The values and limitations of each of the available and potentially available devices should be understood.
4. To have a working knowledge of adjunctive interventional procedures required as retrieval, recovery or “bail out” procedures in endovascular surgery including endovascular and open techniques.
5. To understand laparoscopic and laparoscopically assisted vascular techniques for both arterial, venous and adjunctive vascular procedures.
6. To understand the role of brachytherapy in preventing intimal hyperplasia, both as an independent modality or in combination with metallic devices.
7. To understand and have a working knowledge of venous filters and venous devices.

V. Clinical Applications
1. To understand and have a working knowledge of endovascular and interventional techniques utilizing percutaneous and surgical access for the diagnosis, management and treatment of traumatic arterial and venous injuries.
2. To understand and have a working knowledge of endovascular and interventional techniques utilizing percutaneous and surgical access for the diagnosis, management and treatment of arterial occlusive disease.
3. To understand and have a working knowledge of endovascular and interventional techniques utilizing percutaneous and surgical access for the diagnosis, management and treatment of aneurysmal disease.
4. To understand and have a working knowledge of endovascular and interventional techniques utilizing percutaneous and surgical access for the diagnosis, management and treatment of cerebrovascular disease.
5. To understand and have a working knowledge of endovascular and interventional techniques utilizing percutaneous and surgical access for the diagnosis, management and treatment of venous disease including arteriovenous malformations.
6. To understand the pathophysiology and management of intimal hyperplasia and recurrent disease after endovascular intervention, endovascular graft placement and insertion of an implantable device.
7. To understand the treatment of acute and chronic complications of endovascular techniques and devices. To understand the pathophysiology and management of arterial injuries, endoleaks, migration, embolization, delivery system failures and attachment device failures.
8. To have a working knowledge of recovery, retrieval and “bail out” procedures for endovascular procedures. This includes both catheter based and surgical procedures.

References
18. **Risk Stratification and Risk Factors**  
Bruce S. Cutler, M.D., William C. Mackey, M.D.

I. Cardiac Disease  
1. Recognize the frequent association of coronary artery and peripheral vascular disease.  
2. Understand the risk factors predictive of perioperative myocardial infarction or cardiac death.  
3. Be able to quote basic statistics regarding the frequency of severe CAD in patients with symptomatic peripheral vascular disease.  
4. Be familiar with the early and late cardiac mortality figures following major vascular surgery.

II. Anatomy and Pathophysiology  
1. Describe normal coronary artery anatomy  
2. Understand the clinical significance of chronic stable angina, unstable angina, recent and remote myocardial infarction and congestive heart failure  
3. Understand how an imbalance of myocardial oxygen supply and demand may lead to myocardial ischemia  
4. Describe those factors that may lead to an increased demand for myocardial oxygen, and/or a decreased supply that will contribute to myocardial ischemia.  
5. Understand the clinical and histological difference between a subendocardial and transmural infarction.  
6. Understand the effects of general and regional anesthesia on myocardial oxygen demand and myocardial ischemia.  
7. Understand the most important factors present intraoperatively and in the post-operative period that contribute to myocardial ischemia.

III. Diagnosis  
1. Understand the signs and symptoms of chronic stable angina, unstable angina, myocardial infarction and congestive heart failure.  
2. Know the risks of operation in a patient with a recent myocardial infarction, unstable angina, or poorly compensated congestive heart failure.  
3. Be familiar with the currently used methods for screening for coronary artery disease, and their limitations. (e.g. Dipyridamole thallium scanning, Exercise testing, Dobutamine stress echo, ambulatory Holter monitoring)  
4. Know which patients should undergo a preoperative test for coronary artery disease  
5. Know how to interpret the results of thallium scans  
6. Know what further evaluation a patient with a positive study should have.  
7. Know which patients should have coronary angiography prior to vascular surgery.  
8. Understand that the magnitude of the operation should be tailored to the severity of the patients cardiac risk. Know when to employ an extra anatomic, or limited procedure instead of an intra-abdominal operation.  
9. Understand when, during the course of a vascular operation and subsequent recovery, a patient is most likely to suffer a myocardial infarction.

IV. Treatment  
1. Recognize that most patients with even severe CAD can survive a major vascular operation, but they should have close postoperative cardiology follow up and subsequent consideration for coronary revascularization for the best long term survival.  
2. Know when CABG may be indicated to correct severe CAD prior to peripheral vascular surgery.  
3. Understand the indications for a combined CABG and CEA or AAA operation.  
4. Understand the reasons for controlling myocardial ischemia intraoperatively, and during recovery from a major vascular operation
5. Know how to detect and treat myocardial ischemia postoperatively
6. Know how to diagnose and treat common complications of myocardial infarction.

