NZMA news

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Chairman, NZMA

Funding for Medical Students

The New Zealand Medical Association is the largest medical organisation in New Zealand and the only one that represents all sectors of the medical profession – beginning at the student level and continuing throughout members' medical careers. This year will continue to be a busy one for our advocacy work with a diverse range of issues including medical student tuition fees, medical workforce shortages, GP viability and maternity services.

In terms of medical student tuition fees, the NZMA has welcomed a funding review for medical students, announced by the Minister for Tertiary Education Michael Cullen on 17 February. It's an important step towards reducing the debt burden on medical students and helping to address medical workforce shortages. The NZMA and the NZMSA have been expressing concerns about the fees situation for medical students for a number of years. Recent studies on student debt including one published in the NZ Medical Journal this month (www.nzma.org.nz), highlight that medical tuition fees strongly influence medical graduates' career decisions. Too many graduates choose to leave the country to work overseas. There are also far fewer graduates going into general practice, which in itself is already facing shortages. The article states that 9% of graduates surveyed would choose general practice. If you take into account that GPs make up 40% of the current medical workforce it is obvious that the shortage is only going to get worse.

While the NZMA fully supports a funding review, it is important for the Government to recognise that there are many factors affecting medical workforce shortages and we will continue to urge the Government to adopt a comprehensive strategy to resolve the need for medical workforce development.

NZMA Supporting Medical Students

We have a close relationship with the New Zealand Medical Students' Association and advocate alongside the NZMSA in its work, especially in the battle against the rising cost of medical education, particularly high student debt and fees. To highlight the negative effects of high student debt we published, in conjunction with NZMUSA and NZMSA, a casebook called 'Doctors & Debt: The Effect of Student Debt on New Zealand's Doctors.'

Too many graduates choose to leave the country to work overseas. There are also far fewer graduates going into general practice, which in itself is already facing shortages.

Having undertaken much work to raise awareness about debt and its effect on medical students – helping to promote the issue onto the Government's agenda – we were heartened at Labour's policy to abolish interest payments on student debts.

The NZMA also provides significant support for NZMSA including funding the attendance of the NZMSA President at NZMA forums and Council meetings, and we provide financial support for the NZMSA President to attend the Australia Medical Students Association Conference.

Advocacy for Doctors in Training

We have a Doctors-in-Training Council, of which the President of the NZMSA is a member. The Council advises the NZMA Board on issues relevant to doctors-in-training. At the DITC meeting in February a range of topics were discussed including UK registration, PGY1, medical workforce development, RDA MECA negotiations and the rural curriculum.

Other NZMA Advocacy 2006

The worsening shortage of medical professionals in New Zealand is a critical issue and needs to be addressed urgently. The NZMA has made a submission on a paper written by the Medical Reference Group, which looks at medical workforce shortages and covers issues such as student debt, retention and training.

The NZMA has continued to advocate for the right of private sector doctors to set their own fees. A six-weekly meeting has been established between GP leaders and the Ministry of Health to enable GP leaders to have more effective input into policy and implementation.

Find out more about the NZMA on the website:
www.nzma.org.nz

Prescribing of Antiepileptic Drugs (AEDs) during pregnancy

Continued from page 18

Epilepsy is a difficult condition to manage during pregnancy for a number of reasons. Firstly, inadequately controlled epilepsy is associated with dangers to mother and foetus. Secondly, most AEDs have known teratogenic effects.[11]

Anti-epileptic drugs (AEDs) can be divided into two groups:

Traditional (older) AEDs: Older drugs introduced before 1990 – Carbamazepine, Phenytoin, Sodium Valproate, Primidone, Phenobarbital

Newer AEDs: Newer drugs introduced after 1990 – Topiramate, Lamotrigine, Gabapentin, Vigabatrin

Teratogenicity with AEDs

The incidence of giving birth to a child with serious malformation in the general population is 2-3%. This rises to 4% if the mother is epileptic. If AEDs were taken by the mother, the incidence rises further to 5%. Abnormalities are more likely if mother takes more than one AED. However, these women are more likely to have severe epilepsy which may account partially for the increased risk.[3] Recent data from the Australian Pregnancy Register for Women on AEDs showed that out of 403 pregnancy outcomes for women taking AEDs, 87.8% resulted in a healthy birth, 6.5% had foetal malformations and the remaining 5.7% had spontaneous abortions or premature death in utero.[6]

Foetal malformations associated with AEDs

AEDs cause a characteristic pattern of abnormalities of varying severity commonly known as 'foetal anticonvulsant syndrome.' The problems seen most frequently are the minor dysmorphic abnormalities affecting the face and digits. Major abnormalities are attributed to all traditional AEDs but occur less frequently compared to minor abnormalities[3] (see Table 4).

