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Smoke detectors and house fires

Alarms failed because detectors were not installed or maintained properly

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Three themes recur in injury prevention: the need to implement fully what is already known, a preference for passive strategies over active ones, and pressure to evaluate new programmes formally. It is unusual for all to be reflected in a single paper, but each is evident in the report by DiGuiseppi et al in this issue (p 995).[1] It is also unusual for a report to be as flawless as this one seems to be. The scientific literature is plagued with overworked phrases such as "landmark" and "milestone," yet this study describing the results of a cluster randomised trial of a distribution programme for smoke detectors fully deserves such accolades. To have evaluated a safety programme by using this immaculate design is a huge credit to the investigators and their funding bodies. It is also to the credit of this journal and its reviewers to publish a report whose findings are "negative." Or are they?

For evangelists of injury prevention everywhere the greatest challenge is to implement what has been shown to be efficacious.[2] Smoke detectors must surely rank high on the list of measures that appear to work, alongside seat belts or bike helmets, although none has been subjected to the rigours of a randomised trial. Nevertheless, it is widely agreed that these devices are efficacious. What is not known is how effective they are under specific circumstances, such as when the target is a population at high risk. The central finding of this large and complex undertaking is that simply distributing smoke alarms to such a population does not protect it against fires if the devices are not operational. This finding should surprise no one. It could easily have been assumed by health authorities that the benefits of a distribution programme were so self evident that the programme need not be studied. The sobering finding demands further explanation.

One possibility is that the study groups were not large enough. When asked questions about sample size, pundits often suggest that you estimate the required number and then double or triple it—and, even when you do you are still likely to come up short. This study had enough power to detect an intervention effect as large as that found in a previous study on which it was modelled—that is, an 80% reduction.[3] From a public health perspective, however, a much smaller reduction would be a great success, and ideally the sample should have been large enough to be able to detect it. But in light of the direction of the estimates, a much larger sample is unlikely to have yielded a different result.

A more likely explanation is that the effectiveness of any safety measure depends on whether it is used as intended. No one would declare a new drug to be useless if it were not taken in the proper dose or proper manner. The same applies to smoke detectors. The alarm prompts action that removes potential victims from danger.

One should remember that this remarkable report was not intended as a trial of the efficacy of smoke detectors. It aimed at examining a specific distribution programme, which, in spite of the earlier study, was well justified because it was not known if the same findings would apply to the low income, multiethnic communities in London.[3] Moreover, DiGuiseppi and her colleagues noted a flaw in the design in the study by Mallonee. As this was an observational, not experimental, study the choice of groups made regression towards the mean a likely explanation for its apparent effectiveness.

What makes the conclusion by DiGuiseppi and her colleagues so frustrating and disappointing is that, in contrast, their study was exemplary in every conceivable respect. They took account of all the niceties of the cluster randomised trial—a situation where opportunities for inappropriate statistical assumptions abound. Specifically, they examined clustering effects and showed them to be negligible. They took steps to provide for the cultural differences between their target group and those in the American study. They followed the CONSORT guidelines to the letter.[4] This is truly a model of how every safety programme should be evaluated—and evaluated they must be because not only are precious resources wasted on ineffective programmes but in some situations programmes may prove unexpectedly harmful.[5]

So what went wrong? For one reason or another, in spite of all efforts to improve adherence, too few of the distributed detectors were working properly when inspected. This reflects the need for more passive measures and less reliance on active—for example, educational—approaches. Like seat restraints, which must be properly fastened on each trip, smoke detectors require periodic checks to ensure that they are working properly.

