increased gradually to every 4 to 6 weeks. Treatment may be continued for up to 5 years after remission.

Improvement may not be seen until a total dose of 300 to 500 mg has been given. If no major improvement has occurred after a total of 1 g has been given (excluding the test dose) therapy should be stopped; alternatively in the absence of toxicity, 100 mg may be given weekly for a further 6 weeks; should there be no response at this dose other forms of therapy should be tried. In patients who relapse while receiving maintenance therapy, the interval between doses should be reduced to one week and should not be increased again until control has been obtained; however, if no response is obtained within 2 months, alternative treatment should be used. It is important to avoid complete relapse since a second course of gold therapy is not usually effective.

For doses in juvenile idiopathic arthritis, see Administration in Children, below.

NSAIDs may be continued when sodium aurothiomalate therapy is begun.

Other gold compounds that have been used include auranofin (p.25), aurothioglucose (p.26), aurotioprol (p.26), gold keratinate (p.62), and sodium aurotiosulfate (below).

**Administration in children.** For children with progressive juvenile idiopathic arthritis the suggested initial weekly dose of sodium aurothiomalate is 1 mg/kg by deep intramuscular injection to a maximum of 50 mg weekly (one-tenth to one-fifth of the calculated initial weekly dose may be given for 2 to 3 weeks to test the patient's tolerance). Weekly doses should continue until signs of remission occur, at which point the dosage interval may be increased to fortnightly. With full remission, the dosage interval may again be increased gradually to every 4 weeks. If no improvement has occurred after 20 weeks, the dose could be raised slightly or another antirheumatic drug tried. For the view that use of gold compounds is no longer appropriate to treat juvenile idiopathic arthritis see Rheumatic Disorders, below.

**Asthma.** For comment on the use of parenteral gold compounds in the treatment of asthma, see under Auranofin, p.26.

**Pemphigus and pemphigoid.** Corticosteroids are the main treatment for blistering in pemphigus and pemphigoid (p.1582). Intramuscular gold therapy has been used concomitantly to permit a reduction in corticosteroid dosage although evidence for the steroid-sparing effect is lacking;[1,2] it has been suggested that gold therapy should be reserved for patients who cannot tolerate corticosteroids or in whom they are contra-indicated.[1]


1. Bystryn J-C, Steinman NM. The adjuvant therapy of pemphigus: an update. *Arch Dermatol* 1996; **132:** 203–12.
2. Pandya AG, Dyke C. Treatment of pemphigus with gold. *Arch Dermatol* 1998; **134:** 1104–7.


**Rheumatic disorders.** Gold compounds are among the disease-modifying antirheumatic drugs (DMARDs) that may be used in the treatment of rheumatoid arthritis (p.11). Although toxicity has now reduced its popularity, intramuscular gold has long been used for the treatment of rheumatoid arthritis[1-4] and is often the standard against which the efficacy of other treatments is measured. Oral gold is less toxic but is also much less effective. It is unclear if there are differences between available intramuscular forms, but a study[5] in 120 patients converted from aurothioglucose to aurothiomalate found that 29 withdrew from the latter drug within 12 months, mostly because of lack of efficacy or the development of adverse effects not seen with the previous drug.

Gold compounds have also been used in the treatment of juvenile idiopathic arthritis (p.10); however, the *BNFC* states that gold is no longer used for this indication.

Gold compounds may also be of benefit in psoriatic arthritis (see under Spondyloarthropathies, p.13).


1. Epstein WV, *et al.* Effect of parenterally administered gold therapy on the course of adult rheumatoid arthritis. *Ann Intern Med* 1991; **114:** 437–44.
2. Anonymous. Gold therapy in rheumatoid arthritis. *Lancet* 1991; **338:** 19–20.
3. Klinkhoff AV, Teufel A. How low can you go? Use of very low dosage of gold in patients with mucocutaneous reactions. *J Rheumatol* 1995; **22:** 1657–9.
4. Clark P, *et al.* Injectable gold for rheumatoid arthritis. Available in The Cochrane Database of Systematic Reviews; Issue 4. Chichester: John Wiley; 1997 (accessed 13/11/06).
5. van Roon, EN, *et al.* Parenteral gold preparations: efficacy and safety of therapy after switching from aurothioglucose to aurothiomalate. *J Rheumatol* 2005; **32:** 1026–30.


