ARTERIOSCLEROSIS

Stenting (Percutaneous Coronary Intervention (PCI)) has long been the treatment of choice for stable angina patients. Stents are a loser. They are "good" for 6 months to a year at most, then the blockage returns. Slamming an embolism against the arterial wall with angioplasty, then pinning it there with a stent can save a life that would have otherwise been lost to myocardial infarct. So, stents should be considered a crisis therapy emergency procedure only, designed to buy time in the case of a heart attack.

Why did cardiologists believe in the procedure for so many years, and why do most cardiologists still perform the procedure even though the evidence is overwhelming that stents are of no long-term benefit? The problem stems from a misunderstanding of what causes heart attacks. The outdated theory (that persists to this day in the minds, or at least the wishful thinking, of most cardiologists, (and is still believed by the general public) is that heart attacks result from a blocked artery that finally either became so gradually blocked that it restricted blood flow, causing insufficient oxygen to the heart --- or --- suddenly became totally occluded because a chunk of plaque broke off the hardened arterial wall thus totally closing the artery whose flow was already severely impaired.

The theory was plausible, but research beginning back in the mid-1980s began to expose it as false. In 1986 research done at the University of Washington at Seattle showed that heart attacks occurred not in the areas of coronary arteries that were maximally blocked, but rather in areas of the artery where there was little or no plaque --- and certainly not enough to be stented or bypassed. Cardiologists scoffed at the research. Their "pipe-cleaner" model of coronary disease resulting from plaque accumulating slowly over decades --- finally to the point where one day no blood can get through and the patient has a heart attack --- had served them so well (and so lucratively). Angioplasty with a stent --- opening arteries by pushing plaque back against the narrowed artery wall, allowing some blood to get through --- or bypass surgery --- was the basis of their entire profession, and they were going to stick with it.

Still back in the 1980s, researchers in the Cleveland Clinic started looking directly into patients' coronary arteries with ultrasound cameras. It was found that arteries were full of plaque, but almost none of it was obstructing blood vessels. The new model of myocardial infarct is that it is not the plaque that

produces the narrowings, but the hundreds of other areas that are potential embolisms, and one of those bursts, forming a clot.

Finally by the late 1990s, the overwhelming research showed that arteriosclerosis is a systemic metabolic disease --- associated with inflammation of the arterial walls. Research shows conclusively that, at the most, 20% of heart attacks occur in the areas of major blockage. Instead, in 80% of cases a heart attack occurs when an area of plaque bursts, a clot forms over that area, and blood flow is abruptly blocked --- blocked in an area that was not significantly occluded. Again, the source of the plaque is nowhere near an area that would have been stented or bypassed --- which is why stenting or even bypass surgery offers zero protection against myocardial infarct.

The sudden release of a clot also explains why there are so many sudden heart attacks --- completely unexpected, even in those who routinely do jogging or other exercise with no chest pain or shortness of breath. If a narrowed artery were the culprit in heart attacks, exercise would have caused severe chest pain. But no, what happens is that one of hundreds of vulnerable plaques experiences an inflammatory reaction, clots, and blocks the artery in a place that was previously adequate in flow.

The outdated model persists so pervasively in the minds of the general public that patients with no symptoms whatsoever are undergoing the stenting procedure based on nothing more than the finding that there is some blockage in the coronary arteries. Far more than a million stent operations are done every year --- and most of those are done on people with no symptoms. --- Most often a patient goes to a cardiologist having been referred with a vague complaint such as indigestion or shortness of breath, or simply because a scan revealed calcium deposits.

The cardiologist, being a one-trick-pony, puts the patient in the cardiac catheterization room, does an angiogram examination, finds the inevitable sclerotic arteries (common in almost everyone over age 53 these days) and inevitably the patient comes out of the catheterization room to learn he has been awarded a brand new stent. Patients are thrilled because they are convinced that opening up the coronary blockages will save their life.

Pursani, et al. Percutaneous coronary intervention versus optimal medical therapy in stable coronary artery disease. Circ Cardiovasc Interv. 2012 Aug.

CONCLUSION: In this most rigorous and comprehensive analysis in patients with stable coronary artery disease, PCI, as compared with OMT, did not reduce the risk of mortality, cardiovascular death, non-fatal myocardial infarction, nor revascularization PCI.