Clearly, however, this population, for reasons that remain unclear, failed to adhere to the seemingly simple steps needed to install and maintain the detectors. How then can we prevent house fires among poor or elderly people, or homes with children? These results

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show that a simple give away is not the entire answer. For this population at least it may be necessary for public health officials to use more passive detectors, install and maintain them, or to require sprinklers in all new housing for people with low incomes. Disappointing as the results must be for this team, encouraging byproducts of their work are new directives from the local housing authority in the United Kingdom urging some such actions.[6]

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Treating acute gouty arthritis with selective COX 2 inhibitors

Preliminary evidence supports their relative efficacy and safety

Few arthritides are as painful, incapacitating, and stressful as a severe attack of acute gout, pseudogout, or calcific periarthritis. Successful treatment of these acute microcrystalline events depends on early use of an effective and safe anti-inflammatory drug in full dosage. The sooner such treatment is started the more rapid and complete the response. Treatment options include colchicine, non-steroidal anti-inflammatory drugs (NSAIDs), and corticosteroids including adrenocorticotrophic hormone.[1] Although colchicine is traditionally rooted in the treatment of acute gout, in recent years its use has declined steadily.[1] Its drawbacks include slow onset of action, narrow ratio of benefit to toxicity, and reduced efficacy when used more than 24 hours after the an attack begins. Colchicine (0.6 mg orally every 2 hours, up to 4-6 mg/day) is now reserved for patients without renal, hepatic, or bone marrow disease, in whom the more effective NSAIDs are contraindicated or poorly tolerated. Intravenous colchicine is best avoided given its potential for serious toxicity, which potentially can result in myelosuppression, hepatic necrosis, renal failure, hypotension, seizures, and death.

Intra-articular corticosteroids (for example, methylprednisolone acetate 5-25 mg per joint), systemic corticosteroids (oral prednisone 20 mg/day tapered off over 4-10 days, or intramuscular triamcinolone hexacetonide 60 mg/day, repeated in 1-4 days), and corticotrophin (40-80 IU every 6-24 hours) are valuable, highly effective, and relatively safe alternatives in patients with acute microcrystalline synovitis in whom neither NSAIDs nor colchicine are recommended. Such patients include elderly people and those with renal insufficiency, hepatic dysfunction, cardiac failure, peptic ulcer disease, and hypersensitivity to NSAIDs.[1] The duration of treatment is usually short, and side effects due to steroids are rare.[1]

Non-salicylate NSAIDs are the drugs of choice in the treatment of acute crystal induced arthritis.[1] Although no comparative studies have been conducted, NSAIDs are generally better tolerated and have more predictable therapeutic effects than colchicine. The patient is usually supplied with the appropriate NSAIDs (preferably carried with the person, for all too often gout strikes when the patient is far from home) and instructions to how to self treat the acute episode at the first "twinge" of an attack. No clear advantage is known of any one NSAID over another, but large initial doses are recommended: indomethacin 150-200 mg/day, naproxen 1000 mg/day, or diclofenac sodium 150 mg/day.[1] Although adverse reactions may occur, the duration of treatment with NSAIDs is generally short (4-8 days), and serious toxicity leading to drug withdrawal (such as gastrointestinal bleeding) is rare.

Conventional NSAIDs exert their anti-inflammatory effects mainly through inhibition of the enzyme cyclo-oxygenase, which catalyses the conversion of arachidonic acid to proinflammatory prostaglandins, particularly prostaglandin E_2. These play a major part in both experimental and clinical crystal induced inflammation, and act synergistically with other mediators (for example, bradykinin, leukotriene B4) to enhance capillary dilatation, pain sensitivity, and neutrophil chemotaxis.[2] Cyclo-oxygenase exists in two isoforms: cyclo-oxygenase-1 and cyclo-oxygenase-2.[3 4] Cyclo-oxygenase-1 is constitutively expressed in most tissues and is relatively unaffected by inflammatory mediators. It supports biosynthesis of prostanoids required for normal homeostatic "housekeeping" functions such as renal blood flow and maintaining the integrity of gastric mucosa. By contrast, cyclo-oxygenase-2 is constitutively expressed in a few tissues but is highly inducible in response to cytokines, endotoxin, mitogens, and growth factors, which implies a role in inflammation, infection, and cellular proliferation. In crystal and other inflammatory arthritides, cytokines—for example, interleukins, IL-1, IL-6, and IL-8—increase production of prostaglandin via induction of cyclo-oxygenase-2 expression in synoviocytes, and macrophages.[3 4] Although both cyclo-oxygenase-1 and cyclo-oxygenase-2 isoenzymes are expressed in mononuclear cells from gout and pseudogout synovial effusions, the exact role of cyclo-oxygenase-1 in inflammation is poorly understood.[5] Urate crystals

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