GUIDELINES

Prevention and treatment of surgical site infection: summary of NICE guidance

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This is one of a series of *BMJ* summaries of new guidelines, which are based on the best available evidence; they highlight important recommendations for clinical practice, especially where uncertainty or controversy exists. The supporting evidence statements, further information about the guidance, and the members of the development goup are in the version on bmj.com.

Why read this summary?

The consequences of infection at the site of surgery can be devastating to the patient and costly to manage, often requiring readmission to hospital. Surgical site infections account for 14% of all healthcare acquired infections.[1] This article summarises the most recent guidance from the National Institute for Health and Clinical Excellence (NICE) for the prevention and management of surgical site infection.[2]

Recommendations

NICE recommendations are based on systematic reviews of best available evidence. When minimal evidence is available, recommendations are based on the guideline development group's opinion of what constitutes good practice. Evidence levels for the recommendations are in the full version of this article on bmj.com.

Information for patients and carers

Provide patients and carers with clear and consistent, easily understood information and advice throughout all stages of their care. Information should include:

- The risks of surgical site infections, how to prevent them, and how they are managed using an integrated care pathway
- How to recognise a surgical site infection and who to contact if they are concerned
- How to care for their wound after discharge from hospital.
- Whether they have been given antibiotics before or after an operation.

Preoperative phase

Showering

- Advise patients to shower or have a bath using soap (or help them with this) either the day before, or on the day of, surgery.

Hair removal

- Do not use hair removal routinely to reduce the risk of surgical site infection.
- If hair removal is necessary, use electric clippers with a single-use head on the day of surgery rather than razors, which increase risk of surgical site infection.

Theatre wear

- Give patients specific theatre wear that is practicable for the operation, with consideration for their comfort and dignity.
- Staff should wear specific non-sterile theatre wear in all operating areas and keep their movements in and out of these areas to a minimum.
- The operating team must remove artificial nails and nail polish, and where possible, hand jewellery, before operations.

Nasal decontamination

- Do not routinely use nasal decontamination with topical antimicrobial agents aimed at eliminating *Staphylococcus aureus* to reduce the risk of surgical site infection.

Mechanical bowel preparation

- Do not use mechanical bowel preparation routinely to reduce the risk of surgical site infection.

Antibiotic prophylaxis and treatment

- Give antibiotic prophylaxis to patients before:
  -Clean surgery involving the placement of a prosthesis or implant
  -Clean-contaminated surgery
  -Contaminated surgery.
- Give antibiotic treatment (in addition to prophylaxis) to patients having surgery on a dirty or infected wound.
- Consider giving a single intravenous dose of antibiotic prophylaxis on starting anaesthesia, or earlier for operations in which a tourniquet is used. Repeat the dose if the operation is longer than the half life of the antibiotic given.
- Do not use antibiotic prophylaxis routinely for clean, non-prosthetic uncomplicated surgery.

Intraoperative phase

Hand decontamination

- Before the first operation the members of the operating team should wash their hands with antiseptic surgical solution, using a single-use brush or pick for the nails, and should ensure that hands and nails are visibly clean.
- Before subsequent operations, wash hands with either an alcoholic hand rub or an antiseptic surgical solution. If hands are soiled then wash them again with an antiseptic surgical solution.

Sterile gowns and gloves

- The operating team should wear sterile gowns in the operating theatre during the operation.
- Consider wearing two pairs of sterile gloves when there is a high risk of glove perforation and the consequences of contamination are serious.

Incise drapes

- If an incise drape is required, use one that is impregnated with iodophore unless the patient has an iodine allergy.

Antiseptic skin preparation

- Prepare the skin at the surgical site immediately before incision using an aqueous or alcohol based antiseptic preparation (povidone iodine or chlorhexidine are most suitable).

Diathermy

- Do not use diathermy for surgical incision to reduce the risk of surgical site infection.

Maintaining patient homoeostasis

- Maintain adequate perfusion, oxygenation, and temperature control during surgery.
- Do not give insulin routinely to patients who do not have diabetes to reduce risk of surgical site infection by optimising postoperative blood glucose levels.

Wound irrigation and intracavity lavage

- Do not use wound irrigation or intracavity lavage to reduce the risk of surgical site infection.

Antiseptic and antimicrobial agents before wound closure

- Do not repeat skin disinfection during an operation or use topical cefotaxime in abdominal surgery to reduce the risk of surgical site infection.

