1. NAME OF THE MEDICINAL PRODUCT

Sedacoron® 150mg – concentrate for infusion

2. QUALITATIVE AND QUANTITATIVE COMPOSITION

1 ampoule contains 3ml = 150mg amiodarone hydrochloride concentrate for infusion (= 50mg/ml) as well as 150mg polysorbate 60 in aqueous solution.
For excipients, see 6.1.

3. PHARMACEUTICAL FORM

Concentrate for solution for infusion.

4. CLINICAL PARTICULARS

4.1. Therapeutic indications

Prevention and treatment of life-threatening or severe impairing arrhythmias or arrhythmias refractory to conventional therapy or in cases where other antiarrhythmics are not tolerated.

- Ventricular tachyarrhythmias, including hemodynamically unstable ventricular tachycardia:
  Complex ventricular extrasystole of higher degree
  Recurrent ventricular tachycardia
  Recurrent ventricular fibrillation
- Supraventricular arrhythmias:
  Atrial fibrillation and -flutter
  Paroxysmal supraventricular tachycardia
  Atrioventricular (AV) nodal tachycardia
  WPW-syndrome

4.2. Posology and method of administration

The dosage must be adjusted to meet the individual requirements of each patient, based on the clinical response. It should be striven for the lowest effective dose.
Ventricular extrasystoles usually require higher dosages than supraventricular ones.
If signs or symptoms of pulmonary toxicity occur, it is recommended that Sedacoron® therapy is to be withdrawn until the cause has been determined. If pulmonary toxicity is related to Sedacoron®, withdrawal of Sedacoron® is recommended. Usefulness of steroid therapy is controversial, but such therapy may be useful for severe toxicity. If symptoms of neurotoxicity occur, dosage reduction is recommended; rarely, withdrawal of Sedacoron® may be necessary. If photosensitivity occurs, dosage reduction and use of a sunscreen are recommended. Nausea and vomiting may be relieved by reduction of dose or administration of Sedacoron® in divided doses. If epididymitis occurs, dosage reduction or withdrawal of Sedacoron® is recommended.
For intravenous short-time rsp. long-term infusion per infusion pump. For solution either 5 % isotonic glucose or 0.9 % physiological saline solution can be taken.
Bolus injection is to be avoided.
Sedacoron® should not be mixed with other drugs in the infusion rsp. injection solution or in the same syringe to avoid chemical incompatibilities.
To avoid phlebitis after a long-term infusion a central venous catheter should be attached.
Because of the individual variations of the dosages, the following instructions are only Created by kallemguidelines – especially in children.

Initial short-time infusion:

limited heart function	2.5mg/kg body weight (=0.05ml concentrate)
good cardiovascular function	5.0mg/kg body weight (=0.1ml concentrate)

Long term infusion:
For infusion over 24 hours the daily dose is to be soluted in ca. 500–1000 ml of 5 % glucose. If 0.9 % saline solution is taken, per 150mg Sedacoron® (1 ampoule) at least 250 ml are to be used.
Daily dose: 10–20mg Sedacoron®/kg body weight corresponding to 600–1200mg per patient (4–8 ampoules). The highest daily dose of 1500mg Sedacoron® (10 ampoules) per patient should not be exceeded.
Because of its poor venous tolerance it is recommended to change to oral application (Sedacoron® 200mg – tablets) as soon as possible. As soon as an adequate response has been obtained, oral therapy should be initiated concomitantly with the usual loading dose (200 mg three times a day). Intravenous Sedacoron® should then be phased out gradually. This process is necessary to reach the steady-state-level as soon as possible.
During an emergency, under clinical care, including monitoring of ECG and blood pressure, Sedacoron® may be injected directly intravenously. Therefore 5mg/kg body weight (1–2 ampoules) are administered extremely slowly (within 1–3 minutes). This should not be repeated for at least 15 minutes, even if the first injection was not the highest possible dosage.