Gorenoi, et al. [PCI in addition to optimal medical therapy for stable coronary artery disease --- a systemic review and a meta-analysis.] Dtsch Med Wochenschr. 2014 May.

PCI showed no benefit on the risk of death nor of myocardial infarction. The PCI reduced the number of angina attacks in 29% of the patients.

Stergiopoules, et al. Initial coronary stent implantation with medical therapy versus medical therapy alone for stable coronary artery disease. Arch Intern Med. 2012 Feb.

Now, more and more cardiologists are honest enough to admit that stenting is of no long-term benefit, and certainly does not prevent heart attacks. But most still cling to the notion that PCI is an effective way to relieve angina pain. And it does in many cases.

The supposition is that it controls angina pain during exertion such as exercise or even simple activities of daily living such as walking up the stairs. In 2017 Cardiologists were shocked by a study that showed stenting is no better than a placebo. In that study, some patients got stents, while others were put under anesthesia and were told they got stents, but received a sham intervention. --- At the 6 week evaluation, both the stent group and the sham stent group experienced the exact same benefits --- entirely the placebo effect in action.

The results were so shocking that some Cardiologists actually stopped using the stenting procedure altogether. Still, the majority of Cardiologists continue to insert stents despite any evidence that they do any objective good. [----- Can you imagine if an alternative health care practitioner recommends nutrition supplementation for a patient with full knowledge that the supplements do absolutely no good, but takes patients' money anyway just because some placebo effect is achieved? That doctor would be shut down faster than you can say, "Quack"!]

But stenting is such a popular procedure and so phenomenally lucrative ($$$) to Cardiologists, so --- another study was funded for the same mainstream

research team that did the first study --- in a desperate attempt to "prove" that the original study was false and that stenting is legitimate after all. This second study is dressed up (---twisted, distorted, and thus invalidated) so that stenting now appears (to anyone who fails to examine the study objectively) to be beneficial after all.

While the original study exposing the uselessness of stenting used a 1 month evaluation prior to the study to diagnose angina patients, the second study diagnosed "angina" patients on the spot, using measures that are completely invalidated. In other words, many of the patients in the second study did not really have angina at all.

In the follow up study designed to disprove the first study, the patients not only received the stent, but also were medicated with Heparin and Nitroglycerin. The original study did not use preloading with drugs. Do you suspect any chance that the benefits reported in the second study were from the Nitroglycerin and Heparin rather than the stent?

In the original study a true placebo group of patients was used --- patients who sincerely believed they had received the operation but didn't. In the follow up study there was no placebo group, other than patients who had angina, got nothing at all, and knew nothing had been done.

Harskamp, et al. Rethinking revascularization in patients with stable angina. Expert Rev Cardiovas Ther. 2018 Mar.

From the abstract: Traditional and current perception for benefit of Percutaneous Coronary Intervention (PCI) is that patients with stable angina will obtain symptom relief as well as improved exercise capacity after percutaneous revascularization. This common clinical perception is put to test in the ORBITA trial, the first blinded, randomized placebo-controlled clinical study ever conducted. The authors found no significant improvement in exercise time, functional status, angina relief and quality of life in the PCI group compared with placebo.

The results of the ORBITA study were published in Lancet:
Al-lamee, et al. Percutaneous coronary intervention in stable angina (ORBITA): A double blind, randomized controlled trial. Lancet 2018 Jan.

From the abstract:

BACKGROUND: Symptomatic relief is the primary goal of PCI in stable angina and is commonly observed clinically. However, there is no evidence from blinded placebo-controlled randomized trials to show its efficacy.

METHODS: ORBITA is a blinded, multi-center randomized trial of PCI versus a placebo procedure (sham intervention) for angina relief that was done at 4 study sites in the UK. We enrolled patients with severe (> than 70%) single-vessel stenosis. After enrollment, patients received 6 weeks of medication optimization. Patients then had pre-randomization assessments with cardiopulmonary exercise testing, symptom questionnaires, and Dobutamine stress echocardiography. After 6 weeks of follow-up, the assessments done before randomization were repeated at the final assessment. The primary endpoint was a difference in exercise time increment between groups.

