Plenary Lectures
Vascular surgery (VS) practice has expanded to incorporate interventional procedures, which has stimulated changes in training. The purpose of this study was to compare current VS training and certification in 34 different countries from Europe, Asia, Australia, and the Americas.

Certification

Of the 34 countries surveyed, VS is an independent specialty in 15, i.e., no prerequisite certification in general surgery (GS) or cardiothoracic surgery (CTS) is required. In 10 countries, VS is a subspecialty of GS, meaning that VS certification is permitted only after prerequisite GS certification. Finally, in 9 countries VS is not an accredited surgical specialty. The first country to recognize VS as a specialty was Italy in 1974, when VS was also accorded independent certification in that country. From then until 1988, there was an increasing trend toward certification in VS as a subspecialty, and since 1988 certification in VS as an independent specialty has become predominate (Figure). The most recent country to recognize VS as a subspecialty was Sweden in 2006, and the most recent countries to recognize VS with independent certification were Germany, Taiwan, and the United States in 2005.

Certification of trainees in VS is most frequently performed by national boards or medical associations (n=15), and less frequently by the government (n=9), vascular society (n=6) or the university/hospital that houses the training program (n=4). The most frequently used tool to assess candidate qualification for VS certification is inspection of a case log of recent operations, which is required in 81% of countries that offer VS certification. An oral examination is required in 77% of these countries, while 50% require a written examination to qualify for VS certification. Among countries where VS is an independent specialty, 87% require either an oral or written examination to qualify for certification. Among countries where VS is a subspecialty certification, 70% require either an oral or written examination.

Training

In countries where VS has independent certification, the mean duration of VS training is 3.7 ± 0.9 years with a range of 2-5 years. In these countries, there is an associated mean training in general or core surgery of 2.3 ± 0.7 years with a range of 1-3 years. Thus, the mean total duration of training to qualify for VS certification is 5.9 ± 1.0 years, with a range of 4-8 years in those countries where VS is an independent specialty. In countries where VS is a subspecialty following certification in GS, the mean duration of VS training following GS training is 2.4 ± 0.5 years with a range of 2-3 years. The associated
prerequisite GS training is a mean duration of 5.0 \pm 1.1\ years
with a range of 3.6-8\ years. Thus, the mean duration of total GS
plus VS training in countries where VS is a subspecialty is 7.4 \pm 
1.2\ years with a range of 6.9-9\ years. Among countries where VS
is not a certified specialty, VS training is incorporated either
into GS or CTS residency training, sometimes as additional
training in special vascular units, or sometimes as additional
non-accredited training following GS residency. Among these
countries the mean duration of initial GS (or occasionally CTS)
training is 5.4 \pm 1.8\ years with a wide range of 2-8\ years.
Additional optional VS training is a mean of 2.1 \pm 1.0\ years
with a range of 2-3\ years. Thus, in countries without VS
certification, the minimum total length of training required to
practice VS is a mean of 7.1 \pm 1.7\ years with a range of 5-9
years. The mean duration of specific VS training in countries
with independent certification is significantly longer than
countries with subspecialty certification or no certification in VS
(P<.01). The mean duration of GS and total training in
countries with independent certification is significantly shorter
than countries with subspecialty certification or no certification
in VS (P<.01).

Nearly all countries have established a minimum volume
of major open vascular operations that are required during
training and for certification in VS. Among countries with
independent VS certification, the minimum number of major
open vascular operations is 151 \pm 78 (mean \pm standard deviation).
Among countries with subspecialty VS certification, the minimum required number of major open
vascular operations is somewhat lower, 113 \pm 53 (P<.10), but
this number does not include operations that may have been
performed during prerequisite GS training. There is
substantial variation in the minimum number of major
vascular operations required even among countries with
independent VS certification, however, ranging from 30 in
Italy to 300 in Australia-New Zealand. Most countries (69%)
have established a minimum volume requirement for minor
open vascular operations such as varicose vein treatment.
Among countries with independent certification, the mean
minimum volume is 95 \pm 55, while in countries with
subspecialty certification the mean minimum volume is 58 \pm
38 (P<.06). Only 35% of countries have established a
minimum volume of minor vascular operations to be
performed as teaching or first assistant, and these numbers
vary widely. Among countries with independent VS
certification, 71% have developed a minimum volume requirement for interventional-endovascular procedures
compared with only 37% of countries with subspecialty
certification (P<.05). The mean minimum volume requirement for interventional procedures among countries
with independent VS certification is 52 \pm 45, significantly
more than the mean minimum volume requirement of 19 \pm
9 in countries with subspecialty certification (P<.03). Based
on this analysis, countries with independent VS certification
have higher minimum case volume requirements for VS
training and certification compared with countries where VS
has subspecialty certification status.