Pulmonary Disease

I. Introduction
1. Recognize that many of the same risk factors that accelerate the development of peripheral vascular disease, also cause the development of chronic obstructive pulmonary disease (COPD)
2. Understand that long operations, intra-abdominal and thoracic incisions, and poor left ventricular function increase the risk of pulmonary complications even in the absence of underlying COPD.
3. Understand that cardiac and other co-morbid conditions are more important in determining postoperative pulmonary complications than pre-existing pulmonary disease.

II. Diagnosis
1. Know the risk factors for pulmonary disease, including: history of tobacco use, chest wall deformities, industrial dust exposure, previous pulmonary resection, dyspnea on mild exertion, pulmonary hypertension, recurrent respiratory tract infections, bronchospasm, obesity, advanced age and hypercapnia or hypoxia at rest.
2. Understand the signs and symptoms of COPD.
3. Know what to look for in the physical examination of a patient with suspected pulmonary insufficiency.
4. Understand that clinical assessment is at least as accurate as routine preoperative pulmonary function tests in predicting which patients will have a postoperative pulmonary complication.
5. Understand that the primary benefit of preoperative pulmonary function studies is to make the diagnosis of pulmonary disease and as an aid in choosing between treatment alternatives.
6. Understand that there is no pulmonary function test, or index that can accurately predict that a patient will need prolonged postoperative mechanical ventilation.
7. Understand that general anesthesia interferes with pulmonary gas exchange and pulmonary defense mechanisms, particularly the mucociliary transport mechanism.
8. Know how to interpret the results of pulmonary function tests, and know which patients might benefit from the perioperative use of bronchodilators, antibiotics, inhalers etc.
9. Know which patients might benefit from a preoperative pulmonary or anesthesia consultation to help with the operative and postoperative management of a patient with known pulmonary insufficiency.

III. Treatment
1. Understand how to reduce the pulmonary risk of a vascular operation by the choice of operation and anesthesia.
2. Understand which pulmonary conditions may benefit from the perioperative use of steroids, bronchodilators, antibiotics and inhalers.
3. Understand the causes and treatment of the adult respiratory distress syndrome (ARDS).

References

Cardiac Disease
4. Hertzer NR, Beven EG, Young JR, et al. Coronary artery disease in peripheral vascular patients: A

Pulmonary
19. **Coagulation Disorders**  
Richard M. Green, M.D., Donald Silver, M.D.

I. **Heparin**  
1. To understand the role of antithrombin III and the dual action of heparin on thrombin (factor II) and factor Xa (IX a and XI a also).  
2. To be familiar with its half-life, routes of administration and its uses both in terms of prevention of thrombosis and in treatment for thrombotic conditions.  
3. To be familiar with the hematologic and nonhematologic complications.  
4. To understand the intraoperative use including monitoring techniques and reversal.  
5. To understand the mechanism of action and complications of protamine sulfate.

II. **Low Molecular Weight Heparin (LMWH)**  
1. To understand the rationale for its development and its advantages over unfractionated heparin.  
2. To understand the different mechanism of action as compared to unfractionated heparin.  
3. To understand why it can be used without monitoring.  
4. To understand why it is less hemorrhagic than unfractionated heparin.  
5. To understand the clinical applications particularly in the patient with heparin induced thrombocytopenia and prophylaxis for venous thrombosis.  
6. To understand the cost benefits of outpatient treatment of venous thrombosis.

III. **Heparin-induced Thrombocytopenia (HIT)**  
1. To understand the incidence of the syndrome in patients receiving heparin, the incidence of thrombotic complications and the mortality rate.  
2. To understand the risk factors associated with its development.  
3. To understand the differences between Type I and Type II HIT.  
4. To understand the diagnostic criteria necessary to make the diagnosis.  
5. To understand the pathophysiology of antibody formation.  
6. To understand the principles of management.  
7. To understand the limitations of the various diagnostic tests including platelet aggregation studies, the serotonin release assay and the PF4/heparin ELISA assay.  
8. To understand when further anticoagulation is indicated and what agents are available and under development.