FINDINGS: 230 patients underwent randomization, with 105 patients assigned PCI and 95 assigned the sham intervention. Lesions had mean area stenosis of 84.4%, fractional flow reserve of 0.69, and instantaneous wave-free ratio of 0.76. There was no significant difference in the primary endpoint of exercise time increment between groups. There were no deaths. Serious adverse events included 4 pressure-wire related complications in the placebo group, and 5 major bleeding events, including 2 in the PCI group and 3 in the sham group.

INTERPRETATION: In patients with medically treated angina and severe coronary stenosis, PCI did not increase exercise time by more than the effect of a placebo procedure. The study emphasizes that the efficacy of invasive procedures such as PCI must be assessed with a placebo control using a sham intervention, just as is the standard for pharmeco therapy.

A meta-analysis was performed on all randomized clinical trials comparing initial coronary stent implantation plus medical therapy to determine the effect on death, non-fatal myocardial infarction, unplanned revascularization, and persistent angina. Eight trials involving over 7,000 patients were analyzed. The mean follow-up was 4.3 years ----- the event rates of stent plus medical therapy versus optimal medical therapy alone were: Death = 8.9% vs 9.1%. Non-fatal MI = 8.9% vs 8.1%. Unplanned revascularization = 21.4% vs 30.7%. Persistent angina = 29% vs 33%.

CONCLUSION: Initial stent implantation for Coronary Artery Disease shows no evidence of benefit compared with initial medial therapy for prevention of death, non-fatal MI, unplanned revascularization, nor angina.

Not only does the stented coronary artery begin occluding again within 6 months, and is significantly occluded within a year after the stent placement --- but the placement of the stent is itself such an irritant that it stimulates proliferation of vascular wall cells --- increasing the rate of re-occlusion. (The "solution" to this problem is drug-eluding stents that release anti-proliferative drugs to slow the tissue re-growth that is stimulated by the stent. These drug-releasing stents do slow the rate at which the artery becomes blocked again, but there are risks.

Since 2006 it has been known that these stents cause a phenomenon called "late stent thrombosis", whereby the blood clotting inside the stent occurs 1 or more years after stent placement. This late stent thrombosis occurs in about 1 in 100 cases, and is fatal in about 1 of 3 of those. The problem of "natural" re-stenosis of the artery (occurring because the systemic nature of the inflammatory disease has not been addressed at its cause), plus the additional rate of re-closure because of the stent placement, is the reason why routinely cardiologists must prescribe two blood thinners after PCI --- Aspirin and something like Plavix.)

While stents are useless, their danger is significant in that about 1 out of 25 PCI procedures results in heart attacks caused by the stent insertion breaking loose a fragment of plaque. Also significant --- studies show that 42% of angina patients experience increased chest pain after PCI. But the complications and risks of bypass surgery are far greater.

Movahed, et al. Decreasing in-hospital mortality rates of patients undergoing PCI with persistent higher mortality rates in women and minorities in the United States. J Invasive Cardiol. 2010 Feb.

The in-hospital mortality rate from PCI was 1.2% in 2004 (decreased from 1.8% in 1995). Death immediately after PCI has increased a bit since 2004.

Absurdity ----- the only perceived benefit of PCI is the relief of chest pain --- yet perhaps the most common side effect of PCI is an increase in chest pain. Consider this study:

Hang, et al. Chest pain after PCI in patients with stable angina. Clin Interv Aging. 2016 Aug.

The number of PCIs in the population of patients with acute coronary syndrome with refractory or progressing angina is increasing. Post-PCI chest pain is one of the common problems of PCI. Its presentation and causes in patients with stable angina are poorly understood. This study shows that 42% of patients undergoing PCI had increased chest pain 24 hours after the procedure. The patients who experienced chest pain experienced abnormal post-PCI electrocardiogram changes and serum cardiac troponin 1 elevation (indicating heart muscle damage after the PCI). The patients suffering chest pain after PCI required repeat PCI.

The bottom line is that permanent results are achieved in the prevention of heart attacks and strokes by approaching arteriosclerosis as a systemic inflammatory condition. Attacking not the localized areas of occlusion, but rather addressing the metabolic needs of the entire coronary arteries (and indeed the entire arterial system) is shown in countless studies to be effective in prevention. Those who cling to the old model of the pipe cleaner find that if you "fix" one segment of a blocked coronary artery, a year later it will be another segment that is occluded and will either "need" another stent (or bypass surgery), or will yield one of those sudden heart attacks.

