Nitric oxide (NO)/nitrogen dioxide (NO2) scavengers

Editor,—After reading with interest the study by Weimann and colleagues1 on a subject that we have recently reviewed,2 we would like to make the following comments.

We were surprised by the hypothesis of improved NO absorption through increasing the potassium hydroxide (KOH) content of soda lime, because Pickett and colleagues3 found a marked reduction in NO2 concentration (80.6% or 100%, depending on the electrochemical monitor used) and to a lesser extent in NO concentration (13.3% or 8.2%) using ethyl violet IntersorbTM soda lime without KOH. Moreover, Ishibe and colleagues4 found similar absorption properties for two types of soda lime (100% for NO2 from 11.9% to 97.1% for NO with Soda SorbTM, and from 20.6% to 90.1% for NO with Wako lime-A™, depending on the respective concentrations of NO and NO2 passed through the absorber). The absorbers had the same indicator agent (ethyl violet) but with one major variation in chemical composition—the presence of 5% KOH in Soda Sorb™. It was stated that NO2 is mainly absorbed by chemical neutralization with ethyl violet soda lime, but NO is absorbed with equilibrium amounts of NO2 only where they coexist.2,3 For this reason, it would have been interesting if the authors had presented the different rates of NO2 production for the three concentrations of NO (20, 40, and 80 ppm) and fractions of inspired oxygen (0.25, 0.75, and 0.99) used, as well as the different levels of NO and NO2 absorptions under these such conditions. This would explain the levels of NO absorption observed for Drägersorb 800® (2%) and Sofnolime™ (3%): 2 ppm of NO2/80 ppm of NO = 2.5% absorption for NO.

Moreover, the authors mentioned the concentration of NO (100 ppm) in the gas cylinder but not that of NO2. This is important because it was previously indicated6 that a cylinder of 993 ppm NO contained as much as 17 ppm of NO2 (in our case, 900 ppm NO cylinders contain less than 5 ppm of NO2). Weimann and colleagues reported a maximum concentration of 2 ppm NO2 measured in the extreme conditions of testing. Until now only two abstracts7,8 have reported partial absorption of NO2 using ethyl violet soda lime, while other reports (reviewed in Francœur et al9) indicated almost full absorption. The apparent discrepancy may be explained by technical reasons, as suggested by Weimann.3 In particular, we noted a difference in the chemical composition of the soda lime preparations with a higher content of calcium (around 75%) and sodium (around 3%) hydroxides.5,6 Weimann and colleagues suggest a ceiling effect in NO2 absorption with increased KOH content: there was no difference between Special-1 (3.0% KOH) and Special-2 (7.3% KOH) in NO2 absorption (respectively, 47% and 46%). We think that this ceiling effect would reveal a lack of correlation between the content of KOH and the level of NO2 absorption. From all these results, it would appear that the primary determinant of NO2 absorption is the content of calcium hydroxides, and that KOH has only a limited role.

We also noted major mistakes in the references cited in the discussion. First, Stenqvist and colleagues10 did not report a NO2 absorption “by almost 80%”; but between 87.5% and 100%. Second, we have reported above the exact levels of NO absorption found in the study by Ishibe and colleagues.3 Third, the paper by Pickett and colleagues,3 presented in Weimann’s discussion, is not corrected with technical modifications.8 As outlined by the authors,9 other materials such as molecular sieves 5A,12 ABEK HgCONO-P3,13 or charcoal14 have been found efficient for NO and NO2 adsorption and useful for environmental protection. Moreover, Vaahs and colleagues15 stated that NOXon – the NO2-filter material for a safe application of NO-gas and first option (Siemens-Elema, Solna, Sweden), etc.—raises questions over the need for continuous monitoring of inspired NO concentrations. A regular check might be sufficient.


Editor,—Thank you for giving us the opportunity to comment. In their letter, Drs Troncy, Francœur, and Blaise question the need for continuous monitoring of inspired NO concentrations during inhaled NO treatment and state that instead “a regular check” may be sufficient. We strongly disagree with this statement.