Wound dressings

- Cover surgical incisions with an appropriate interactive dressing at the end of the operation.

Postoperative phase

Postoperative cleansing and dressing changes

- Advise patients that they may shower safely 48 hours after surgery.
- Use an "aseptic" non-touch technique for changing or removing surgical wound dressings.
- For wound cleansing, use sterile saline up to 48 hours after surgery. Use tap water after 48 hours if the surgical wound has separated or has been surgically opened to drain pus.

Wound healing by primary intention

- Do not use topical antimicrobial agents to reduce the risk of surgical site infection.

Wound healing by secondary intention

- Refer to a tissue viability nurse (or another healthcare professional with tissue viability expertise) for advice on appropriate interactive dressings.
- Do not use chlorinated solutions (such as Eusol) and gauze; moist cotton gauze; or mercuric antiseptic solutions.

Management of surgical site infection

- When surgical site infection is suspected, either de novo or because of treatment failure, use an antibiotic that covers the likely causative organisms, taking into consideration local resistance patterns and the results of microbiological tests.
- If debridement is required, do not use chlorinated solutions, gauze, dextranomer, or enzymatic treatments.

Specialist wound care services

Although no direct evidence exists to support the provision of specialist wound care services for managing surgical wounds that are difficult to heal, a structured approach to care (including preoperative assessments to identify individuals with potential wound healing problems) is necessary for improving overall management of surgical wounds. To support this structured approach, better education of healthcare workers, patients, and carers, and sharing of clinical expertise will be needed.

Overcoming barriers

Although the seriousness of surgical site infection is recognised, reduction of its incidence requires an understanding of several complex issues. For example, surgical operations involve different anatomical sites, are done for different clinical reasons, and may be performed in various locations.

Understanding the significance of surgical site infection requires surveillance using clear definitions and methods, and standardisation of these is currently lacking. The reduction in the rates of surgical site infection has relied on strict surgical discipline, and the evidence base for many measures is generally weak and contains data from a range of heterogeneous procedures and circumstances. There is plainly a need for research projects to improve the evidence base,

preferably from multicentre randomised controlled trials.

The use of antibiotic prophylaxis is evidently effective in reducing the incidence of surgical site infection, but the potential cost in terms of antibiotic resistance may be high and needs to be defined. The importance of tissue viability experts in the management of complicated surgical site infections is clear, but implementation of the wound healing recommendations will require adequate resources to train appropriate healthcare professionals.

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Competing interests: DL is a part time medical adviser to Renovo on trials of antiscarring agents, and he has a small number of shares in the company. Previously, DL has advised Arizant and Inditherm, which make warming products. He also advises Hutchinson on the development and evaluation of a new device to measure tissue oxygen. The department of wound healing at Cardiff University, where DL is a visiting professor, has received financial support from a large number of companies who have developed products in this area. DL is part of a research group in Salisbury that is partly funded by ConvaTec, and financial support from Ethicon is pending, through a competitive grant from its Foundation, Tyco, Coloplast, Novartis, and possibly Insense; much of this work relates to postoperative surgical site infection care. DL has had many charitable and industry grants for research work in antibiotic prophylaxis and treatment, dressings, and tissue perfusion. He has received financial support from several companies to attend and give papers to meetings of international societies and for educational activities; most of this funding related to the Surgical Infection Society and the European Wound Management Association (previously president) and the European Tissue Repair Society (current board member).

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2 National Institute for Health and Clinical Excellence. *Surgical site infection*. 2008. (Clinical guideline 74.) www.nice.org.uk/CG74.

Commentary: Controversies in NICE guidance on surgical site infection

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Major innovations and improvements have taken place over the past 20 years in the management of infected surgical sites, including the development of sophisticated wound dressings and specialist tissue viability nurses. However, we should not be complacent about surgical site infection as recent surveillance data for England show the rate to be as high as 10.5% after small bowel surgery,[1] and a Scottish study found a rate of 5.3% for clean abdominal (hernia) surgery.[2] As acknowledged in the latest guidance from the National Institute for Health and Clinical Excellence (NICE),[3] many confounding factors affect the identification of surgical site infection, not least 41 definitions and a variation in surveillance methods. However, it has been clear for almost three decades that the routine collection and dissemination of rates of surgical site infection results indirectly in a worthwhile reduction.[4] The Scottish Surveillance of Healthcare Associated Infection Programme reported significant reductions in the rate surgical site infection in inpatients after hip and knee arthroplasty over time.[5]