IV. **Coumadin**  
1. To understand the mechanism of action including the roles of proteins C and S.  
2. To understand why heparin should be given for the first 3-4 days of coumadin treatment.  
3. To understand the medical conditions, foods and common drugs that affect coumadin’s anticoagulant activity.  
4. To understand how to minimize the complications of coumadin therapy.  
5. To understand the American College of Chest Physicians recommendations of appropriate INR levels. This should include a working knowledge of the conditions which require higher levels.  
6. To understand how and when to reverse anticoagulation in patients with and without hemorrhage.  
7. To understand how to manage patients requiring surgery.

V. **Antiplatelet therapy**  
1. To understand the role of platelets in primary and secondary hemostasis.  
2. To understand the role of platelets in pathologic thrombosis.
3. To understand the structure of the platelet and the function of each zone.
4. To understand the sequence of platelet activation including a knowledge of the glycoprotein complexes and the role of von Willebrand’s factor.
5. To understand the various platelet agonists and antagonists are their relative strengths.
6. To have a working knowledge of antiplatelet agents currently available and their mechanisms of action. This includes an understanding of the relative strengths of the antagonists: aspirin, ticlopidine, dextran, and dipyridamole.
7. To be familiar with the mechanism of action of some antiplatelet agents under investigation including von Willebrand factor monoclonal antibody, aurantricarboxylic acid, glycoprotein IIb/IIIa receptor antagonists, thromboxane/endoperoxide receptor inhibitors, prostaglandin E1, prostacyclin, proteolytically inactive mutant thrombins, and trapidil.

VI. The Detection of Abnormal Bleeding
1. To understand the relevant historical information in patients with a bleeding disorder.
2. To understand the coagulation studies that should be done routinely and those that should be done when a bleeding disorder is suspected.
3. To understand the importance of spontaneous ecchymosis and petechiae.
4. To understand the specific clinical presentation, genetic transmission and factor deficiency in hemophilia A, hemophilia B and von Willebrand's disease.
5. To understand the purpose of the bleeding time, the significance and common causes of an abnormal test.
6. To understand how to evaluate the intrinsic coagulation cascade and what drugs and factor deficiencies affect it.
7. To understand the significance of circulating inhibitors such as the lupus anticoagulant.
8. To understand how to evaluate the extrinsic coagulation cascade and what drugs or factor deficiencies affect it.
9. To have a working knowledge of the work-up and management of perioperative bleeding.

VII. The Use of Blood Products for Surgical Bleeding
1. To understand the risks of blood products and why transfusion practices have changed.
2. To understand the indications for red cell transfusions including a knowledge of the myocardial work requirements at hemoglobin levels of <7 g/dL, between 8 and 10 g/dL and >10 g/dL.
3. To understand the risks of and indications for administration of fresh-frozen plasma and cryoprecipitate.
4. To understand the indications for platelet transfusions in asymptomatic patients, patients who require a surgical procedure, and patients who have spontaneous bleeding.

VIII. Use of Desmopressin (DDAVP) in Vascular Surgery
1. To understand the properties, mechanism of action, and indications for its use.
2. To understand the phenomenon of tachyphylaxis including why it occurs and its significance.

IX. Hypercoagulability Syndromes
1. To understand the significant history, work-up and treatment for antithrombin III deficiency, protein C and S deficiency, factor V (Leiden) mutation [activated protein C resistance].
2. To understand the role of pregnancy and oral contraceptives on thrombosis.
3. To understand the need for thromboembolism prophylaxis in the various acute phase reactions such as trauma or operation.
4. To understand the significance of antiphospholipid antibodies including the types of patients at risk and the management implications.
5. To understand the role of screening in routine patients and high risk patients.
6. To understand the effects of coumadin, heparin and antiplatelet agents on lab measurements for hypercoagulability.
7. To understand the differential diagnosis and management of intraoperative clotting including the management of intimal injury, heparin induced thrombosis or antithrombin III deficiency.

X. Ancrod
1. To understand the derivation, mechanism of action, and uses.
2. To understand its effect on blood viscosity and its possible benefit in patients with arterial ischemia.
3. To understand the differences between the fibrinolytic activity of ancrod as compared to urokinase.
4. To understand the risks of too rapid defibrination.
5. To understand the risks of a lack of fibrin on wound healing.
6. To understand the management of ancrod induced bleeding complications.
20. **Diagnosis and Management of Miscellaneous Vasculogenic Problems**
Blair A. Keagy, M.D., Mark A. Farber, M.D., Sean D. O’Donnell, M.D., John J. Ricotta, M.D.

I. Anatomy and Pathophysiology
A. Raynaud’s Syndrome
1. To understand the epidemiology and pathophysiology surrounding Raynaud’s Syndrome.
2. To define the epidemiologic parameters involved in Raynaud’s Disease.
3. To define the physiologic mechanism occurring in Raynaud’s Phenomenon.
4. To define the criteria for obstructive Raynaud’s Syndrome.
5. To define the role of adrenergic receptors in the cause of Raynauds.