Nitric oxide inhalation treatment is not yet approved for clinical use and has still to be treated as an investigational drug. Therefore all possible measures should be undertaken to minimize harm to patients treated with this drug—as stated in the Declaration of Helsinki. This requirement has led to the development of safety
guidelines for the clinical use of inhaled NO that include the continuous measurement of NO and NO2. This capability is also included in the standards proposed by the American Food and Drug Administration (FDA) for NO and NO2 monitoring devices. Several studies showed that current NO delivery devices are not as accurate as suggested by Troncy and colleagues. This is important since the conversion rate of NO to NO2 is determined by the square of the NO concentration, the O2 concentration, and the residence time of NO with O2, where the latter is a result of the chosen mechanical ventilator setting. The frequent changing of settings of these variables—essential during treatment of the critically ill patients who require NO treatment—and the possible inaccuracy of NO delivery may lead to rapid and unpredictable changes in NO2 concentration, which should therefore be monitored continuously. Accordingly, all the NO delivery devices mentioned by Troncy are capable of continuous NO and NO2 monitoring. Nevertheless, not all of these devices are available in or meet the medical safety regulations of all countries.

There is clear evidence that NO2 inhalation may be toxic to the lung, even in the concentrations that may occur during clinical NO administration (for review see our original report). Furthermore, Troncy and colleagues point out that NO cylinders may be contaminated by large amounts of NO2—that is, 17 ppm NO2 (Tibballs and colleagues) or 12 ppm NO2 (Stenqvist and colleagues). Such contamination may be harmful if not recognized by inspired NO2 monitoring.

Besides our report and the two abstracts cited by Dr Troncy, two other publications showed only partial absorption of NO2 by soda lime. Stenqvist and colleagues used Q-Sorb, which contains either yellow or violet and stated that the NO2 concentration was reduced by about 75%—which we referred to as “almost 80%.” Westfelt and colleagues also reported NO2 absorption of only 65–70% using the same soda lime preparation (Q-Sorb). Only partial NO2 absorption by soda lime and the possibility of new NO2 formation in the inspiratory limb between the soda lime absorber and the Y-piece emphasize the need to monitor NO2 at the inspiratory side of the Y-piece. Our data did not reveal any correlation between the NO2 concentration and the NO extraction rate, as proposed by Ishibe and colleagues.

The study by Stenqvist and colleagues has been cited correctly (see above). We would like to thank Dr Troncy for clarifying the actual values for NO absorption found by Ishibe and colleagues. The values cited from Pickett and colleagues are, in fact, derived from their later study, which we did not state in our paper.

Our data suggest that NO2 absorption by soda lime may be correlated with the potassium hydroxide (KOH) content of the soda lime preparation: the higher the KOH content the more NO2 is absorbed. The effect of KOH content on NO2 absorption may already be maximal at 3.0%, which would explain the fact that Special-2 soda lime was not shown to absorb more than Special-1.

We showed clear differences between the five studied soda lime preparations but there was no significant difference in their calcium hydroxide content. Therefore, our data do not support the conclusion by Dr Troncy that the content of calcium hydroxide is the primary determinant for NO2 absorption by soda lime. We note that Dr Troncy and colleagues agree with us that different technical approaches for examining a certain scientific question may lead to different results. In our original report we discussed in detail the technical differences that may be responsible for the different results of the studies of Pickett and colleagues, Ishibe and colleagues, and our own. In contrast to the studies by Pickett and colleagues and Ishibe and colleagues, our set up was specifically designed to mimic the clinical situation as closely as possible. Further investigations may be required to elucidate the exact mechanisms by which NO and NO2 absorption by soda lime occurs and how it may be improved. However, current published reports agree that certain soda lime preparations can absorb NO2 and therefore may be useful in reducing the risk of potentially harmful side effects and in increasing the safety of NO inhalation treatment.

Meanwhile, the use of inhaled nitric oxide therapy should carefully follow the recent recommendations of an expert panel, which include that “NO and NO2 should be monitored continuously at the inspiratory side of the Y-piece ...”.