The justification for a national guideline on surgical site infection that encompasses prevention is therefore compelling. The authors have selected 44 clinical questions on the prevention and treatment of such infection (by no means all the potential areas of uncertainty)—such as the use of masks and caps in theatre and plastic ring drapes in abdominal surgery—inevitably reflecting the interests of the multidisciplinary Guideline Development Group. The group consulted over 200 stakeholders during guideline development, producing recommendations for practice and research, with nine key priorities for implementation. As acknowledged in the guideline, much of surgical care is ritualistic, particularly during the intraoperative period when a package of care is delivered, often with little or no evidence of the effectiveness of individual components on preventing surgical site infection (such as removing nail polish and hand jewellery, covering hair, and ensuring theatre discipline). Yet many of these practices are engrained in surgical practice, and strict surgical discipline is promoted and rightly supported by the Guideline Development Group as little evidence exists to prompt a need for change.

The best evidence for preventing surgical site infection relates to prophylactic administration of anti-infective agents, for which recommendations are almost consistently based on rigorous, well conducted trials and reviews. However, the guideline exposes the paucity of high quality research on surgical infection: dozens of included trials are small, underpowered to detect surgical site infection or differences in the infection rates; have limited follow-up; or investigate out of date interventions. Methodological deficiencies are particularly apparent in the section on postoperative care; large multicentre studies on the

management of surgical wounds, appropriate dressings, and secondary healing are needed.

The guideline lists recommendations for practice, acknowledging when these are based on insufficient evidence, no evidence, and evidence of no effect, and stating when they are based on consensus agreement by the Guideline Development Group's expert group. Unfortunately, some recommendations advise healthcare professionals to "refer to local protocols" or are vague. For example, in the section for patients and carers, the guidelines state that patients should be informed of the risk of surgical site infection, although what the level of risk is (such as by procedure or age) or how best to impart this information is not presented.

We support the call for a standardised approach to monitoring surgical site infection at a national level. Surveillance of surgical site infection after orthopaedic surgery is mandatory in the United Kingdom, yet numbers of hospitals voluntarily submitting data for other procedures are low (for example, fewer than 10 participating hospitals reported data on 50 or more procedures involving small bowel, gastric, and hepatobiliary surgery since 1997).[1]

Postoperative surgical site infection is a common and costly problem for the NHS and patients, so the NICE guideline is timely and relevant. The guidance encourages standardisation in surgical care in an attempt to prevent and treat such infection. However, the guideline does not set standards for acceptable rates of surgical site infection or highlight areas of good practice. Twenty years ago we reported rates of surgical site infection in abdominal surgery and the difficulties in collecting the data.[6] A universal, accurate system for data collection remains elusive. Hopefully this guideline will not only improve the management of surgical site infection but more importantly reduce the incidence.

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LESSON OF THE WEEK

Giant cell arteritis causing aortic dissection and acute hypertension

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Recent onset hypertension should prompt investigation for secondary causes

Recent onset hypertension should prompt investigation for secondary causes. This search routinely includes screening for mineralocorticoid excess, renal artery stenosis, and, in selected cases, phaeochromocytoma. We present the case of an elderly woman with recent onset hypertension, hypokalaemia, and a raised erythrocyte sedimentation rate. The cause of the hypertension was presumed to be aortic dissection with compromised renal blood flow, but the erythrocyte sedimentation rate provided the clue to the final diagnosis.

Case report

A 74 year old woman was referred because of general malaise and hypertension. During the previous months she had reported fatigue, lack of appetite and weight loss, and muscle weakness. About three weeks before admission she had had sudden onset abdominal pain and diarrhoea, for which no medical aid was sought and which subsided spontaneously after a few days. Her medical history was unremarkable. In particular, her blood pressure had been only mildly raised (160/90 mm Hg, measured repeatedly during previous years, most recently two months before admission).