### Preparations

***BP 2008:*** Sodium Aurothiomalate Injection;
***USP 31:*** Gold Sodium Thiomalate Injection.

**Proprietary Preparations** (details are given in Part 3)
***Austral.:*** Myocrisin; ***Austria:*** Tauredon; ***Canad.:*** Myochrysine; ***Cz.:*** Tauredon; ***Denm.:*** Myocrisin; ***Fin.:*** Myocrisin; ***Ger.:*** Tauredon; ***Gr.:*** Miocrin; Myocrysine†; Tauredon; Tuaredon†; ***Hung.:*** Tauredon†; ***Irl.:*** Myocrisin; ***Neth.:*** Tauredon; ***Norw.:*** Myocrisin; ***NZ:*** Myocrisin; ***Port.:*** Tauredon; ***S.Afr.:*** Myocrisin†; ***Singapore:*** Miocrin; ***Spain:*** Miocrin; ***Swed.:*** Myocrisin; ***Switz.:*** Tauredon; ***Thai.:*** Myocrisin; ***UK:*** Myocrisin; ***USA:*** Aurolate; Myochrysine.

## Sodium Aurotiosulfate *(rINN)*

Aurotiosulfate de Sodium; Aurotiosulfato de sodio; Gold Sodium Thiosulphate; Natrii Aurotiosulfas; Natrii Aurotiosulphas; Natriumaurotiosulfaatti; Natriumaurotiosulfat; Sodium Aurothiosulphate; Sodium Dithiosulfatoaurate.

Натрия Ауротиосульфат

$Na_3Au(S_2O_3)_2,2H_2O$ = 526.2.

*CAS — 10233-88-2 (anhydrous sodium aurotiosulfate); 10210-36-3 (sodium aurotiosulfate dihydrate).*
*ATC — M01CB02.*
*ATC Vet — QM01CB02.*

### Profile

Sodium aurotiosulfate has a gold content of about 37%. It has similar actions and uses to those of sodium aurothiomalate (p.122). It is given by intramuscular injection in a usual dose of 56.1 mg every 5 to 7 days.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***Arg.:*** Crytion; ***Chile:*** Crytioro; ***Ital.:*** Fosfocrisolo.

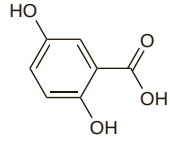
## Sodium Gentisate *(rINN)*

Gentisate de Sodium; Gentisato de sodio; Gentisato Sodico; Natrii Gentisas. Sodium 2,5-dihydroxybenzoate dihydrate.

Натрия Гентизат

$C_7H_5NaO_4,2H_2O$ = 212.1.

*CAS — 490-79-9 (gentisic acid); 4955-90-2 (anhydrous sodium gentisate).*



*(gentisic acid)*

**Pharmacopoeias.** In *Fr.*

### Profile

Sodium gentisate has been used as an analgesic in the treatment of musculoskeletal and joint disorders. It is also used as a preservative.

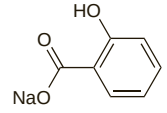
## Sodium Salicylate

Natrii salicylas; Natrio salicilatas; Natriumsalicylat; Natriumsalisylaatti; Nátrium-szalicilát; Salicilato sódico; Salicylan sodný; Sodium, salicylate de; Sodu salicylan; Sodyum Salisilat. Sodium 2-hydroxybenzoate.

Салицилат Натрия

$C_7H_5NaO_3$ = 160.1.

*CAS — 54-21-7.*
*ATC — N02BA04.*
*ATC Vet — QN02BA04.*



**Pharmacopoeias.** In *Eur.* (see p.vii), *Int.*, *Jpn*, *US*, and *Viet.*

**Ph. Eur. 6.2** (Sodium Salicylate). Colourless small crystals or shiny flakes, or white or almost white, crystalline powder. Freely soluble in water; sparingly soluble in alcohol. Store in airtight containers. Protect from light.

**USP 31** (Sodium Salicylate). Amorphous or microcrystalline powder or scales. It is colourless or has not more than a faint pink tinge. It is odourless or has a faint characteristic odour. A freshly made 10% solution in water is neutral or acid to litmus. Freely (and slowly) soluble in water and in glycerol; very soluble in boiling water and in boiling alcohol; slowly soluble in alcohol. Protect from light.

### Adverse Effects, Treatment, and Precautions

As for Aspirin, p.20.

Although sodium salicylate has been used in the treatment of rheumatic fever, its high sodium content may cause problems in patients with cardiac complications.