4.3. Contra-indications

Except under special circumstances, this medication should not be used when the following medical problems exist:

- Hypersensitivity to Sedacoron® or one of the components of the drug
- Severe arterial hypotension
- Severe congestive heart failure (mild negative inotropic effect of Sedacoron® usually does not cause congestive heart failure)
- Cardiovascular collapse and shock
- Sinus bradycardia
- All forms of conduction delay including sinuauricular and nodal conduction delay, sick sinus syndrome, atrioventricular (AV) block 2nd or 3rd degree without pacemaker (risk of complete heart block)
- Severe thyroid disease, iodine allergy, lung fibrosis, severe liver parenchyme damage, concomitant therapy with MAO inhibitors
- The content of benzylalcohol (60 mg/ampoule = 20 mg/ml) may cause toxic reactions in infants and children up to 3 years. Especially in prematures these reactions can be irreversible, therefore the risk-benefit-ratio must be carefully validated before the administration.
- Pregnancy and breast feeding
- Severe respiratory failure and congestive heart failure are also contraindications when using Sedacoron® as a bolus injection

4.4. Special warnings and special precautions for use

Risk-benefit should be considered when the following medical problems exist:

- Moderate thyroid disease (compensation is necessary)
- Congestive heart failure
- Respiratory failure
- Atrioventricular (AV) block 1st degree
- Reduced hepatic function (reduced metabolism; lower doses may be required)
- Concomitant combination with other antiarrhythmics, beta-blockers and calcium channel blockers
- Hypokalemia (may render Sedacoron® ineffective or arrhythmogenic; should be corrected prior to initiation of Sedacoron® therapy)
- Caution is recommended also during open-heart surgery in patients receiving Sedacoron® because of the risk of hypotension upon discontinuation of cardiopulmonary bypass.

The induction of an Sedacoron® therapy is recommended in a hospital.
As usual in cardiology a thorough risk-benefit consideration should be carried out before an Sedacoron® therapy. An initiation rsp. a change in the dosage requires regular monitoring (ECG and blood pressure) of the patient. Attention has to be paid to adverse reactions during the therapy, together with regular cardiological monitoring (about three-monthly).
Because of the long lasting half-life of Sedacoron®, therapeutically sufficient blood levels are measured some weeks after the discontinuation of the Sedacoron® therapy; the patients still have no discomfort. Life threatening arrhythmias may occur after an ongoing decrease of the blood level. Therefore a thorough monitoring is indicated after discontinuation of Sedacoron® therapy.
Geriatrics: The elderly tend to be more sensitive to the effects of Sedacoron®.
Pediatrics: When Sedacoron® is used concomitantly with digoxin, the interaction has been reported to be more acute in children than in adults. In addition, onset and duration of action of Sedacoron® may be shorter in pediatric patients.
Eyes: Slit-lamp and funduscopy examinations recommended prior to and during the therapy and if symptoms of ocular toxicity occur.
Thyroid: The clinical diagnosis of a hyper- or hypothyreosis in patients receiving Sedacoron® is sometimes not possible, therefore thyroid function determinations are recommended prior to initiation, during and up to one year after withdrawal of Sedacoron® therapy. Because of the iodine-content of Sedacoron® some of the usual tests are insufficient, T3, T4 and TRH-TSH tests should be carried out. If a hyper- or hypothyreosis is suspected, the Sedacoron® dosage has to be reduced or withdrawn.
The following symptoms can be signs of a disturbed thyroid function:
Hypothyreosis: weight gain, weakness, bradycardia which is more intensive than expected with Sedacoron®.
Hyperthyreosis: loss of weight tachycardia, tremor, nervousness, increased transpiration and intolerance to heat, reappearance of arrhythmias or angina pectoris, heart failure.
Liver: Prior to the therapy and during the treatment liver enzyme determinations are recommended: Alanine aminotransferase (ALT [SGPT]), alkaline phosphatase and aspartate aminotransferase (AST [SGOT]). Especially in patients receiving high maintenance doses; dosage reduction rsp. withdrawal of Sedacoron® is recommended if concentrations increase to 3 times of the normal or double in patients with elevated baseline concentrations, or if hepatomegaly occurs.
Lung: Chest X-ray is recommended prior to initiation of therapy and at 3- to 6-monthly intervals during therapy to detect diffuse interstitial changes or alveolar infiltrates associated with pulmonary toxicity. Special caution is indicated in patients with pulmonary disease in their case history.
Auscultation of the chest (recommended at periodic intervals; presence of rales, decreased breathing sounds, or pleuritic friction rub may indicate pulmonary toxicity), pulmonary function test, chest x-ray. Bronchoscopy with lung biopsy (may be useful if symptoms of pulmonary toxicity occur which cannot be diagnosed from a chest x-ray).
Nervous system and skin: If neurological symptoms or severe photosensitivity occurs, Sedacoron® should be withdrawn. The skin should be protected from sunlight rsp. UV-radiation during and for several months following withdrawal of treatment; sunburns may occur even through window glass and thin cotton clothing; the use of protective clothing and barrier sunscreen is recommended.
In patients taking Sedacoron® and suffering from weakness, the syndrome if inappropriate ADH secretion (SIADH) should be considered, and serum sodium levels, osmolality, urine osmolality and urine sodium concentrations measured.
The side-effects of Sedacoron® may be intensified after kind of surgery or emergency treatment. Serum calcium, digoxin and Sedacoron® in the serum should show normal rsp. therapeutic levels. Negative inotropic rsp. chronotropic substances should be discontinued before the surgery.