On examination she had severe hypertension (215/125 mm Hg) but no other abnormalities. Results of vascular, funduscopic, and abdominal examination were normal. Routine laboratory tests showed a raised erythrocyte sedimentation rate (85 mm/h), low plasma sodium (131 mmol/l) and potassium (2.8 mmol/l), moderately raised serum creatinine (111 μmol/l), and mild chronic metabolic alkalosis (arterial pH 7.51, bicarbonate 32 mmol/l). Urinary potassium concentration was 51 mmol/l, and there was no proteinuria or

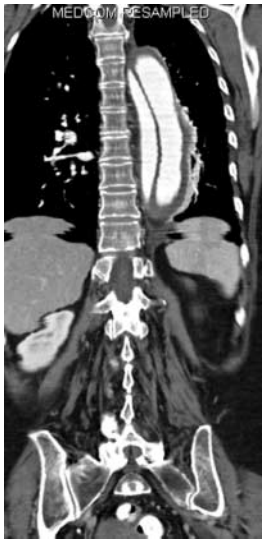

Fig 1 | Frontal plane reconstruction of computed tomography angiography, showing the thoracic part of a type B aortic dissection

haematuria. A chest x ray and an electrocardiogram were both normal.

She was diagnosed as having hypertension and hypokalaemia. As we suspected that she had primary hyperaldosteronism, we ordered a plasma renin-aldosterone profile and started oral nifedipine and intravenous potassium. Her blood pressure hardly responded to 30 mg of extended release nifedipine (given twice), but adding only 5 mg of the angiotensin converting enzyme (ACE) inhibitor lisinopril caused a sharp drop in blood pressure, from 220/105 to 135/70 mm Hg within two hours. After a few days, the laboratory called to report that her plasma renin activity was very high (30 ng/ml/h; normal value 0.3-3.5 ng/ml/h), with a plasma aldosterone concentration of 1.3 nmol/l (normal value 0.03-0.35 nmol/l). Thinking she might have renal artery stenosis we ordered CT (computed tomography) angiography, which showed an aortic dissection (type B: originating distal to the left subclavian artery; fig 1). The coeliac trunk and superior mesenteric artery originated from the false lumen, which ended at the level of the right renal artery. The left renal artery was stenotic with signs of atherosclerosis, with an atrophic left kidney. The surgeon decided not to operate on the aorta because no acute or alarming symptoms (pain, leakage, critical organ ischaemia) were present. Groin pulsations and leg arterial pulsations and pressure were rechecked and were found to be normal. She was further treated with low dose ACE inhibitors. Syphilis was excluded by serological tests.

Her further clinical course was uneventful. When we reviewed her case before scheduled discharge, we again noted the raised sedimentation rate, which had persisted throughout admission. The combination of a high sedimentation rate and aortic disease in an elderly woman triggered our suspicion of giant cell arteritis. Although her temporal arteries seemed completely normal, we took a 2 cm biopsy from the right temporal artery. Microscopic examination showed classic signs of giant cell arteritis (fig 2). She was treated with prednisone 60 mg daily. A year after she was admitted, she was doing well with 5 mg of prednisone and a low dose ACE inhibitor. Her erythrocyte sedimentation rate was 17 mm/h and her blood pressure 140/80 mm Hg.

Discussion

We believe that this patient had symptoms associated with her vasculitis during the months before admission. The aortic dissection may have explained her abdominal pain and diarrhoea (transient intestinal ischaemia) and was presumably responsible for compromised perfusion of the right kidney, which caused high renin, new onset hypertension. The left kidney was atrophic, suggesting longstanding renovascular disease, which had not caused hypertension before she developed aortic disease. Thus, an important lesson is that acute hypertension, in particular with signs of activation of the renin-angiotensin-aldosterone system (hypokalaemia, mild hyponatraemia, hyper-reninaemia, strong blood pressure response to ACE inhibition or angiotensin receptor blockade), should trigger suspicion of aortic disease, especially in elderly people. Aortic dissection can present with few symptoms and be easily overlooked. Hypertension occurs in about half of patients with aortic dissection and may be caused by "pseudocoarctation" (flow reduction through the true lumen, resulting in upper body hypertension) or involvement of the renal artery, or both. However, many cases seem to be idiopathic.[1] Marked hyper-reninaemia, as in our patient, is compatible with renal hypoperfusion, which may point towards both pseudocoarctation or renal artery involvement.