The use of aspirin and other acetylated salicylates is generally not recommended for children because of the risk of Reye's syndrome, unless specifically indicated. Some licensed drug information extends this precaution to sodium salicylate.

**Effects on the eyes.** Retinal haemorrhages were reported in a 60-year-old woman taking sodium salicylate 6 g daily by mouth for 2 months and in a 10-year-old girl taking sodium salicylate, 4 g daily by mouth, for 40 days.[1] In both cases the haemorrhages gradually resolved after the treatment was stopped.


1. Mortada A, Abboud I. Retinal haemorrhages after prolonged use of salicylates. *Br J Ophthalmol* 1973; **57:** 199–200.


### Interactions

For interactions associated with salicylates, see Aspirin, p.23.

### Uses and Administration

Sodium salicylate is a salicylic acid derivative that has analgesic, anti-inflammatory, and antipyretic actions similar to those of aspirin (p.23). Sodium salicylate 1 g is equivalent to about 1.1 g of aspirin. It is used in the treatment of pain, fever, and in rheumatic disorders such as osteoarthritis and rheumatoid arthritis. The usual oral dose of sodium salicylate for pain or fever is 325 to 650 mg every four hours as required. The oral dose for rheumatic disorders is 3.6 to 5.4 g daily in divided doses. Sodium salicylate has also been used in the symptomatic treatment of rheumatic fever but its high sodium content may cause problems in patients with cardiac complications.

Sodium salicylate has also been given by intravenous infusion and topically.

### Preparations

***USP 31:*** Sodium Salicylate Tablets.

**Proprietary Preparations** (details are given in Part 3)
***Canad.:*** Dodds†; Saliject; ***NZ:*** Hairscience Shampoo†; ***Turk.:*** Enter-Sal; ***UK:*** Jackson's Pain & Fever.

**Multi-ingredient:** ***Braz.:*** A Saude da Mulher; Abacateirol†; Pilulas De Witt's†; ***Canad.:*** Plax; Thunas Tab for Menstrual Pain†; ***Chile:*** Eucerin Shampoo Anticaspa; ***Fr.:*** Brulex; ***Ger.:*** Gelonida NA†; ***Hong Kong:*** Gly Thymol; ***S.Afr.:*** Colphen; Doans Backache Pills; Ilvico; TCP; ***UK:*** Antiseptic Mouthwash; Doans Backache Pills; TCP; ***USA:*** Cystex; Scot-Tussin Original 5-Action; Tussirex; ***Venez.:*** Boncilin†; Inquilim†.

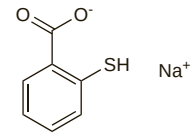
## Sodium Thiosalicylate

Tiosalicilato sódico. Sodium 2-sulfanylbenzoate.

Тиосалицилат Натрия

$C_7H_5O_2NaS$ = 176.2.

*CAS — 134-23-6.*



### Profile

Sodium thiosalicylate is a salicylic acid derivative (see Aspirin, p.20) that has been used parenterally in the treatment of musculoskeletal disorders, osteoarthritis, rheumatic fever, and acute gout.

### Preparations

**Proprietary Preparations** (details are given in Part 3)
***USA:*** Rexolate†.

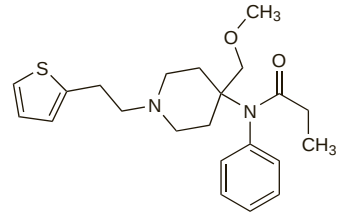
# Sufentanil *(BAN, rINN)* ⊗

R-30730; Sufentaniili; Sufentanilis; Sufentanilo; Sufentanilum; Szufentanil. *N*-{4-(Methoxymethyl)-1-[2-(2-thienyl)ethyl]-4-piperidyl}propionanilide.

Суфентанил

$C_{22}H_{30}N_2O_2S$ = 386.6.

*CAS — 56030-54-7.*
*ATC — N01AH03.*
*ATC Vet — QN01AH03.*



**Pharmacopoeias.** In *Eur.* (see p.vii).
**Ph. Eur. 6.2** (Sufentanil). A white or almost white powder. Practically insoluble in water; freely soluble in alcohol and in methyl alcohol. Protect from light.

### Sufentanil Citrate *(BANM, USAN, rINNM)* ⊗

Citrato de sufentanilo; R-33800; Sufentaniilisitraatti; Sufentanil citrát; Sufentanil, citrate de; Sufentanil Sitrat; Sufentanilcitrat; Sufentanili citras; Sufentanilio citratas; Szufentanil-citrát. *N*-{4-(Methoxymethyl)-1-[2-(2-thienyl)ethyl]-4-piperidyl}propionanilide citrate.