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Transposition of rotameter tubes

Editor,—Walmsley and Holloway are right to highlight the need for comprehensive checking. Considering the Association of Anaesthetists’ 1990 checklist unsuitable for daily pre-use checks, COVERS® was introduced in this hospital as a pre-use checklist and covers the whole 1990 Association revision. Also recognising the need for a formal schedule of comprehensive checking, COVERS® is backed up by the performance of an extended checklist based on the 1990 recommenda- tions which is carried out by the operating department assistant together before use, whenever a machine is returned from service or breakdown, and also midway between scheduled services.

Our audit confirmed the benefits of this routine. The combination is appropriate for the delivery of safe anaesthesia and the management of risk within the directorate.

Editor,—Thank you for giving me the opportunity to reply to Dr Lake’s comments about our letter. The COVERS comprehensive check list described by Krimmer and colleagues for use on machines after a service deals with some of the points we suggested in our letter. However, even this checklist—although it states that the flow through the oxygen and nitrous oxide flowmeters should be set at 5 litres/min, and not approximately 5 litres/min as suggested by the Association of Anaesthetists of Great Britain and Ireland check list—would not necessarily prevent a switch of flowmeters. Although the COVERS check list is extremely thorough, perhaps we could suggest adding a further point to the checklist by making sure that the flowmeters is labelled appropriately above each flow control valve. Perhaps also, as suggested in our letter, the flowmeters should be designed so that they could not be interchanged. In summary, although COVERS is a more thorough and comprehensive checking system than the AAGBI guidelines, particularly after anaesthetic machine servicing, we are still not convinced that it would necessarily always pick up problems such as flowmeter transpositions similar to the one described in our letter.

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Editor,—I write regarding the letter from A J Walmsley concerning transposition of rotameter tubes.1 ISO 5358 1980 and BS 4272, part 3, 1989, JIS T 7201, 1990, and ASTM F1161, 1988, all have similar wording to convey the same message. Blease Medical Equipment has been producing anesthetic machines for many years. We have used length and diameter indexed tubes for a very long time, as have all our competitors.

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Glaxo Wellcome plc are currently developing a glycine-free preparation, and trials are under way in the USA looking at its epidural and intrathecal use. In view of the above known risks we feel that the use of remifentanil for axial blocks is currently contraindicated.

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Sevoflurane for intubation of an infant with croup

Editor,—Barker describes his routine use of sevoflurane for inhalational induction in children with difficult or “easy” airways.1 We would like to report its use in the acute situation for intubation of a 22 month old, 15 kg male infant with “croup.”

The history was complicated but included several days of fluctuating stridor during which an opinion had been obtained from a consultant otorhinolaryngologist with a special interest in infants. A lateral x ray of the infant’s neck had shown subglottic narrowing. On the day in question the stridor had worsened and it was agreed by all concerned that urgent intubation was now necessary.

The boy was brought to the anaesthetic room resting over his grandfather’s shoulder. An experienced surgeon was in attendance. Induction of anaesthesia was with sevoflurane in oxygen 8 litres/min using an Ayres’ T-piece and face mask with the child seated upright on his grandfather’s lap. We started at sevoflurane 4%, increasing to 6% and then 8% when we were confident this would be tolerated. As soon as the child was judged adequately anaesthetised, a 20G cannula was inserted into an arm vein. Pulse oximetry, ECG, and non-invasive blood pressure monitoring were started. Intubation was attempted under deep oxygen–sevoflurane anaesthesia. At laryngoscopy a normal epiglottis and vocal cords were seen. A size 4.0 mm plain tracheal tube was chosen. Initial attempts at intubation failed because of the subglottic obstruction. It was easy to ventilate the infant’s lungs using a mask and oropharyngeal airway, so atracurium 150 µg and suxamethonium 30 mg were given intravenously. The trachea was intubated by the more experienced paediatric anaesthetist (APM) using an uncut 4.0 mm plain tracheal tube (Portex). Pushing the tube past the subglottic region—the infant was transferred to the regional paediatric intensive care unit for further management.