B. Neurogenic Thoracic Outlet Syndrome
1. To understand the anatomy of the thoracic outlet and the anatomic predisposition to developing TOS, including osseous abnormalities, and soft tissue abnormalities.
2. To understand the association of trauma, both direct and indirect, with the development of TOS.
3. To define the histological changes described in the scalene muscles of patients with TOS.

C. Causalgia/Reflex Sympathetic Dystrophy
1. To understand the pathogenesis of causalgia including that of artificial synapses, and the cycle of reflexes.
2. To define the clinical stages of Drucker, along with their characteristics and symptoms.

D. Vasculogenic Impotence
1. To describe the physiology involving erection including the blood supply, and innervation.
2. To define the differences associated with organic, psychogenic, neurogenic, and vasculogenic impotence.

E. Pediatric Vascular Disorders
1. To define and recognize the various congenital vascular lesions in children.
2. To recognize the problems associated with hemangiomas.
3. To understand renovascular hypertension in the pediatric population.
4. To understand the pathophysiology of renal vein thrombosis.

II. Diagnostic Evaluation
A. Raynaud’s Syndrome
1. To understand the clinical presentation of patients with Raynaud’s Syndrome. and their presenting symptoms.
2. To recognize the associated diseases.
3. To define the appropriate use of laboratory testing in the diagnosis of Raynauds, including the occlusive digital hypothermic challenge test, angiography and plethysmography.

B. Neurogenic Thoracic Outlet Syndrome
1. To define the demographic aspects of patients presenting with TOS.
2. To recognize the symptoms associated with the disease, including pain, parasthesias, and associated symptoms.
3. To recognize the musculoskeletal disorders that mimic TOS.
4. The utilization of diagnostic tests including the Tinel and Phalen test, Adson test, and the arm stress test, and recognize the physical findings suggestive of TOS along with their sensitivity and shortcomings.
5. To understand the role of ancillary diagnostic tests in the work-up of TOS including, but not confined to, chest radiographs.
6. To recognize the need for a complete neurologic examination in these patients.
7. To define the role of electrophysiology studies in the evaluation including ulnar nerve conduction velocities, electromyography, and somatosensory evoked potentials.

C. Causalgia/Reflex Sympathetic Dystrophy
1. To understand the presenting symptoms and differential diagnosis of causalgia.

D. Vasculogenic Impotence
1. To understand the role of non-invasive vascular testing.
2. To understand the role of indirect neurologic testing in impotence.
3. To recognize the usefulness of intracavernous papaverine injection and angiography in the diagnosis of impotence.

E. Pediatric Vascular Disorders
1. To recognize the clinical presentation of renal vein thrombosis in children and its diagnostic evaluation.

III. Treatment
A. Raynaud’s Syndrome
1. To recognize the medications that should be avoided in these patients.
2. To define the use of sympatholytic agents, and their replacement by calcium channel blockers.
3. To recognize the role of new therapies for treatment.

B. Neurogenic Thoracic Outlet Syndrome
1. To identify the role of conservative treatment for TOS.
2. To define the operative treatment of TOS including the choices for operative exposure, role of scalenectomy, concept of total decompression, and rationale for sparing the first rib.
3. To recognize the complications associated with the procedure including nerve, vascular, and lymphatic injuries.

C. Causalgia/Reflex Sympathetic Dystrophy
1. To define the timing of operative therapy for RSD, along with its results.
2. To identify the complications surrounding the procedure and the disease.

D. Vasculogenic Impotence
1. To understand the means of prevention of impotence during surgical procedures of the aorta and the results of appropriate revascularization.
2. To define the risk of impotence with associated vascular procedures.
3. To define the role of revascularization of the penis.

E. Pediatric Vascular Disorders
1. To define the treatment strategies for renal vein thrombosis.
2. To understand the treatment options in children with congenital vascular lesions.

References
21. **Non-Atherosclerotic Vascular Diseases**  
William L. Smead, M.D., R. Eugene Zierler, M.D.