Суфентанила Цитрат

$C_{22}H_{30}N_2O_2S,C_6H_8O_7$ = 578.7.
*CAS — 60561-17-3.*
*ATC — N01AH03.*
*ATC Vet — QN01AH03.*

**Pharmacopoeias.** In *Eur.* (see p.vii) and *US.*
**Ph. Eur. 6.2** (Sufentanil Citrate). A white or almost white powder. Soluble in water and in alcohol; freely soluble in methyl alcohol. Protect from light.
**USP 31** (Sufentanil Citrate). A white powder. Soluble in water; sparingly soluble in alcohol, in acetone, and in chloroform; freely soluble in methyl alcohol. Store at a temperature of 25°, excursions permitted between 15° and 30°.

**Stability.** Sufentanil (as the citrate) diluted to 50 micrograms/mL with sodium chloride 0.9% remained stable for at least 14 days when stored at room temperature in PVC reservoirs for portable patient-controlled systems.[1]

1. Chapalain-Pargarde S, *et al.* Microbiological and physicochemical stability of fentanyl and sufentanil solutions for patient-controlled delivery systems. *J Pain Symptom Manage* 2006; **32:** 90–7.

## Dependence and Withdrawal

As for Opioid Analgesics, p.101.

## Adverse Effects, Treatment, and Precautions

As for Opioid Analgesics in general, p.102 and Fentanyl, p.56.

**Breast feeding.** Concentrations of sufentanil were similar in colostrum and serum in 7 women given sufentanil by continuous epidural infusion during the first postoperative day after caesarean section. In the light of its poor oral availability such an amount was not considered to be a hazard to the breast-fed infant, and a maternal dose of 5 micrograms/hour epidurally was considered to be safe for such infants.[1]

1. Ausseur A, *et al.* Continuous epidural infusion of sufentanil after caesarean section: concentration in breast milk. *Br J Anaesth* 1994; **72** (suppl 1): 106.

**Effects on the cardiovascular system.** For a reference to the effects of sufentanil on histamine release compared with some other opioids, see under Pethidine, p.114.

**Effects on the nervous system.** There have been reports of tonic-clonic movements or seizures in a few patients receiving sufentanil.[1] There was no evidence of cortical seizure activity in a patient whose EEG was recorded,[2] suggesting that the observed myoclonus was not a convulsion or seizure.

1. Zaccara G, *et al.* Clinical features, pathogenesis and management of drug-induced seizures. *Drug Safety* 1990; **5:** 109–51.
2. Bowdle TA. Myoclonus following sufentanil without EEG seizure activity. *Anesthesiology* 1987; **67:** 593–5.

**Effects on the respiratory system.** Sufentanil, like other opioid agonists, causes dose-related respiratory depression. There have been reports of significant respiratory depression associated with chest wall rigidity in the early postoperative period after anaesthesia with intravenous sufentanil.[1,2] Respiratory depression has also been reported after intrathecal sufentanil for postoperative analgesia[3] and labour pain.[4]. A retrospective chart review[3] of a 6-year period, during which 4870 patients received intrathecal sufentanil for the management of labour pain, found that the case above was the only one of respiratory arrest reported in the group.

1. Goldberg M, *et al.* Postoperative rigidity following sufentanil administration. *Anesthesiology* 1985; **63:** 199–201.
2. Chang J, Fish KJ. Acute respiratory arrest and rigidity after anesthesia with sufentanil: a case report. *Anesthesiology* 1985; **63:** 710–11.
3. Fournier R, *et al.* Respiratory depression after 5 μgrams of intrathecal sufentanil. *Anesth Analg* 1998; **87:** 1377–8.
4. Ferouz F, *et al.* Risk of respiratory arrest after intrathecal sufentanil. *Anesth Analg* 1997; **85:** 1088–90.

**The elderly.** The pharmacokinetics of sufentanil in elderly patients have been variable in different studies, but a review[1] considered that there had been no evidence overall for differences between the elderly and younger adults. Nevertheless, as with fentanyl, reduced initial doses have been advised in the elderly.

1. Monk JP, *et al.* Sufentanil: a review of its pharmacological properties and therapeutic use. *Drugs* 1988; **36:** 286–313.