If stents are a loser, what about coronary bypass surgery? --- Same story as the stent. It is a perfectly appropriate and potentially life-saving procedure during a crisis, but it does absolutely nothing to prevent heart attacks. Like a stent, bypass surgery does relieve angina in many cases. But there is no increased protection from heart attacks, nor any benefit in preventing death from all causes in stable angina patients.

The cost/benefit analysis on bypass surgery must be considered on a patient-by-patient basis.

The first consideration is that while coronary artery bypass may relieve symptoms such as angina, it actually increases the risk of stroke. Another consideration is that brain damage from oxygen being cut off during the procedure is a problem, particularly in elderly patients. But even in relatively young people the blood flow deficiency to the brain can cause cognitive decline --- and --- sometimes during the procedure small bits of debris are released, causing in embolism blockage of blood flow and resulting in mini-strokes.

Another consideration is that the graft used to bypass a blocked artery is not permanent. Most of the grafts fail in one way or another eventually, with an average life of maybe 10 years. Within 15 years at the most a patient can expect to have to repeat the procedure all over again. ----- But again, the most important emphasis must be on the undeniable fact that even though the "fix" provided by the grafted bypass improves circulation to the heart, it is based on the failed model of cardiovascular disease explained in the section above on stenting. In other words, the bypasss, just like stenting, fails to address the primary cause of the arteriosclerosis --- that it stems from metabolically driven ImmunoNeuroEndocrine stress and its associated INFLAM-AGING. Targeting blocked blood vessels that are not the primary cause of the problem, and not even the immediate precipitating cause of a heart attack, is silly --- and all the sillier if the underlying cause of the arteriosclerosis is not addressed.

Physicians think they are addressing those causes with statin drugs to lower cholesterol, plus blood thinners. We strongly suggest you read our Articles on statin drugs to fully expose the Cholesterol Myth, and our Article on the risks vs benefits of Aspirin therapy.

Stenting and coronary artery bypass surgery are not the only potentially damaging weapons wielded by cardiologists …

CT Scans and Nuclear Stress Tests yield damagingly high levels of radiation. Once a cardiologist has put the fear of a heart attack in a patient's mind, that patient is likely to yield to these tests --- even though they rarely are beneficial.

Studies show that even in those who have been admitted to the hospital with what may appear to be a heart attack --- the one instance when extraordinary scanning procedures might be justified --- the patients do not get out of the hospital sooner. They do not get treated faster, and most importantly, they do not have better outcomes. Patients who undergo CT Scans and Nuclear Stress Tests have exactly the same rate of bypass surgery as those who do not get the scans, and they have the exact same risk of heart attack over the following month. In other words, the scans do absolutely nothing of benefit, and merely add hundreds or thousands of dollars to the hospital bill.

Yes, but better safe than sorry? No. The tests are not harmless. CT Scans involve hundreds of times the levels of radiation of the typical Xray, and a

coronary angiography blasts the patient with up to 800 times the radiation of a chest Xray. --- That radiation does not disappear after the test. And some patients are subjected to many of these scans over the course of their years under the care of a cardiologist. The toxic radiation exposure damage is cumulative. CT Scans alone are now considered a major cause of cancer.

Researchers at the National Cancer Institute have calculated the risk, and find that the 72,000,000 CT Scans carried out in 2007 will lead to 29,000 cancers down the road. Another study finds that as much as 1 in 400 CT Scans leads to a cancer. That is the risk of just a single scan. Those risks compound rapidly for those who are exposed to 2, 3 or even dozens of scans over the years.

Will NUTRI-SPEC supplements do anything to remove plaque in the arteries?

NUTRI-SPEC supplements may stop and will definitely slow the progression of the plaquing. ---- But --- that only works if the patient ceases doing all the pernicious practices that caused the plaquing to start with. Keep in mind that arteriosclerosis is the deposition of calcium in the arterial intima resulting from an inflammatory process in that tissue.

That inflammation is mediated via mainly macrophages, but also mast cells. Both macrophages and mast cells produce Prostaglandin D2 and Interleukin 6, and macrophages also produce Interferon-gamma. All of those are extremely pro-inflammatory. NUTRI-SPEC Metabolic Balancing or Diphasic Nutrition Plan will specifically address those inflammatory factors, with Taurine, Diphasic AM and Diphasic PM being the most critically needed.