I. Immune Arteritis  
1. To understand the basic pathologic mechanisms of vascular injury in the immune arteritis syndromes.  
2. To define a classification system for the vasculitides on the basis of clinical findings, pathology, and prognosis in a way that assists in the diagnosis and management of these patients, recognizing the considerable overlap that exists among these diseases. This classification system would include the systemic necrotizing vasculitides, the hypersensitivity vasculitides, giant cell arteritis, and a diverse miscellaneous group.  
3. To recognize the clinical problems which should alert the physician to consider a systemic vasculitis, particularly ischemic symptoms involving multiple organ systems in patients under 55 years of age, involving organs and limbs in distributions not typical for atherosclerosis, and occurring at an unusually accelerated pace.  
4. To understand the basic clinical laboratory tests useful in the diagnosis and management of vasculitis. Diagnostic tests range from routine evaluations to more specific tests for defining the immunophysiology. Management requires testing to define the current inflammatory status as well as specific measurements of organ function.  
5. To understand the role of arteriography and the characteristic findings in the arteritis syndromes of surgical significance.  
6. To recognize the role of tissue biopsy in establishing a firm diagnosis.  
7. To recognize the clinical presentation of polyarteritis nodosa, the laboratory and pathologic features of the disease which establish the diagnosis, and the complications of the disease with surgical significance.  
8. To be familiar with the small vessel complications of hypersensitivity angiitis, a large category of vasculitides with a wide variety of etiologies including infection, drug and chemical allergies, connective tissue diseases, neoplasm, Henoch-Schönlein purpura, serum sickness, cryoglobulinemia, and a large miscellaneous category represented by chronic active hepatitis, primary biliary cirrhosis, inflammatory bowel disease, and intestinal bypass surgery. To understand the medical and surgical management of ischemic complications of these disorders.  
9. To recognize the giant cell arteritis group of diseases which includes temporal arteritis and Takayasu-Onishi disease. To understand the distinctive arteriographic patterns of these disorders and the medical and surgical treatment strategies.  
10. To be familiar with the miscellaneous vasculitis syndromes of surgical significance, including Kawasaki’s syndrome, Behcet’s disease, Cogan’s syndrome, and Buerger’s disease.

II. Fibromuscular Dysplasia  
1. To understand the pathologic classification of fibromuscular dysplasia: intimal fibroplasia, medial fibroplasia, medial hyperplasia, and perimedial dysplasia.  
2. To recognize the vascular beds most frequently affected by this disorder (renal, cerebrovascular, mesenteric, and aortoiliac arteries) and the symptoms with which patients most frequently present.  
3. To recognize the arteriographic patterns distinguishing each of the types of fibromuscular disease from each other and atherosclerosis.  
4. To understand the natural history of fibromuscular disease in its various locations and its impact on clinical decision making.  
5. To understand the various treatment options available including endovascular techniques and surgical bypass.

III. Adventitial Cystic Disease
1. To understand this rare condition producing arterial stenosis or occlusion in young patients, its clinical presentation, arteriographic features, operative findings, and management options.

IV. Popliteal Intrapartment Syndromes
1. To understand the clinical presentation of this congenital anomaly predominantly affecting young men, its characteristic noninvasive vascular laboratory and arteriographic findings (provocative testing), and the available treatment alternatives.
2. To be familiar with the various anatomic variants which produce the abnormal relationship between the popliteal artery and the medial head of the gastrocnemius muscle.
3. To recognize the characteristic findings suggesting the adductor canal syndrome in which the junction of the superficial femoral and popliteal arteries is compressed by the tendinous insertion of the adductor magnus muscle at Hunter’s canal.

V. Compartment Syndromes
1. To understand the multiple etiologies of compartment syndromes which have in common the production of sufficient compartmental pressure to compromise blood flow to the tissues within it, conditions which decrease compartmental volume or increase compartmental content or provide excessive external pressure.
2. To recognize those clinical situations in which compartment syndrome is more likely to develop complicating vascular injury or disease: prolonged ischemia, coexistent shock, preoperative neurologic deficits, pre- or intraoperative edema, combined arterial and venous injury, or concomitant crush injury.
3. To recognize the symptoms and signs of elevated compartment pressure and the tests available to confirm the diagnosis.
4. To understand the indications for fasciotomy and the surgical techniques available.
5. To understand the medical management of established rhabdomyolysis.

VI. Congenital Arterial Conditions
1. To be familiar with the various types of abdominal coarctations and their clinical presentations and natural history.
2. To understand the role of arteriography in the diagnosis of the problem and the planning of surgical treatment of abdominal coarctation.
3. To be familiar with the surgical options for repair and renal revascularization in patients with abdominal coarctation.
4. To recognize the arteriographic findings in patients with a persistent sciatic artery and the potential surgical implications.