Viral croup or, more properly, acute laryngotracheobronchitis, is usually caused by paramyxovirus virus types I and II, although other viruses have been implicated. It affects children between the ages of six months and three years, with a peak age of two years. Acute laryngotracheobronchitis may affect the entire laryngotracheobronchial tree, but inflammation of the subglottic region—the narrowest portion of the upper airway in infants—produces the typical signs and symptoms.2 This results in gradually worsening inspiratory stridor progressing to fatigue and ventilatory failure. If medical treatment fails to relieve ventilatory distress, an artificial airway must be considered. The current recommendation is inhalational induction with carefully titrated halothane in 100% O₂ and intubation performed with the child breathing spontaneously.3


Remifentanil

Editor,—We read with interest Dr Dresner and colleague’s letter1 and Drs Morton and McClure’s reply2 regarding the use of remifentanil as part of an epidural test dose.

Morton and McClure disagree with the possible use of remifentanil because of “unknown risks.” Glycine is present in a concentration of 13 mg per 1 mg formulation of Ultiva (remifentanil) and the drug’s data sheet states that this is “contraindicated for epidural and intrathecal use.”3 It has been shown to be neurotoxic in rats,4 to cause encephalopathy in high doses,5 and to cause reversible motor impairment after intrathecal delivery.6 Clinical trials by Glaxo Wellcome in dogs showed that when injected intrathecally the buffer alone (glycine) caused hind limb muscle twitching, pain, and convulsions in high doses. Interestingly no such effects were seen with the remifentanil/glycine preparation (Lupton S, personal communication; Glaxo Wellcome plc).

The use of the COVERS checklist is not necessarily prevent a switch of flowmeters. Although the COVERS check list is extremely thorough, perhaps we could suggest adding a further point to the checklist by making sure that the flowmeters is labelled appropriately above each flow control valve. Perhaps also, as suggested in our letter, the flowmeters should be designed so that they could not be interchanged. In summary, although COVERS is a more thorough and comprehensive checking system than the AAGBI guidelines, particularly after anaesthetic machine servicing, we are still not convinced that it would necessarily always pick up problems such as flowmeter transpositions similar to the one described in our letter.

A J WALMSLEY
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In view of the above known risks we feel that the use of remifentanil for axial blocks is currently contraindicated.
We believe that this is the first report of the use of sevoflurane under these circumstances. Its use has also been described for the management of the difficult airway in adults in whom awake intubation is not possible,1 but not in those in whom stridor was a feature. Barker also describes the use of sevoflurane for children with difficult airways resulting from anatomical problems, but not from acute stridor. Inhalational induction of general anaesthesia is often used in children with, until recently, halothane as the preferred agent. The low blood gas partition coefficient (0.6–0.7) and pleasant, non-pungent odour of sevoflurane allow rapid smooth induction. Various investigators have described the induction characteristics of sevoflurane and halothane in children. Black and colleagues suggested that sevoflurane is better than halothane at providing a pleasant rapid induction without any airway complications and being free of appreciable effects on pulmonary ventilation, circulation, and neurological function.5 Epstein and colleagues showed that induction of anaesthesia was faster with sevoflurane than with halothane, and that vital signs were more stable. They concluded that sevoflurane was an excellent agent for inhalational induction of anaesthesia in children.6 However, it must be remembered that because of its low blood–gas partition coefficient, awakening will also be rapid. Once induced with sevoflurane, there is the theoretical risk of the child becoming “light” if there is difficulty securing an airway.

On the basis of our single experience we suggest that sevoflurane is a suitable choice of agent for the inhalational induction of anaesthesia in infants with stridor who require intubation. We recommend, however, that any anaesthetist using sevoflurane in this way should be experienced in its use for the inhalational induction of anaesthesia in infants undergoing routine surgery.

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Risk factors for cardiovascular death

Editor,—The excellent case–control study by Howell and colleagues1 does, with considerable authority, confirm the validity of previous cohort studies which identified both previous myocardial infarction and renal impairment as independent risk factors for cardiac death following anaesthesia and surgery. Their findings that diabetes mellitus is not even a univariate predictor is interesting, but not surprising and warrants further consideration. They postulate that there may have been some bias in attributing deaths in diabetic patients to their diabetes rather than to cardiovascular complications. However, it is generally accepted that the reverse is true,2 and therefore this is not the explanation.