A second important lesson from this case is that aortic disease in an elderly patient with a high erythrocyte sedimentation rate should lead to consideration of giant cell arteritis. This disorder is also known as temporal artery arteritis and is most common in women over 50 (incidence 20-30 per 100 000).[2] About 10-15% of patients with temporal arteritis have clinically manifest aortic involvement, which presents as aneurysm or dissection of the aorta.[3] Aortic inflammation may represent a distinct clinical subtype of giant cell arteritis, but the pathogenic mechanism is probably similar to other subtypes.[4] A histopathological study showed that aortic involvement occurs frequently but has a subclinical course in most patients.[5] A recent study using positron emission tomography confirmed that subclinical inflammatory involvement of the aorta in patients with temporal arteritis or polymyalgia rheumatica is common: up to 75% of cases have abnormal findings suggesting aortic wall inflammation.[6] Positron emission tomography may be particularly useful to identify aortic inflammation in people who have negative findings on temporal artery biopsy, as obtaining a biopsy from the aorta to confirm the diagnosis is often not possible. Apart from infectious aortitis, Takayasu disease is important in

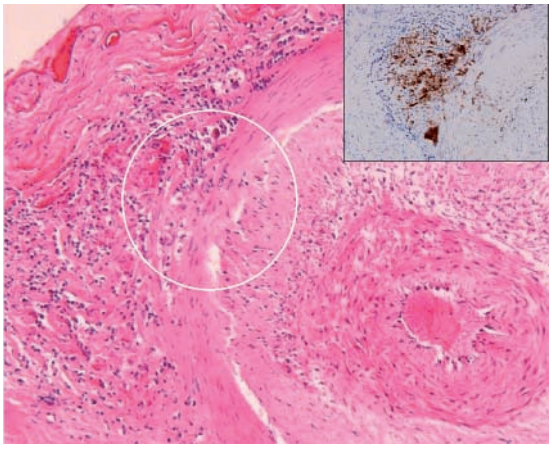

Fig 2 | Cross section of temporal artery biopsy. Main panel (HE stain, magnification x250) shows marked intimal hyperplasia. Circled area shows periadventitial inflammatory cells (mainly histiocytes and lymphocytes) invading the adventitia. Top right corner (immunohistochemical staining of the encircled area for CD68 positive cells) shows predominance of histiocytes, including giant cells

the differential diagnosis but usually presents at an earlier age and does not affect the temporal arteries.

In our experience, it is not uncommon for aortic giant cell arteritis to present without signs of temporal arteritis or abnormal temporal artery histology.[7] This case shows that even though a temporal artery seems normal, a biopsy is needed when there is clinical suspicion of giant cell arteritis. Recent onset hypertension in elderly patients may point towards aortic disease, and aortic disease, especially in combination with an acute phase reaction, may the presenting sign of giant cell arteritis.

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10-MINUTE CONSULTATION

New patient asking for a benzodiazepine prescription

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Your final patient on a Friday is a 26 year old man who is new to the list. He asks you for a repeat prescription for two months of diazepam, 5 mg up to four a day. He says he has been taking these for a while for his "nerves" and he has run out. You do not hold this patient's records.

What issues you should cover

You have to weigh up whether the risks of prescribing diazepam to this particular patient outweigh those of not prescribing. You should be as helpful as possible while checking whether he genuinely needs the prescription.

Is it appropriate to prescribe diazepam to this patient?

History—Is any information available about his previous consumption and medical history? This may be a repeat prescription slip or packaging giving details of the drug and amount prescribed. It may be possible to speak confidentially to his previous doctor or pharmacist by telephone while he waits (or early the next week). Most genuine patients can provide information about their previous general practitioner or pharmacy and are willing to wait while checks are made.

Indication—It is worth exploring why benzodiazepine treatment was started. However, whether it is an appropriate treatment for the long term may need to be explored in later consultations.

Dependency—Is the dosage he asks for likely to have resulted in dependency? Does he show signs of withdrawal? Anxiety, sweating, tremor, and insomnia are common features. Severe withdrawal symptoms, including seizures, are associated with prolonged use, a high dosage, short acting and potent benzodiazepines, and rapid withdrawal.

Is it safe to prescribe?

Take advice—Your computer system and drug formulary have information about interactions and contraindications.

Comorbidity—A history of seizures should lead to caution and a slow withdrawal of benzodiazepines, because of the risk of recurrence.

Polypharmacy—Benzodiazepines, where part of polypharmacy, are more likely to result in overdoses and, particularly in elderly patients, confusion and falls.