VII. Diseases of the Arterial Media
1. To understand the pathologic changes of cystic medial necrosis which result in the clinical problems of aortic dissection, spontaneous rupture, and aneurysm formation.
2. To recognize the classic abnormalities associated with Marfan’s syndrome and the typical cardiovascular complications.
3. To understand the natural history of Marfan’s syndrome and the management options available to treat these cardiovascular problems.
4. To recognize the characteristic abnormalities in patients with Ehlers-Danlos syndrome and the issues of surgical significance including aneurysm formation, dissection, and spontaneous rupture.
5. To be familiar with the vascular changes in patients with pseudoxanthoma elasticum, arterial stenosis/occlusion and hypertension.
6. To recognize the changes associated with arteria magna syndrome and the role of arteriography in diagnosis, treatment, and patient follow-up.
VIII. Errors in Homocysteine Metabolism
1. To understand the inborn error of metabolism that produces homocysteinuria and the associated multiple abnormalities including mental retardation, lens ectopia, rapidly progressive premature atherosclerosis, and thromboembolic disorders.
2. To be familiar with the heterozygous trait which results in homocysteinemia and premature atherosclerosis, potentially ameliorated by treatment with folic acid, pyridoxine, and vitamin B₁₂.

IX. Hyperviscosity Syndromes
1. To understand the myeloproliferative disorders and serum protein abnormalities that result in arterial or venous thromboembolism.

X. Arterial Infections
1. To recognize the symptoms and signs of arterial infections and the most common responsible pathogens.
2. To understand the etiologies of arterial infection including bacterial endocarditis, mycotic or infected aneurysms, drug abuse, iatrogenic contamination, and contiguity to adjacent infection.
3. To recognize the most effective techniques for obtaining positive cultures on which to base antibiotic treatment in patients with arterial infections.
4. To be familiar with the principles and treatment strategies for the management of arterial infection.

XI. Vasospastic Disorders
1. To understand the classification of cold sensitivity of the Raynaud type (Raynaud’s disease and Raynaud’s phenomenon).
2. To recognize the common clinical presentations of vasospasm due to cold sensitivity.
3. To be familiar with the noninvasive diagnostic evaluation of digital ischemia and vasospasm.
4. To understand the features of uncommon vasospastic disorders, including livedo reticularis, acrocyanosis, and erythromelalgia.
5. To be familiar with the various treatment approaches to primary and secondary vasospasm.

References
22. **Arteriovenous Malformations and Arteriovenous Fistulae**
Michael A. Golden, M.D., James C. Stanley, M.D., Thomas C. Naslund, M.D.

I. Anatomy and Pathophysiology
1. To understand the pathophysiology of arteriovenous malformations (AVM) and arteriovenous fistulae (AVF). This includes the rare forms (congenital and acquired) and the more common forms (traumatic and iatrogenic) of arteriovenous communications.
2. To define the influences of age, location, presenting symptoms and past medical, surgical and traumatic history on the etiology of arteriovenous communications, and to recognize the importance of syndromes with AVM.
3. To understand the common risk factors for the development of acquired arteriovenous communications, and how to anticipate and minimize the risks.
4. To understand the clinical settings associated with congenital AVM and to recognize them without delay.
5. To understand the early and the late important hemodynamic properties and effects of arteriovenous communications, and the effects of these changes on perfusion.
6. To understand the adaptive responses to the abnormal hemodynamics associated with arteriovenous communications.
7. To understand the natural history of arteriovenous communications as a function of the type of communication, (etiology, location, size, comorbidity and complications).
8. To understand the principles for the creation of arteriovenous communications for therapeutic indications, such as dialysis access, and distal extremity bypass grafts and venous bypass grafts.
9. To understand the technical considerations for the creation of arteriovenous communications for therapeutic indications, such as dialysis access, and distal extremity bypass grafts and venous bypass grafts.
10. To understand the complications and problems with therapeutic arteriovenous communications.

II. Diagnostic Evaluation
1. To understand the patterns of presentation of patients with arteriovenous malformations (AVM) and arteriovenous fistulae (AVF). This includes the rare forms (congenital and acquired) and the more common forms (traumatic and iatrogenic) of arteriovenous communications.
2. To understand the role of history and physical examination in the diagnosis of arteriovenous communications.
3. To define appropriate, cost effective diagnostic testing for arteriovenous communications.
4. To understand the role of the vascular diagnostic laboratory for the diagnostic evaluation of arteriovenous communications.
5. To understand the role of magnetic resonance imaging and magnetic resonance angiography for the diagnostic evaluation of arteriovenous communications.
6. To understand the role of contrast angiography for the diagnostic evaluation of arteriovenous communications.
7. To understand the role of diagnostic studies for the selection of the patient and site, and the preparation for the creation of a therapeutic arteriovenous communication.
8. To understand the diagnostic evaluation of the complications and problems with therapeutic arteriovenous communications.