For decades, research studies have seemed to indicate a link between healthy intestinal microbiota and resistance to cardiovascular disease. Recently, that link has been substantiated.

In particular --- a healthy microbiota prevents arteriosclerosis --- the hardening of the arteries that predisposes to both heart attacks and strokes. One recent study shows that some people have higher or lower degrees of arterial plaque than would be expected by other risk factors such as high blood pressure, high triglycerides, low HDL cholesterol, abdominal obesity and other risk factors known to be associated with hardening of the arteries. Some individuals have

none of the risk factors, yet their arteries already show calcium plaquing, while some with multiple risk factors have arteries that are perfectly clear.

The significant difference between these two groups is the bacteria in the gut.

As we know, the deposition of calcium plaques in the arteries is an inflammatory process. The most direct cause is an immune system with excessive activated macrophages producing the pro-inflammatory cytokine Interleukin 6. (One objective indicator that this pathological process is occurring is the blood level of C-Reactive Protein (CRP).)

--- Where do the excess activated macrophages come from? Macrophages (and mast cells) mainly dwell in the intestinal mucosa. From there, they are activated. Activated by what? A key trigger is the presence of toxins from nasty critters in the gut, and a deficiency of the healthy bacteria. The macrophages are activated in the gut, and then are dispersed throughout the body --- causing a virtual tsunami of inflammatory processes --- including inflammation of the arterial walls. That inflammatory process involves oxidation of LDL cholesterol, and subsequently the deposition of calcium plaques blocking the arteries.

Additional Note: Helicobacter Pylori is the bacterium that causes stomach ulcers and some duodenal ulcers. But recent studies also show that it can almost triple the risk of arteriosclerosis. The coronary artery calcium score is far higher in those in whom H. Pylori is cultured from the gut, and the risk of coronary artery sclerosis is compounded in those who have not only H. Pylori, but elevated triglycerides.

Another note on the connection between intestinal microbiota and heart disease: Long-term antibiotic use drastically increases the risk of death from heart disease. Yes, drastically. The risks begin after just two months of antibiotic use --- and is particularly a problem for women over 60. Many women over 60 take long-term antibiotics for chronic urinary tract infections. But the statistics are startling --- the risk of death from heart disease after 2 months of antibiotic use increases by 58% --- and the odds of death from any cause increases by 28%.

--- Quite simply, antibiotic use for that long devastates the population of beneficial bacteria. Antibiotics kill a disproportionate percentage of the beneficial bacteria relative to pathogens. The deficiency of healthy microbiota deprives the patient of the beneficial immune system anti-inflammatory effects that derive from healthy microbiota.

There is one antibiotic so strongly linked to death from cardiovascular disease that it does not require a 2 month course of treatment to increase risk. That antibiotic is Clarithromycin (Biaxin). A study published in Cardiology warned of this risk back in 2008. The study showed that just one single round of Clarithromycin treatment increases the risk of death by 21% over the next 6 years. Another study published in 2014 confirms the danger --- showing that the drug increases the risk of death in heart patients. ----- This antibiotic is prescribed with irresponsible frequency --- more than 3 million prescriptions per year nationwide --- for even simple non-clinically significant infections such as sinusitis and bronchitis. No one with even a slight risk of cardiovascular disease should consider taking this drug.

Antibiotics are not the only drugs that increase the risks of cardiovascular disease. All anticholinergic drugs dramatically increase the risk of stroke. Anticholinergic drugs include medications from several different families of drugs prescribed for a broad diversity of symptoms. Anticholinergics include beta blockers (ironically, a blood pressure med prescribed to high cardiovascular risk patients), Benadryl, Immodium, Seraquil, Valium, Xanax, and anything containing codeine.

Taking anticholinergic drugs increases the risk of stroke by 59%. Think of that, then consider how often these are prescribed --- and the huge percentage of the population that takes them routinely. --- Furthermore, these drugs not only increase the risk of having a stroke, but almost double the chances of dying from a stroke if you have one --- increasing the risk of death from a stroke by 86% --- and this is all according to the International Journal of Epidemiology. The biggest risk is in those with a high intake of these drugs --- in other words, those that take 2 or 3 anticholinergic drugs. That includes hundreds of thousands and perhaps millions of people.