There is a large variation in the number of VS training
programs and the number of trainees per year among
countries with VS certification. When normalized according
to population, countries with independent VS certification
produce an average of 5.4 \pm 2.8 VS trainees per year for
each million persons \geq 65\ years of age. This is significantly
more than the number of vascular trainees per year of 3.0 \pm
1.8 per million persons \geq 65\ years of age in those countries
where VS has subspecialty certification status (P<.02).

The number of major open vascular operations actually
performed by VS trainees is recorded in 67% of countries
with independent VS certification, and averaged 208 \pm 176
operations in 2005. Only 37% of countries with
subspecialty VS certification record the number of major
open vascular operations performed by VS trainees, which
averaged 91 \pm 95 in 2005 (P<.07 vs. countries with
independent VS certification). The average number of
interventional procedures (both diagnostic and therapeutic,
counting one procedure per patient encounter) was
recorded by fewer countries, but averaged significantly
more in those with independent VS certification (104 \pm
103) compared with those countries with subspecialty VS
certification (21 \pm 14, P<.05). Thus, there was a larger
operative and interventional experience by trainees in
countries with independent certification compared with subspecialty VS
certification, but this comparison did not include
procedures performed during prerequisite GS training in
countries with subspecialty VS certification.

Conclusions

Considerable variation exists in VS training in
different countries. There is a clear international
movement toward independent VS certification, with
longer VS specific training but shorter overall residency
duration. Counties with independent VS certification
require more vascular operative experience and produce
more trainees per year to serve their elderly population.

Derived from: Cronenwett JL, Liapis CD. Vascular
surgery training and certification: an international

Figure: Status of vascular surgery certification in surveyed countries by year.
The interest of physicians and researchers for von Willebrand factor was originally prompted by the established role of this adhesive multimeric protein (the larger in size in human plasma) in supporting primary hemostasis, as well as for causing, when defective or dysfunctional, von Willebrand disease, the inherited bleeding disorder called after the name of the Finnish pediatrician who first described it in 19241.

Evidence for the more recently established role for von Willebrand factor in thrombosis2 stems mainly from several epidemiological studies demonstrating that high plasma levels of the protein are associated with a mildly increased risk of atherothrombosis3, and from the recognition that the presence in plasma of ultralarge, highly thrombogenic multimeric forms of von Willebrand factor is often associated with the clinical prototype of microvascular thrombosis due to disseminated intravascular platelet aggregation, i.e., thrombotic thrombocytopenic purpura4. It remains to be seen whether or not the addition of von Willebrand factor measurements to classical measurable risk factors truly helps to improve our capacity to identify individuals at risk of thrombosis; and whether or not qualitative changes of von Willebrand factor are better predictors of the risk of atherothrombosis than quantitative changes. This is currently an interesting and potentially promising field, that can only be explored when relatively simple laboratory methods capable of identifying and measuring the most thrombogenic forms of von Willebrand factor will be developed.

References

The association between air pollution and human morbidity is known for over half a century. The strongly increased mortality, which was observed during episodes of extreme pollution, such as in the Meuse valley in 1930 and during the “London fog incident” in 1952, have directed research towards understanding the cause of mortality. Air pollution is caused by a mixture of various gases (CO, O₃, S0₂ and NOₓ), water dissolved solutes and particulate matter. Particulate matter (PM), particularly with small diameter, below 10 mm (PM₁₀) has the worst outcome on human health.