III. Treatment
1. To understand the role of conservative management for arteriovenous communications.
2. To understand the role of catheter based intervention in the treatment of arteriovenous communications.
3. To understand the role of open surgery in the treatment of arteriovenous communications.
4. To understand the interactions of the treatments and the expected impact of combinations of treatments of
arteriovenous communications.
5. To understand the principles for the creation of arteriovenous communications for therapeutic indications, such as dialysis access, and distal extremity bypass grafts and venous bypass grafts.
6. To understand the technical considerations for creation of arteriovenous communications, for therapeutic indications, such as dialysis access, and distal extremity bypass grafts and venous bypass grafts.
7. To understand the complications and problems with therapeutic arteriovenous communications.
8. To understand the advantages and disadvantages of therapeutic AVF.

References


23. **Vascular Access**
Mitchell H. Goldman, M.D., Enrico Ascer, M.D., Gary Peterson, M.D.

**I. Anatomy and Pathophysiology**
1. To know that arterial and venous anatomy involved in the commonly placed grafts and sited for hemodialysis in the upper and lower extremities; know the options for unusual grafts sites when extremities are not available.
2. To know the local and systemic, anatomic effects of creating an arteriovenous fistula for the purpose of hemodialysis.
3. To know the anatomic landmarks for the various routes of access to the circulation for the use of chemotherapy, chronic infusion, obtaining blood samples, and physiologic monitoring.
4. To know the hemodynamic and physiologic effects of creating an arteriovenous fistula; understand the effects of large and small fistulae on the adjacent arteries and veins and on the body as a whole.
5. To know the anatomic and physiologic etiologies for arterial steal, decreased extremity flow and venous hypertension in AV fistulas created for hemodialysis.

**II. Diagnostic Evaluation**
1. To know the physical exam and diagnostic tests used in selecting a site for a vascular access including Allen’s test, use of duplex screening of veins, and stereography.
2. To know the diagnostic tests used in evaluating an arteriovenous access with high resistance, poor pressure, thrombosis, and infection.
3. To know the complications of obtaining access to the central circulation and the diagnostic examinations and tests used to diagnose pneumothorax, misplaced line, pseudoaneurysm, venous thrombosis, and hemorrhage.
4. To know the use of duplex scanning in the evaluation of AV accesses.

**III. Treatment**
1. To know the uses and benefits of using autologous or synthetic grafts for the purpose of hemodialysis including the locations, timing of placement, maturation of and longevity of the various access routes and grafts.
2. To know the treatment of complications of arteriovenous fistulas for hemodialysis including infection, steal syndrome, aneurysms, venous hypertension, thrombosis, stenosis, and the failing graft.
3. To know the use of revision, patching, extending, banding, angioplasty and stenting as methods of prolonging AV access.
4. To know the advantages, techniques and commensurate applications of each route of access to the circulation for the use of administering chemotherapy, chronic infusions, obtaining blood samples and hemodynamic monitoring.
5. To know the complications of the above routes and their treatment.
6. To know the catheter types, their advantages, available for gaining access to the circulation.
7. To know the long term outcome and patencies of the various access types.

**References**
3. Rivers SP, Scher LA, Veith FJ. Correction of steal syndrome secondary to hemodialysis access fistulas: a
24. **Sympathectomy**
Thomas S. Riles, M.D.

I. Anatomy and Physiology
1. To understand the basic anatomy of the autonomic nervous system including the course of sympathetic fibers through the spinal cord, the location of the sympathetic ganglia and the course of the post synaptic fibers.
2. To understand the relationship between the sympathetic fibers and the abdominal aorta and iliac vessels.
3. To understand the functions of the sympathetic nervous system and the pathologic conditions resulting from abnormal sympathetic activity.
4. To understand the potential beneficial effects of sympathetic ablation and possible adverse side effects.

II. Diagnostic Tests to Evaluate Sympathetic Function
1. To understand the basis of various tests to assess sympathetic activity.
2. To be aware of the limitations of the diagnostic tests used to assess sympathetic activity.