Table 4: Foetal malformations associated with AEDs[3]

Minor abnormalities	Major abnormalities
V-shaped eyebrows Low-set ears Broad nasal bridge Irregular teeth or wide mouth Hypertelorism (wider than normal space between the eyes) Hypoplasia of nails & distal phalanges	Orofacial clefts Congenital heart disease (septal defects) Neural tube defects

Mechanism of teratogenesis due to AEDs

Several mechanisms have been proposed for teratogenic effects of AEDs. One theory, which focuses on the genetic predisposition principle of teratogenicity, is that unstable epoxides could interfere with the normal developmental process. Detoxification of these epoxides requires the enzyme epoxide hydrolase and a genetic defect in the activity of this enzyme in the foetus can increase teratogenic risk. Free radicals produced during the metabolism of AEDs can be cytotoxic. A genetic defect of free-radical scavenging enzymes can cause an excessive foetal exposure to these cytotoxins.

Another postulated mechanism for teratogenicity of AEDs is the induction of folic acid deficiency. Folic acid antagonists such as phenytoin, barbiturates, sodium valproate, and carbamazepine can cause folate malabsorption thus reducing serum folic acid levels which are thought to influence neural tube defects. The Medical Research Council study in 1991 reported a 70% reduction of neural tube defects recurrence among pregnant women who were supplemented with folic acid 4mg before conception and during gestation. All pregnant women are now advised to take 400mcg during the first 3 months of their pregnancy. All pregnant women with a family history of malformation or who are in a high risk category e.g. epileptics are advised to take 5mg of folic acid daily prior to conception and during the first 3 months of their pregnancies.

Vitamin K is needed so that the liver can produce clotting factors (eg prothrombin). The hepatic enzyme inducing AEDs (phenytoin, henobarbital, carbamazepine and topiramate) can cause vitamin K deficiency in the foetus. This results in early haemorrhagic disease of the newborn which manifests as intracranial haemorrhage in the newborn at birth. The exact mechanism of this defect is unknown but may involve induction of foetal liver microsomal enzymes that deplete the already low reserves of foetal vitamin K. This results in suppression of vitamin K dependent coagulation factors II, VII, IX and X. (Briggs & PJ article part 2) Vitamin K supplementation during pregnancy and for the neonate immediately after birth is recommended to prevent this from occurring. There are several regimens for vitamin K supplementation in pregnant women taking AEDs. The consensus guidelines recommend an oral daily dose of 20mg vitamin K to the mother from the 36th week of pregnancy and to give 1mg of vitamin K intramuscularly to the newborn at birth. The foetal dose is repeated after 12 hours. [3, 11, 12, 13, 14]

Clinical implications of pregnancy induced pharmacokinetic changes

Approximately one third of women with epilepsy will have an increase in seizure frequency when they become pregnant. Major seizures during pregnancy can lead to foetal hypoxia and lactic acidosis. Falls resulting from seizures can lead to trauma, early labour or miscarriage. This may occur due to hormonal changes or sleep deprivation but the main reason is thought to be pharmacokinetic changes which occur during pregnancy. These changes include an expansion in plasma volume, increased clearance rate and a change in protein binding.[12]

As most AEDs are acidic or neutral they are highly bound to serum albumin. During late pregnancy albumin levels fall with a corresponding decrease in the fraction of bound drug. This decrease in plasma protein binding leads to more free drug available for metabolism and clearance. The net effect is a decrease in the total (unbound and protein bound) plasma concentration of the AED during pregnancy.