**Handling.** Avoid contact with skin and the inhalation of particles of sufentanil citrate.

**Obesity.** The elimination half-life and volume of distribution of sufentanil were increased in obese subjects.[1,2] Licensed product information recommends that for obese patients more than 20% above ideal body-weight the dosage of sufentanil should be determined on the basis of the patients' lean body-weight.

1. Schwartz AE, *et al.* Pharmacokinetics of sufentanil in the obese. *Anesthesiology* 1986; **65** (suppl 3A): A562.
2. Schwartz AE, *et al.* Pharmacokinetics of sufentanil in obese patients. *Anesth Analg* 1991; **73:** 790–3.

## Interactions

For interactions associated with opioid analgesics, see p.103.

**Benzodiazepines.** For the effects of using opioids such as sufentanil with benzodiazepines, see Analgesics under Interactions of Diazepam, p.989.

## Pharmacokinetics

After parenteral doses sufentanil citrate has a rapid onset and short duration of action. The terminal elimination half-life of sufentanil is about 2.5 hours. It is extensively bound to plasma proteins (about 90%). It is metabolised in the liver and small intestine by *N*-dealkylation and *O*-demethylation and the inactive metabolites are excreted in the urine and faeces. About 80% of a dose is excreted within 24 hours and 2% is eliminated as unchanged drug. Sufentanil crosses the placenta and is distributed into breast milk.

◊ The pharmacokinetics of sufentanil have been reviewed.[1,2] Sufentanil is very lipid-soluble. Like alfentanil it is highly bound to plasma proteins, mainly to $\alpha_1$-acid glycoprotein. The elimination half-life lies between that of alfentanil and fentanyl. The manufacturers of sufentanil have given values for a three-compartment pharmacokinetic model with a distribution half-life of 1.4 minutes, a redistribution half-life of 17.1 minutes, and an elimination half-life of 164 minutes. Accumulation may be relatively limited when compared with fentanyl. In practice the pharmacokinetics of sufentanil may vary according to the age and condition of the patient and the procedures undertaken. For example, the elimination half-life of sufentanil has been reported to be longer in patients undergoing cardiac surgery (595 minutes),[3] in hyperventilated patients (232 minutes),[4] in those undergoing abdominal aortic surgery (more than 12 hours),[5] and in ventilated intensive care patients under sedation (25.5 hours).[6]

1. Monk JP, *et al.* Sufentanil: a review of its pharmacological properties and therapeutic use. *Drugs* 1988; **36:** 286–313.
2. Scholz J, *et al.* Clinical pharmacokinetics of alfentanil, fentanyl and sufentanil: an update. *Clin Pharmacokinet* 1996; **31:** 275–92.
3. Howie MB, *et al.* Serum concentrations of sufentanil and fentanyl in the post-operative course in cardiac surgery patients. *Anesthesiology* 1984; **61:** A131.
4. Schwartz AE, *et al.* Pharmacokinetics of sufentanil in neurosurgical patients undergoing hyperventilation. *Br J Anaesth* 1989; **63:** 385–8.
5. Hudson RJ, *et al.* Pharmacokinetics of sufentanil in patients undergoing abdominal aortic surgery. *Anesthesiology* 1989; **70:** 426–31.
6. Ethuin F, *et al.* Pharmacokinetics of long-term sufentanil infusion for sedation in ICU patients. *Intensive Care Med* 2003; **29:** 1916–20.

**Administration.** References to the pharmacokinetics of sufentanil given epidurally,[1,2] intrathecally,[1] or transdermally.[3]

1. Ionescu TI, *et al.* Pharmacokinetic study of extradural and intrathecal sufentanil anaesthesia for major surgery. *Br J Anaesth* 1991; **66:** 458–64.
2. Hansdottir V, *et al.* The cerebrospinal fluid and plasma pharmacokinetics of sufentanil after thoracic or lumbar epidural administration. *Anesth Analg* 1995; **80:** 724–9.
3. Sebel PS, *et al.* Transdermal absorption of fentanyl and sufentanil in man. *Eur J Clin Pharmacol* 1987; **32:** 529–31.