III. Clinical Uses of Sympathectomy
1. To understand the historic and current role of sympathectomy for arterial occlusive disease.
2. To understand the probable outcome when sympathectomy is used for ischemic ulcers, gangrene, rest pain, and the differences in clinical response for diabetes and non-diabetes.
3. To be aware of the role of sympathectomy for Buerger’s disease, embolic disease, Raynaud’s phenomenon, causalgia and post traumatic rest pain, and hyperhidrosis.

IV. Surgical Technique
1. To be aware of the technique for surgical ablation of the lumbar sympathetic chain as well as the technique for chemical ablation.
2. To be aware of the surgical technique for thoracodorsal sympathectomy.
3. To understand the potential complications from lumbar and thoracoabdominal sympathectomies and how to reduce the risk of complication.

References
25. **Portal Hypertension**  
Robert W. Hobson, III, M.D.

I. Anatomy and Pathophysiology  
1. Describe the anatomy of the liver and its portal and arterial circulations.  
2. Understand the relationships of extra- and intrahepatic pathological abnormalities resulting in portal hypertension and a tendency to variceal bleeding secondary to the elevations in portal pressure.  
3. Define the limits of portal pressure and its influence on variceal bleeding.  
4. Understand the physiology of increased splanchnic blood flow observed in the later stages of intrahepatic and extrahepatic disease. The importance of splanchnic vasodilation and its contribution to portal hypertension should be appreciated.  
5. Understand the hemodynamics associated with the portal hypertension syndrome to include decreases in mean arterial pressure and peripheral resistance, while increases in cardiac index and output are observed. As a result of an associated peripheral vasodilation, describe the neurohumoral pathways which are activated leading to sodium retention, expansion of plasma volume, and increased arterial pressure and cardiac output.  

II. Etiology  
1. Describe intrahepatic and extrahepatic (pre- and posthepatic) causes of obstruction to the portal circulation.  
2. Understand the causes of portal hypertension which are extrahepatic, intrahepatic, sinusoidal and hepatic venous in etiology. Categorize portal vein thrombosis, schistosomiasis, cirrhosis, and Budd-Chiari syndrome in this classification.  
3. Understand and define the determinants of variceal bleeding.  

III. Diagnostic Evaluation  
1. Define the Child’s classification  
2. Understand the clinical evaluation of the portal hypertensive patient and describe the stigmata of liver disease detailed during a history and physical examination.  
3. Describe the importance of liver function studies in the Child’s classification.  
4. Understand angiographic imaging of the portal vein by selective splanchnic angiography. Alternative techniques including computed tomography and magnetic resonance imaging may also contribute and should be understood in the evaluation of these patients.  
5. Describe the role for hemodynamic measurements including wedge hepatic venous pressure as well as duplex imaging of the portal vein.  

IV. Management  
1. Control of acute variceal bleeding.  
   a. Understand the circumstances of variceal bleeding, its mortality in relationship to the Child’s classification, and the natural history of bleeding.  
   b. Understand the role of fluid management, pharmacological treatment with splanchnic vasoconstrictors (vasopressin), vasodilators (nitroglycerin) and other pharmacologic agents.  
   c. Understand the role of the Sengstaken-Blakemore and Linton tubes in the control of acute variceal bleeding.  
   d. Describe the value of endoscopic sclerotherapy in the management of acute variceal bleeding. Understand the efficacy and timing as well as the technique used for endoscopic injection.  
   e. Describe endoscopic variceal band ligation and percutaneous transhepatic embolization in the control of variceal bleeding.  
2. Surgical Management of Portal Hypertension
a. Understand the historical development of the Eck fistula and its impact on the surgical management of portal hypertension.
b. Understand the difference between total portal-systemic shunts and selective (distal splenorenal) shunts.
c. Describe the non-shunt surgical management of varices including the Womack and Sugiura procedures.
d. Describe the development and use of intrahepatic shunts (transjugular intrahepatic portosystemic shunts-TIPS).
e. Describe the advantages of the TIPS procedure for acute variceal bleeding and the anticipated mortality when compared with portal-systemic shunts.
f. Understand the role of liver transplantation in patients with portal hypertension and variceal bleeding.

V. Describe a current clinical algorithm for the management of variceal hemorrhage.
   a. Understand the role of early endoscopic diagnosis in the control of variceal bleeding.
   b. Understand that endoscopic sclerotherapy will control the majority of patients with acute variceal bleeding, while balloon tamponade or TIPS may be required in the remainder of patients.
   c. Understand options for non-alcoholic and alcoholic patients with controlled or recurrent bleeding: selective variceal decompression with distal splenorenal shunt, sclerotherapy with or without pharmacological agents, and liver transplantation.

References