Perioperative cardiac complications are almost universally associated with the presence of underlying coronary artery disease. In order for a risk factor to reliably predict a cardiovascular event within a 30 day period, it must be able to select a population in which important coronary artery disease exists at the time. A history of angina, previous myocardial infarction, previous heart failure, and/or peripheral vascular disease will, with a reasonable degree of certainty, select such a population. Intuitively, a history of diabetes will not. Although coronary artery disease is the leading cause of death within the diabetic population, its presence is related to the duration of disease and not just the diagnosis. Thus, although a diagnosis of diabetes will reliably predict long term cardiovascular events, it will not do so over the short term. Many diabetic patients undergoing surgery will not have coronary artery disease, and this I suspect is the explanation for the poor performance of diabetes as a predictor of cardiovascular death in Howell’s study.

All this does not mean diabetes is not a risk factor, but rather other elements—in particular duration of disease and age of the patient—must also be incorporated before assigning risk. For example an elderly type II non-insulin-dependent diabetic could be considered at risk five years after the diagnosis of diabetes. However a type I insulin dependent diabetic may not be at significant risk of coronary artery disease until 15 to 20 years after diagnosis (although at this time he may still be younger than the previous patient). Although this makes risk prediction a more complex issue, accurately predicting a major cardiovascular event over the short term in a well controlled patient undergoing elective surgery is a complex and difficult task.

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Editor,—We are grateful for these comments on our study and for the opportunity to reply. The proposed explanation for the failure of diabetes mellitus to predict following anaesthesia and surgery may well be valid. As we pointed out, if this is the case, then the duration of diabetes should be included in the predictive model. We did not collect these data on our subjects and are therefore unable to test the hypothesis that including the duration of diabetic disease would improve the predictive power of the model. Adding this continuous variable would, of course, increase the number of adverse outcomes which needed to be studied to achieve adequate statistical power. Nevertheless, in the light of these comments, duration of diabetes should probably be examined as a potential predictive variable in future studies of perioperative risk.

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Walking extradurals

Editor,—We read with interest the editorial by Elton, Ali, and Mushambi on “walking extradurals” in labour1 and concur with their cautious assessment of the benefits of the combined spinal–epidural technique (CSE) for the obstetric patient. We too feel that inadvertent insertion of an extradural catheter into the subarachnoid space through a spinal needle puncture site is unlikely and would offer other more plausible mechanisms for the occasional unexpectedly high block obtained with the CSE technique. Blumgart and colleagues showed comparable cephalad extension of a pre-existing subarachnoid block in obstetric patients after extradural injections of saline or bupivacaine,2 concluding that the enhancement of the block was largely a volume effect. In an animal study, Swenson and colleagues found markedly increased morphine concentrations in cerebrospinal fluid following administration of extradural morphine adjacent to a dural puncture, compared with a control group.3 These studies suggest that there may be fundamental differences in the effects of drugs delivered extradurally using CSE compared with traditional extradural anaesthesia, and reinforce the need for cautious incremental dosage of local anaesthetics and opioids when this technique is employed.

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To date CSFs have been used for analgesia in more than 10 000 parturients at Queen Charlotte’s Hospital (and Crowhurst JA, Plaat F, unpublished data) and for analgesia and surgical anaesthesia in many more thousands of patients in Europe and elsewhere. Apart from successfully allowing > 80–90% of labouring parturients to be ambulant, one of the most significant features of the low dose bupivacaine-opioid CSE technique is the omission of the traditional epidural “test dose.”12 This aspect was not addressed by Elton and colleagues.

The authors state that the first use of any epidural catheter must be under the direct supervision of trained personnel, particularly when a test dose has not been given. This philosophy, based on traditional test doses, disregards the relative safety of today’s low dose combinations, even when they are given through misplaced or displaced catheters.65 Small doses of bupivacaine (2.5–10 mg) and fentanyl (20–25 μg) produce very predictable effects when given incrementally into the intrathecal or vascular compartments. Such effects are mild when compared with the potentially catastrophic sequelae which may accompany a positive traditional test dose of 15–20 mg of bupivacaine, or 45–50 mg of lignocaine with adrenaline. Of course, careful supervision of all drug administration is mandatory.