Inflammation-causing arteriosclerosis starts out as a Dysaerobic/catabolic/ oxidative insult to the tissues, which then elicits an Anaerobic/anabolic defense. If a patient keeps eating HOHUM PUFAs, continues eating the sugars and excess carbs that stimulate excess production of insulin and insulin resistance, or, remains systemically Alkaline (so the calcium easily precipitates out of the blood), it is just a long, uphill battle.

One popular alternative to thinning the blood with Warfarin or Plavix is to supplement with nattokinase. (Nattokinase is available from many health food sources.) Nattokinase is a powerful fibrinolytic enzyme. As such, it does more than just "thin the blood" --- it specifically breaks down fibrin, the source of clots. One nice thing about nattokinase is that it works by an entirely different mechanism than does Warfarin or Plavix. That means it can be used in conjunction with Warfarin or Plavix and thus allow for a lower dose of the prescribed blood thinner.

However, caution must be exercised since using nattokinase concomitantly with Warfarin or Plavix can precipitate a hemorrhagic crisis from the blood becoming too thin. The remedy for the problem of blood becoming too thin is to decrease the Warfarin or Plavix and maintain the nattokinase (since it addresses one of the causes of the arterial plaquing (excess fibrinogen), instead of just thinning the blood by destroying vitamin K as the drugs do). But, no cardiologists will cooperate by reducing the dose of their prescribed blood thinner as long as the prothrombin time is not as ridiculously high as they like to keep it.

Caution: There are potential problems with nattokinase. Since it is such a strong proteolytic/catabolic enzyme, it can contribute to catabolic breakdown of tissues. Specifically, there is the possibility (however unlikely) that by its thrombolytic effect, it can cause lysis but also detachment of a thrombus from the surface on an arterial wall. So ironically, the beneficial effects of breaking down the fibrin can actually precipitate an embolism crisis.

The other potential problem from nattokinase, especially when used in conjunction with Plavix or Warfarin, is that it will so thoroughly thin the blood as to lead to hypovolemia, weakness, and loss of plasma into the interstitium, with edema collecting (particularly in the lower legs).

There is another supplement that can be used to either replace or augment the effects of Warfarin and Plavix, and that is Serraflazyme (available from

Cardiovascular Research). Like nattokinase, Serraflazyme is an enzyme that works to thin the blood by an entirely different mechanism than does the Warfarin or Plavix. It is specifically corrective of one of the mechanisms of arterial plaquing.

If Serraflazyme or nattokinase is taken, they absolutely must be taken on an empty stomach or they do zero good. So, first a.m. is ideal and at least 15 minutes before either the second or third meal of the day. They should be taken 3 or 4 tablets twice daily.

Additional Red Flag note: Neither nattokinase nor Serraflazyme should ever be given to a patient who has had a hemorrhagic stroke. (For that matter, such patients should never be given Warfarin or Plavix or Aspirin either.)

Even more important than thinning the blood, and even more important than decreasing the degree of blockage in the coronary arteries is <u>increasing</u> arterial compliance. In other words, through NUTRI-SPEC, arteries should become more elastic, and less subject to vasoconstriction, and more responsive to vasodilation. Formula ES, Taurine, Diphasic A.M., and Diphasic P.M. are essential. For anyone with a Sympathetic tendency, Complex S is also critical. For anyone with an Anaerobic tendency, Oxy Tonic is essential. (--- Revici found that arteriosclerosis could be induced in lab animals by giving them a diet extraordinarily high in both cholesterol and salt (not high in cholesterol alone). --- But --- on the same high cholesterol and high salt intake, no arterial plaquing occurred when the animals were supplemented with Oxy Tonic.)

Self-diagnose hardening of the arteries? ----- There is a surprisingly easy examination you can do on yourself. If you look in the mirror and you see what is called Frank's Sign, then you have a bright red flag waving that you have arteriosclerosis. More than 40 studies have found a link between Frank's Sign and hardened arteries, and some studies show a direct link to stroke risk.

What is Frank's Sign? Look in the mirror, and examine your earlobes. If there is a crease through the earlobe making it look as if the earlobe was folded and then unfolded (and the crease may run horizontally or diagonally), that is a positive Frank's Sign. Believe it or not, studies show that Frank's Sign is as

reliable in predicting arteriosclerosis as high-priced invasive testing done in the doctor's office or hospital.