**Children.** Neonates (up to 1 month old) had a significantly lower plasma clearance rate and greater elimination half-life than infants (1 month to 2 years), children, and adolescents.[1] Others[2] have found that infants and small children (1 month to 3 years) with cardiac disease had higher clearance rates and shorter elimination half-lives than reported for adults. Older children (aged 2 to 8 years) with no history of cardiac, renal, or hepatic disease have also been noted to have shorter elimination half-lives and higher clearance rates than adults.[3]

1. Greeley WJ, *et al.* Sufentanil pharmacokinetics in pediatric cardiovascular patients. *Anesth Analg* 1987; **66:** 1067–72.
2. Davis PJ, *et al.* Pharmacodynamics and pharmacokinetics of high-dose sufentanil in infants and children undergoing cardiac surgery. *Anesth Analg* 1987; **66:** 203–8.
3. Guay J, *et al.* Pharmacokinetics of sufentanil in normal children. *Can J Anaesth* 1992; **39:** 14–20.

**Hepatic impairment.** Because of the efficient hepatic extraction and clearance of sufentanil[1] liver dysfunction might be expected to affect its pharmacokinetics. However, elimination kinetics and plasma protein binding were found to be similar in cirrhotic and non-cirrhotic patients after a single dose of sufentanil.[2]

1. Schedewie H, *et al.* Sufentanil and fentanyl hepatic extraction rate and clearance in obese patients undergoing gastroplasty. *Clin Pharmacol Ther* 1988; **43:** 132.
2. Chauvin M, *et al.* Sufentanil pharmacokinetics in patients with cirrhosis. *Anesth Analg* 1989; **68:** 1–4.

**Renal impairment.** The pharmacokinetics of sufentanil were reported[1] to be unaffected in patients with chronic renal failure, although elevated plasma concentrations of sufentanil have been noted[2] in one such patient.

1. Sear JW. Sufentanil disposition in patients undergoing renal transplantation: influence of choice of kinetic model. *Br J Anaesth* 1989; **63:** 60–7.
2. Wiggum DC, *et al.* Postoperative respiratory depression and elevated sufentanil levels in a patient with chronic renal failure. *Anesthesiology* 1985; **63:** 708–10.

## Uses and Administration

Sufentanil, a phenylpiperidine derivative, is an opioid analgesic (p.104) related to fentanyl (p.58). It is highly lipid-soluble and more potent than fentanyl. Sufentanil is used as an analgesic adjunct in anaesthesia and as a primary anaesthetic in procedures requiring assisted ventilation. It has a rapid onset and recovery is considered to be more rapid than with fentanyl. It is also used as an analgesic in the management of postoperative pain and labour pain.

Sufentanil is given as the citrate either intravenously by slow injection or as an infusion, or epidurally. Doses are expressed as the base; sufentanil citrate 15 micrograms is equivalent to about 10 micrograms of sufentanil. Lower initial doses are advised in the elderly and debilitated patients. For obese patients more than 20% above ideal body-weight the dosage of sufentanil should be determined on the basis of the patient's lean body-weight. For details of doses in children, see below. In all patients supplementary maintenance doses should be based on individual response and length of procedure. Doses of up to the equivalent of 8 micrograms/kg of sufentanil produce profound analgesia. Higher doses produce a deep level of anaesthesia but are associated with prolonged respiratory depression and assisted ventilation may be required in the postoperative period.

When used as an analgesic **adjunct to anaesthesia** with nitrous oxide and oxygen for surgical procedures lasting up to 8 hours, the total *intravenous* dosage should not exceed 1 microgram/kg per hour. It is customary to give up to 75% of the dose before intubation followed as necessary during surgery by additional injections of 10 to 50 micrograms or by a suitable continuous or intermittent infusion given so that the total hourly dose is not exceeded. Thus, for an operation lasting 1 to 2 hours the total dose would be 1 to 2 micrograms/kg with 0.75 to 1.5 micrograms/kg being given before intubation.

When used as a **primary anaesthetic** in major surgery *intravenous* doses of 8 to 30 micrograms/kg are given with 100% oxygen; doses of 25 to 30 micrograms/kg block sympathetic response including catecholamine release and are indicated in procedures such as cardiovascular surgery or neurosurgery. Anaesthesia may be maintained by additional injections of 0.5 to 10 micrograms/kg or by a suitable continuous or intermittent infusion given so that the total dosage for the procedure does not exceed 30 micrograms/kg.

In postoperative **pain**, sufentanil is given *epidurally* in an initial dose of 30 to 60 micrograms, which should provide analgesia for 4 to 6 hours. Additional boluses of up to 25 micrograms may be given at intervals of not less than 1 hour if necessary.

Sufentanil is also given *epidurally* for the relief of pain during labour and delivery. Recommended doses are 10 to 15 micrograms given with 10 mL of bupivacaine 0.125% (or its equivalent) with or without adrenaline;