Epidemiological studies have underscored that ambient air pollution is not only linked to pulmonary disease, but have evidenced a relation between overall and cardiovascular mortality and the degree of air pollution on the previous day. Various mechanisms have been advanced to explain this strong epidemiological association, ranging from systemic translocation of ultrafine particles into the bloodstream, over neuronal effects to lung inflammation-related risks. Yet, although the short time frame between exposure to traffic and the onset of myocardial infarction, within 1h is compatible with induction of platelet activation by passage from the lungs of ultrafine PM into the circulation, it took several years of research in experimental animal models to understand how both the direct passage in the circulation of ultrafine PM and lung inflammation contributed to platelet sensitization and to the development of a prothrombotic risk.

Indeed, both the rapid and more progressive lung inflammation-coupled sensitization of circulating platelets culminate in a thrombogenic tendency, at the time when platelets are confronted with mild vascular lesions, such as present in the atherosclerotic blood vessels of elderly individuals. In addition, recent evidence suggests that ambient air pollution itself can speed up the atherogenic process and accelerates plaque formation. Together with poorly understood cardiac effects, executed by air pollutants, all these risk factors tend to elevate the occurrence of cardiovascular events and the probability of platelet-rich thrombosis.

Recent evidence suggests that air pollution also affects clotting assays performed in the plasma of healthy individuals, exposed to particulate air pollutants in the days preceding blood sampling. The negative correlation found between the prothrombin time and the degree of air pollution at the time of sampling, suggested that air pollution may also negatively impact on risk factors, traditionally more associated with venous thromboembolism. The additional finding that air...
pollution also interacts with cigarette smoking to increase plasma homocysteine levels, further completes the list of cardiovascular factors, elevating the risk for thrombosis, via vascular damage, brought about by homocysteine. Recent animal studies have confirmed the existence of an IL-6 dependent relation between air pollution and coagulation activation. These results suggest the existence of feedback loops in which systemic inflammation and hepatic transcriptional regulation of coagulation factor synthesis are central.

Understanding of the procoagulant effects of air pollution, however, is still in its infancy and more detailed work will be required to understand the different procoagulant pathways activated during acute and chronic exposure to particulate matter, in the light of the systemic and vascular inflammatory pathways, that are activated by these pollutants. Only such experimental work will allow the correct evidence-based appreciation of the impact of primary vs. secondary hemostasis in the cardiovascular risk assessment of air pollution, necessary to manage this risk.
Many of the common diseases afflicting hundreds of millions worldwide have a heritable component. 2007 has marked a new era in defining the genetic architecture of common diseases using so-called genome-wide association studies (GWAS). For the first time ever the whole landscape of genomic sequence variation can be surveyed by testing hundreds of thousands of genetic markers. The Wellcome Trust Case Control study is the largest GWAS to date, and analysed 14,000 patient DNA samples representing 7 common diseases and 3,000 DNA samples from healthy controls, as so-called ‘shared controls’ on the Affymetrix GeneChip for 500,000 SNPs. In total 24 new strong association signals were identified and for Coronary Artery Disease (CAD) three loci were replicated by us in a further GWAS in 875 German CAD samples. In our effort to discover further CAD risk genes we integrated the results of our platelet systems biology study (www.bloodomics.org) with those of the WTCCC GWAS. Platelets play a pivotal role in thrombus formation after plaque rupture in the coronary arteries. We postulated that the variability in platelet function observed in the normal population is too a large extent genetically controlled. By a systems biology approach we aimed to better define the relationship between gene sequence variation and platelet function. Firstly, we catalogued all genes transcribed in the megakaryocyte, the platelet precursor and the erythroblast. Secondly, we defined the function of platelets in 500 healthy individuals to identify low and high responders. The comparison of the transcriptome landscapes from the most and least active platelets identified 65 transcripts with significant correlation with function. We selected eight of these and typed SNPs in the corresponding genes in 6,000 CAD patients and 8,000 controls. This identified two more CAD risk loci encoding DNA binding proteins with known repressor function. Studies of laser-induced atherothrombosis in Danio rerio showed that silencing of both genes modified thrombus formation. In conclusion a combination of GWAS and functional genomics studies led to the identification of novel genes implicated in the pathophysiology of coronary artery disease.

References