It is helpful to have the baseline blood levels of AEDs taken at the beginning of pregnancy and also a baseline measurement of serum albumin. In women whose epilepsy is poorly controlled, an increase in seizures is more likely. Frequent or prolonged fits can cause miscarriage, intracranial haemorrhage in mother and premature labour. In extreme cases, seizures can cause alterations in placental blood flow and thus transfer of oxygen and nutrients to foetus, resulting in foetal hypoxia with bradycardia and brain damage. In these women who have poorly controlled epilepsy the dose of the AED may be increased to maintain a therapeutic level. The best practice is to adjust the dose of AED according to the woman's clinical condition corresponding to seizure frequency rather than blood AED levels. In some cases, clinicians measure levels every one to two months. This allows rapid dose adjustment to restore levels if seizures occur. If a dose of AED is increased during pregnancy then it should be titrated down to the original dose in the first few weeks post delivery. [3]

COMMONLY USED AEDS AND TERATOGENIC RISK DURING PREGNANCY

Traditional (older) AEDs introduced before 1990

The rates of major morphological abnormalities after foetal exposure to the older AEDs have been established at 4-6% for carbamazepine and pheytoin and 8% for sodium valproate.

Phenytoin

Risk factor: D [8]

Phenytoin is a hydantoin anticonvulsant whose teratogenic effects were first recognised in 1964. Foetal hydantoin syndrome (FHS) is a characteristic pattern of malformations which was first described in 1968. Clinical features of FHS are shown in Table 5.

Table 5: Foetal Hydantoin Syndrome[8]

Craniofacial	Limbs
Broad nasal bridge Wide fontanelle Low-set hairline Broad alveolar ridge Metopic ridging Short neck Ocular hypertelorism Microcephaly Cleft lip &/ palate Abnormal or low-set ears Epicanthal folds Ptosis of eyelids Coloboma Coarse hair scalp	Small or absent nails Hypoplasia of distal phalanges Altered palmar creases Digital thumb Dislocated hip

Other foetal malformations associated with phenytoin include impaired physical and mental growth, congenital heart defects and cleft lip and/or palate.[15] Phenytoin can cause haemorrhagic disease of the newborn and prophylactic treatment with vitamin K to prevent this condition has been discussed earlier. Phenytoin may also induce folic acid deficiency in the epileptic patient which has been linked to an increased risk of neural tube defects in the neonate. Folic acid supplementation at a dose of 5mg daily is recommended for epileptic women who are contemplating pregnancy and they should continue taking it up to week 12 of their pregnancy.

Phenytoin has non-linear pharmacokinetics and a narrow therapeutic window. It is highly protein bound (90-93%) and is cleared mainly by saturable hepatic metabolism. A fall in the total serum concentration which results in a lack of seizure control requires an increase in dosage of phenytoin. This decrease in protein binding of phenytoin may be an important mechanism for the decrease in total drug concentration during pregnancy as it is the free drug that becomes available for enhanced metabolism.[3, 12]

Sodium valproate (Valproic acid)

Risk factor: D [8]

Sodium valproate is known to increase the risk of neural tube defects. Exposure during days 17 to 30 after conception carries a 1-2% absolute risk of neural tube defects in the neonate. A characteristic pattern of minor facial defects has been associated with sodium valproate which includes trigonocephaly, tall forehead with bifrontal narrowing, epicanthic folds, medial deficiency of eyebrows and flat nasal bridge. The most common major congenital defects observed were neural tube defects, congenital heart disease, cleft lip and palate, genital anomalies and limb defects.[3,8] Some studies refer to this characteristic pattern of malformations as 'foetal valproate syndrome'.

Data from the Australian Pregnancy Register showed that the foetal malformation rate was significantly greater in pregnancies exposed to valproate in the first trimester compared with those exposed to all other AEDs. The mean daily dose of valproate was found to be significantly higher in those women who had children with foetal malformations than in those who had children without foetal malformations (1975mg vs. 1128mg), showing that risk of foetal malformations may be correlated with the dose of valproate used.[6]

Sodium valproate is rapidly absorbed and highly protein bound to plasma albumin (88-92%). The interpretation of its pharmacokinetics is limited by large fluctuations in the concentration-time profile, wide therapeutic index and concentration dependent protein binding. Analysis of unbound sodium valproate concentrations is not routinely done and there is no established therapeutic range. Dose adjustments during pregnancy are best made by clinical observations in conjunction with therapeutic in serum concentrations of sodium valproate.[12]

Carbamazepine

Risk factor: D [8]