4.5. Interaction with other medicaments and other forms of interaction

Because of its slow elimination, Sedacoron® may interact with other medications for weeks to months after it is discontinued.

- The concomitant use of other antiarrhythmics, beta-adrenergic, calcium channel blocking agents and class III antiarrhythmics (e.g. sotalol) results in additive cardiodepressive effects and tachyarrhythmias are possible. Sedacoron® increases plasma concentrations of quinidine, procainamide, flecainide, and phenytoin and also phenazone; concomitant use of Sedacoron® with quinidine, disopyramide, procainamide, or mexiletine, aprindine and propafenone has been reported to result in a more prolonged QT interval and, rarely torsade de pointes, and therefore concomitant use of all class I antiarrhythmics requires great caution; the dose of previously given antiarrhythmics should be reduced by 30 to 50 % and gradually withdrawn; if antiarrhythmic therapy is needed in addition to Sedacoron®, it should be initiated at one-half the usual recommended dose.
- The concomitant use of doxepin is not recommended (conduction disorder, tachycardia, hypotension).
- Combined therapy with the following drugs which prolong the QT interval is contra-indicated due to the increased risk of torsades de pointes; for example:
  - Intravenous erythromycin, co-trimoxazole or pentamidine injection
  - Anti-psychotics e.g. chlorpromazine, thioridazine, pimozide, haloperidol
  - Lithium and tricyclic anti-depressants e.g. maprotiline, amitriptyline
  - Certain antihistamines e.g. terfenadine, astemizole
  - Anti-malarials e.g. quinine, mefloquine, chloroquine, halofantrine
- In the combination with digitalis glycosides Sedacoron® increases serum concentrations of digoxin and probably other digitalis glycosides, possibly to toxic levels; when Sedacoron® therapy is initiated, the digitalis glycoside should be withdrawn or the dose reduced by 50 %; if digitalis glycoside therapy is continued, serum concentrations should be carefully monitored; Sedacoron® and digitalis glycosides may also produce additive effects on sinoatrial [SA] and AV nodes.
- Anticoagulants (major clinical significance), coumarin derivatives (warfarin, dicoumarol, phenprocoumon) (Sedacoron® inhibits metabolism and potentiates the anticoagulant effect, beginning as early as 4 to 6 weeks or months after it is withdrawn; prothrombin times may double or triple, but effect is very erratic; it is recommended that the dose of anticoagulants has to be reduced by one-third to one-half and prothrombin times monitored closely).
- Inhalation anesthetics may potentiate hypotension and atropine-resistant bradycardia.
- The concomitant administration of cholestyramine may decrease the effect of Sedacoron®, the dosage must be adapted.
- Drugs causing potassium- or magnesium depletion (for example: diuretics, loop-diuretics, thiazide, indapamide or systemic corticosteroids, laxatives, tetracosactrin, intravenous amphotericin) may lead to an increased risk of arrhythmias. Potassium or magnesium depletion should be compensated prior and during an Sedacoron® therapy.
- Sedacoron® may increase plasma concentrations of phenytoin, resulting in increased effects and/or toxicity (loss of eyesight, tremor, dizziness).
- Sedacoron® may increase the plasma levels of cyclosporin when used in combination, due to a decrease in the clearance of this drug.
- Photosensitizing medications (concomitant use of Sedacoron® may cause additive photosensitizing effects).
- Potentially severe complications have been reported in patients taking Sedacoron® undergoing general anaesthesia: bradycardia unresponsive to atropine, hypotension, disturbances of conduction, decreased cardiac output.
  A few cases of adult respiratory distress syndrome, most often in the period immediately after surgery, have been observed. A possible interaction with a high oxygen concentration may be implicated. The anaesthesiologist should be informed that the patient is taking Sedacoron®.
- Sodium iodide I 123 or sodium iodide I 131 or sodium pertechnetate Tc 99m (thyroidal uptake may be inhibited by Sedacoron®).
- Laboratory test alterations.
  Some thyroid function tests (iodine uptake [binding] test) are disturbed up to one year after withdrawal of Sedacoron®.
- Sedacoron® (amiodarone) can increase the plasma levels of all medicinal drugs metabolised via CYP (2A6, 2C8/9, 2D6, 3A4). After initiation resp. dose increase of amiodarone, a dose reduction of CYP-metabolised medicinal drugs (e.g. flecainide, simvastatin, etc.) must be considered.