Substance misuse—Benzodiazepine use in association with misuse of alcohol, opiates, or other psychoactive substances raises the risk of respiratory depression and

Effect, half life, and equivalent dose of benzodiazepines

Drug	Effect	Approximate half life (hours)	Approximate equivalent dose
Lorazepam	Short acting	10-20	1 mg
Diazepam	Long acting	20-100	10 mg
Temazepam	Short acting	8-20	20 mg
Chlordiazepoxide	Long acting	5-30	30 mg

This is part of a series of occasional articles on common problems in primary care. The *BMJ* welcomes contributions from GPs.

Further reading

Benzodiazepine addiction, withdrawal and recovery. www.benzo.org.uk (the "Prof Ashton" link is very useful, especially the "Ashton manual")

Voshaar RC, Couvee JE, van Balkom AJ, Mulder PG, Zitman FG. Strategies for discontinuing long-term benzodiazepine use: meta-analysis. *Br J Psychiatry* 2006;189:213-20

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death. Benzodiazepines are misused by a large proportion of polydrug misusers, although legitimate prescriptions are not the major source of misused benzodiazepines, and their street value is low.

What should you prescribe?

Short term prescription—Prescribing a benzodiazepine for a short period such as <5 days may minimise the risk while giving you the opportunity to contact his last doctor for information.

Substitution—Prescribe longer acting benzodiazepines rather than short acting drugs, as they are safer and less likely to be misused and may reduce the likelihood of withdrawal symptoms. Example equivalent doses and half lives are set out in the table. The equivalent doses are a guide, and specific advice should always be obtained from the *British National Formulary* (www.bnf.org/bnf/) or equivalent or from a pharmacist.

Consequences—You should address his expectations about future prescriptions up front, conforming to practice policy and the best evidence.

What you should do

- Assess your personal risk. Patients who are desperate for a drug may be manipulative or aggressive. Examples of manipulative behaviour include playing one doctor off another: "Doctor X understands me and always prescribes what I need." They may pose mild threats such as leaving the list or not leaving the room until they get what they want. All consulting rooms should have panic buttons, and their use should be rehearsed periodically.
- If you have some doubt about prescribing a benzodiazepine or consider that he is at risk from sudden withdrawal, prescribe a few days treatment while you make further checks.
- If you prescribe a drug, advise him to take it at appropriate intervals.
- Arrange a review appointment the next week to discuss possible benzodiazepine withdrawal or substitution and to develop a shared management plan.
- Your practice should have a standard approach to acute prescribing and for tailing off long term prescriptions. Our practices prescribe five to seven days of acute prescriptions, avoiding follow-up on Monday or Friday. Other practices have one doctor who deals with all the practice's cases of drug dependency. If you do not have a standard approach, word may get around about which GPs "prescribe." Audit your practice policy. Primary care trusts' prescribing advisers can often benchmark your practice's prescribing with that of other practices.

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Paper hazards

My first international paper began as pantomime—my first slide was an alien. I asked for my slides: "Wyatt," I said, "WYATT."

The projectionist grunted and rummaged through the boxes on the tiny platform around the projector. Finally, he said, "You didn't give me any."

I nearly began the old music hall refrain, "Oh yes I did," but instead made my way through the audience. I too rummaged through the boxes until I found my slides hidden under the projector. They were necessary as my talk was a series of equations and enzyme pathways. After the projectionist had discarded the wrong slides and finished loading mine, I began again, but not for long.

Suddenly the chairman interrupted: "Thank you, Dr Wyatt. Next speaker please."

"But," I protested, "I've only just begun."

The eminent Harvard biochemist replied, "Your 10 minutes are up." Fortunately the microbiologists were on my side, and he was over-ruled.

In Nigeria, I was to give my evening lecture in the new, modern auditorium. It was open to the cool evening with no side walls, and birds, bats, and moths flew in and out of the light. As I turned to the screen, a sudden breeze lifted my notes one by one and in a beautiful sweeping arc sent them with the birds to the outer darkness of the neighbouring trees. I calmly closed the file and continued. At the end, the students applauded: my subject or my typical English sang froid?

I arrived tired and hungry in Amsterdam after my all night train journey from Vienna, and two accomplished thieves took my bag containing my passport and lecture slides. Next day I explained my problem to my colleagues and asked for their imagination: "This slide," I explained, indicating the blank screen, "would have shown …." They were very kind.

At the Johns Hopkins Hospital, we looked forward to Sir Peter Medawar's talk of his famous experiments. "This mouse," he explained and continued without hesitation as the slide appeared, "heavily disguised as a chicken …." We all cheered. An unforgettable lecture and lecturer.

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