Carbamazepine is a tricyclic anticonvulsant which has been in use since 1962. In earlier reviews, carbamazepine was recommended as the drug of choice during pregnancy as it was thought to present a lower risk to the foetus. However, in 1991 an association between carbamazepine and spina bifida was confirmed. The risk of this defect is thought to be about 1%. [16]

Investigators conducted a prospective study evaluating pregnancy outcomes for 72 women treated with carbamazepine during early pregnancy and compared this group to a control group. The investigators concluded that carbamazepine exposure was associated with a pattern of congenital malformations whose principal features consisted of craniofacial defects, fingernail hypoplasia, developmental delay and neural tube defects. This characteristic pattern of abnormalities was termed 'foetal carbamazepine syndrome'. These defects were noted to be similar to those observed with the foetal hydantoin syndrome described earlier for phenytoin. As both carbamazepine and phenytoin are metabolised through the arene oxide pathway, a possible mechanism for the teratogenicity of these two drugs was proposed which attributed their teratogenicity to epoxide intermediates.[8]

A 2000 study, using data from the MADRE (Malformation and Drug Exposure) surveillance project assessed the human teratogenicity of antiepileptics. 299 infants were exposed in the first trimester of their conception to antiepileptics. Of these, 46 infants were exposed to carbamazepine alone. A statistically significant association $p \leq 0.05$ was found between carbamazepine monotherapy and spina bifida.[8]

Like phenytoin, carbamazepine can also cause folate and vitamin K deficiencies which can be corrected by supplementation.

Carbamazepine is protein bound (70-80%) and has a relatively slow absorption. Carbamazepine is eliminated by hepatic metabolism. Dose intervals and sample times are critical in interpreting serum concentrations. Large peak-trough fluctuations can be minimised by using controlled release formulations. The concentration of the pharmacologically active metabolite (carbamazepine-10-11-epoxide) was reported to increase during pregnancy possibly due to an increase in carbamazepine metabolism. [12]

Newer AEDs introduced after 1990

New AEDs should not be used in pregnant women unless absolutely necessary because there is not much information available about the risks associated with them. The majority of information is from animal reproductive toxicology studies which are not fully predictive of human teratology.

There is little information regarding the pharmacokinetics of the new AEDs in humans and their safety during pregnancy. There have been reports of decreased lamotrigine levels during pregnancy. Topiramate, felbamate and oxycarbazepine have low levels of protein binding. Vigabatrin and gabapentin do not bind to protein. New AEDs are eliminated through the body by renal clearance.[17]

Topiramate

Risk factor: not assigned

Teratology studies in animals reported that topiramate induced right-sided ectrodactyly (congenital absence of all or part of a digit) in rats, whereas rib and vertebral malformations were observed in rabbits. Although topiramate is used widely in the treatment of patients with epilepsy, few pregnant women have taken it. In one case report, multiple minor abnormalities such as a third fontanelle, blunt distal phalanges and fifth nail hypoplasia were seen in a child born to a mother treated with topiramate 700mg twice daily as monotherapy throughout gestation.[17, 18]

Lamotrigine

Risk factor: C [8]

Lamotrigine is chemically unrelated to existing antiepileptic drugs. It is commonly used as an adjunctive therapy for the treatment of partial seizures in patients with epilepsy.[8] Lamotrigine is a weak inhibitor of dihydrofolate reductase. The antifolate activity of other established AEDs is associated with teratogenicity but this has not been proven for lamotrigine. Data on the effects of lamotrigine in pregnant women is limited. Two studies conducted to date were unable to attribute the foetal malformations seen to lamotrigine because the women were exposed to other AEDs during their pregnancy.[3]

An interim report from the Lamotrigine Pregnancy Registry was issued in 2000. A total of 362 prospective pregnancies were enrolled in the registry in the period between 1st September 1992 through to 31st March 2000. Of these, outcomes are known for 244 pregnancies (248 outcomes – including some multiple births). Lamotrigine monotherapy was used in 98 outcomes with the earliest exposure in the 1st trimester. These exposures resulted in nine spontaneous pregnancy losses, 27 elective abortions (two with birth defects), one death of foetus, 14 live infants with foetal malformations and 186 infants without foetal malformation (includes 2 sets of twins). The foetal malformations seen with lamotrigine monotherapy in the 1st trimester were:

Oesophageal malformation
Cleft soft palate
Right club foot

The Lamotrigine Pregnancy Registry advisory committee concluded that the number of exposed pregnancies outcomes represents a sample of insufficient size to reach definite conclusions regarding the safety of lamotrigine in pregnancy.[8, 17, 19]

Gabapentin

Risk factor: C [8]

Gabapentin is an antiepileptic used as an adjunctive therapy for the treatment of partial seizures in patients with epilepsy. Animal studies have shown that gabapentin at high doses is fetotoxic in rodents. Fetotoxicity is manifested as delayed ossification of bones in the skulls, vertebrae, forelimbs and hind limbs. [3, 8]

In 1998, a non-interventional cohort study described the outcomes of pregnancies in women who had been prescribed newly marketed drugs by general practitioners in England. Data was obtained by questionnaires sent out to the prescribing physicians. Gabapentin was taken during the 1st trimester in 17 pregnancies. The outcomes of these pregnancies included two spontaneous abortions, four elective abortions and 11 normal newborns. Although no congenital malformations were observed the study lacked the sensitivity to identify minor anomalies. A review in 1996 reported 16 pregnancies exposed to gabapentin. The outcomes of these pregnancies included five elective abortions, one ongoing pregnancy, seven normal infants and three infants with foetal malformations. No specific information was provided about the foetal malformations.
At present there is limited human data which does not allow an accurate assessment of the safety of gabapentin in pregnancy. A pregnancy register has been established to get more extensive and detailed information about the safety of this new drug in pregnant women.[8, 17]

Vigabatrin

Risk factor: not assigned

Vigabatrin induced cleft anomalies in rabbits when administered during pregnancy. A case report describes a pregnancy exposed to vigabatrin, carbamazepine and dexamethasone. This pregnancy resulted in an infant with multiple congenital abnormalities including bilateral anophthalmia, situs viscerum inversus, levo-isomerism, single ventricle, enlargement of the third ventricle and clubfoot.[17, 20]

SUMMARY

Best Practice guidelines for the management of pregnant women with epilepsy [11, 12, 13, 14, 21, 22]

Before Conception (should begin at least three months before conception)

- Women should be given preconception counselling about the potential risk of increased seizure activity in pregnancy and that the seizures carry a risk to the foetus, to ensure that they do not avoid taking their AEDs.

- Adequate patient information regarding the increased incidence of major malformations and risk of teratogenicity due to AEDs should be provided.

- Women should be referred to a neurologist and obstetrician to reassess treatment. Any medication reduction or substitution should take place before conception.

- Gradual drug discontinuation (over at least three months) should be considered if the patient has been seizure free for 2 or more years.

- Traditional AEDs should be preferred in women of child bearing age planning pregnancy. This is because the patient can be provided with adequate information about the risks and benefits of the AED.

- Doses of AEDs may need adjusting due to pharmacokinetic changes caused by pregnancy.

• Peak serum drug levels should be reduced by increasing the dosing frequency or using low doses of controlled release preparations.

• Avoid multiple drugs therapy as it is associated with an increased risk of foetal malformation. Use the lowest dose possible of a single agent.

• Women should start taking folic acid 5mg daily 3 months before conception and should continue taking it up to week 12 (during the 1st trimester) of the pregnancy.

After Conception

• If epilepsy is well controlled current medication should be maintained.

• Therapeutic drug monitoring should be performed every three to four months or more frequently if seizure control is not achieved.

• Alpha-fetoprotein is a glycoprotein produced initially by the yolk sac and then by the foetal liver and gastrointestinal tract. α-fetoprotein can be measured in amniotic fluid and in maternal serum and is now used widely as a marker in prenatal maternal serum screening programs. A maternal α-fetoprotein test can be performed at 16 weeks gestation as it can detect spina bifida, neural tube defects and major cardiac malformations.

• Targeted foetal ultrasound scan at 18 weeks should be done to detect spina bifida, open neural tube defects and major cardiac malformations.

• Amniocentesis and other specialised tests should be performed as required. Amniocentesis can be offered to women as a somewhat more accurate measure of alpha-fetoprotein levels than maternal serum testing, although clearly there are increased risks associated with this invasive procedure. Generally, amniocentesis is used when satisfactory ultrasound examination is not possible, for example in extremely obese women.