Further, the use of a traditional test dose removes any chance of achieving a selective block, which will permit safe ambulation and other coordinated motor activity.10–11

Second, as your authors acknowledge, meningitis has been associated with CSE, but also with traditional epidural and spinal methods.12–15 We dispute, however, that any “excess incidence” of meningitis has been shown with CSE. Case reports suggest that infectious complications are almost always associated with avoidable factors and careless technique.14–16 We disagree strongly with the statement that traditional epidural techniques are “inherently safe.” Careful patient selection, examination, and scrupulous attention to asepsis and antisepsis is mandatory; and no central, neural blocking technique is for the casual or inexperienced operator. Even with these safeguards, there is always some risk of infection, which for spinal and epidural is undoubtedly greater than for other injection methods such as intravenous, intramuscular, and so on.

Third, improved analgesia, ambulation, and increased maternal satisfaction have inevitably increased patient demand. But this should not be a reason to limit the availability of safe, effective pain relief, which at last is beginning to be regarded as a basic human right.17 Anaesthetists today should be aware that demand for acute pain services is increasing, and that more resources and personnel need to be directed to providing such services.

J A CROWHURST
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Editor,—Thank you for the opportunity to reply to this letter.

Drs Murray and Birks have rightly pointed out that administration of fluid into the epidural space immediately after a subarachnoid block at the same dermatome can result in an unexpectedly high block.

The most likely explanation for an unexpectedly high block following the epidural administration of drugs after combined spinal epidural is the passage of the epidural catheter into the cerebrospinal fluid following inadvertent dural puncture with the Tuohy rather than the spinal needle. Therefore care should be taken with any epidural to detect subarachnoid placement of the epidural catheter and minimize the effects of such administration. This risk should be the same whether this follows an epidural or a CSE. A further possibility is subdural placement of the epidural catheter and subsequent erosion of the catheter through the arachnoid mater into the CSF.12 It is unclear what the incidence of this problem is and whether it is more likely following a CSE.

The effect of fluid in the epidural space on the height of spinal anesthesia is well known, particularly where subarachnoid administration follows an epidural block, compared with an epidurally administered initial dose.2 With such a block is produced almost instantly with a far smaller initial dose, compared with an epidurally administered initial dose.2 With the synergistic opioid-bupivacaine combinations used today, such highly selective blockade is achievable only by minimizing the total dose. One of the fundamental advantages of a CSE is that such a block is produced almost instantly with a far smaller initial dose, compared with an epidurally administered initial dose.2 With CSE, a minuscule first dose is possible because the intrathecal compartment contains less cerebrospinal fluid than epidural tissue and vascularity compared with the epidural space. Thus, initial intrathecal administration fulfills the old pharmacological dictum: the smallest effective dose of any drug is the best dose.

For almost 17 years the bupivacaine–opioid CSE technique has been researched and refined in several countries. It is used not only in obstetrics, but also in many other areas of anaesthetic practice. To date CSFs have been used for analgesia in more than 10 000 parturients at Queen Charlotte’s Hospital (and Crowhurst JA, Plaat F, unpublished data) and for analgesia and surgical anaesthesia in many more thousands of patients in Europe and elsewhere.13

Apart from successfully allowing > 80–90% of labouring parturients to be ambulant, one of the most significant features of the low dose bupivacaine-opioid CSE technique is the omission of the traditional epidural “test dose.”12 This aspect was not addressed by Elton and colleagues.

The authors state that the first use of any epidural catheter must be under the direct supervision of trained personnel, particularly when a test dose has not been given. This philosophy, based on traditional test doses, disregards the relative safety of today’s low dose combinations, even when they are given through misplaced or displaced catheters.65 Small doses of bupivacaine (2.5–10 mg) and fentanyl (20–25 μg) produce very predictable effects when given incrementally into the intrathecal or vascular compartments. Such effects are mild when compared with the potentially catastrophic sequelae which may accompany a positive traditional test dose of 15–20 mg of bupivacaine, or 45–50 mg of lignocaine with adrenaline. Of course, careful supervision of all drug administration is mandatory.

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