• If a woman is taking phenytoin, phenobarbitone or carbamazepine, then oral vitamin K (phytomenadione) at a dose of 20mg daily should be taken by the pregnant woman late in third trimester (week 36 onwards) to prevent neonatal haemorrhage.

LABOUR, DELIVERY AND BIRTH

Delivery should be in hospital due to the increased risk of seizures (1 to 2%) during labour or after birth. Convulsive seizures at the time of labour and delivery are commonly treated with administration of intravenous benzodiazepines or phenytoin. Caesarean section is often necessary.

After Birth

• Infant will need vitamin K administered intramuscularly on delivery with another dose 12 hours later.

• Any increase in the dose of antiepileptic therapy during pregnancy should be reviewed at this time. In the postpartum period, free serum levels should be measured for the first eight weeks to avoid drug toxicity, which may result from the shift back to prepregnancy pharmacokinetics.

• All AEDs are excreted into breast milk. With the exception of phenobarbital, primidone (high concentrations excreted in breast milk) and vigabatrin (can cause visual field defects), mothers taking antiepileptic drugs should be encouraged to breast feed. The amount of drug the baby would receive is likely to be small.

CONCLUSION

Proper seizure control is the primary goal in treating women with epilepsy. Patients should understand the risks associated with uncontrolled seizures as well as the teratogenicity of the AEDs. When AEDs are used during pregnancy, the most appropriate first line drug for the seizure type should be used at the lowest effective dose. Proper management before, during and after pregnancy can lead to a favourable outcome for the majority (90%) of pregnancies in women with epilepsy.

Continuing Education

Readers are invited to submit their answer to the question in the case "What are the risks associated with taking phenytoin during pregnancy?" and to formulate recommendations for treatment of Mrs R's epilepsy during pregnancy.

Submit answers by emailing them to:
medstudent.journal@stonebow.otago.ac.nz

Answers will be published in the next issue of the journal. The best answer will win a medical textbook.

DISCLAIMER

Please note that the best practice guidelines and the information above regarding the management of epilepsy during pregnancy are based on the literature reviewed by the author. This review is intended only as a guide for the clinical management of pregnant women with epilepsy and may not cover all diagnostic or therapeutic options available. Consult the appropriate medical specialists and your local hospital medicines information service for advice on the management of individual patients.

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Clinicians *Medical Education Convention Aotearoa*

Aim: To bring together both clinical and pre clinical medical students from across New Zealand, in order to enhance their interest and motivation for the field of medicine, whilst at the same time developing collegiality and a wider knowledge base.

Method: Meet annually in the form of a convention where students will attend lectures on current research and practice, partake in workshops designed to enhance their clinical skills, discuss issues relevant to medicine now and in the future, and attend social functions that will highlight the importance of collegiality in medicine.

Results: A happier, more motivated and intellectual group of future doctors with an interest in advancing medical knowledge, who are aware of and not afraid to, confront big picture issues. This should in turn lead to higher quality health care provision for all New Zealanders across all specialities.

Conclusion: Set aside the weekend of September 15-17, 2006 and keep an eye out for more information on how you can join the pilgrimage towards a better tomorrow. Register your interest now and receive more information by e-mailing

kilja036@student.otago.ac.nz

Subject: Clinicians MECA'06.

medical leadership development seminar

The New Zealand Medical Students' Association is hosting the inaugural Medical Leadership Development Seminar (MLDS) in Wellington this year. This event will bring together 70 medical student leaders from around the nation and a selection of outstanding speakers from key social, political, humanitarian, management and clinical leadership roles. Speakers include

Hon. Pete Hodgson
Minister of Health

Mr Ron Paterson
Health and Disability Commissioner

Sir Thomas Davis
Former Cook Islands Prime Minister

Hon. Dame Silvia Cartwright
Governor-General of New Zealand

These key speakers will be presenting alongside practising physicians from diverse specialty groups. The MLDS represents a unique opportunity for New Zealand medical students to interact with these outstanding health leaders and gain an awareness of contemporary health issues.

We are very excited about this event and look forward to meeting the participants in July.

New Zealand Medical Students' Association

New Zealand Medical Students' Association
www.nzmsa.org.nz
te rōpū akonga rongoā o aotearoa