4.6. Pregnancy and lactation

Pregnancy:
Amiodarone crosses the placenta; neonatal plasma concentrations of amiodarone desethylamiodarone are 10 % and 25 % of the maternal plasma concentrations, respectively. Although studies in humans have not been done, some reports have indicated an absence of adverse effects when amiodarone was administered late in pregnancy. However, amiodarone can cause fetal harm when administered to pregnant women. Potential adverse effects include bradycardia and effects on thyroid status (iodine is known to cause fetal goiter, hypothyroidism, and mental retardation) in the neonate. There have been a small number of reports of congenital goiter/hypothyroidism and hyperthyroidism.
Sedacoron® must not be administered during pregnancy. Because of the long half-life up to one year after the withdrawal of Sedacoron® a thorough contraception is necessary.
Breast-feeding:
Sedacoron® is excreted in human breast milk. The infant receives approximately 25 % of the maternal dose. Sedacoron® has been shown to cause reduced viability and growth of offspring when used in lactating rats. Mothers should be advised to contact their physician before nursing, since use by nursing mothers is contraindicated.

4.7. Effects on ability to drive and use machines

Depending on individual susceptibility, the patient's ability to drive a vehicle or operate machinery can be impaired, especially in combination with alcohol.

4.8. Undesirable effects

The incidence of side/adverse effects is generally related to dose and duration of therapy. Side effects may appear soon after the initiation of the treatment but also only after several days, weeks, or years after initiation of Sedacoron® therapy and may persist for several months after withdrawal.

Blood and lymphatic disorders:
Very rare <0.01%
Thrombocytopenia.

Bone marrow granulomas.
Haemolytic or aplastic anaemia.

Immune system disorders:
Common >1% to <10%
Allergic reactions: flushing of face, exanthema, and urticaria are possible.
Very rare <0.01%
Anaphylactic shock and benign increase of the intracranial pressure are possible as result of hypersensitivity reactions after intravenous application.

Endocrine disorders:
Very common >10%
Thyroid function: thyroid hormone concentration changes are common and may persist for several months after withdrawal of Sedacoron®. Hyperthyroidism occurs in about 2% of patients, thyrotoxicosis has been reported. Hypothyroidism occurs in less than 10% of patients.
Very rare <0.01%
In single cases, thyreoiditis has occurred.
Syndrome of inappropriate ADH secretion (SIADH) was mentioned in connection with Sedacoron®.

Metabolism and nutrition disorders:
Very rare <0.01%
Hyperglycaemia.
Hyperlipidaemia.
Hypercalcaemia.
Severe weight loss.

Psychiatric disorders:
Common >1% to <10%
Dose-dependent undesirable effects: sleep disorder, nightmare, confusion, depression.

Nervous system disorders:
Very common >10%
Ataxia occurs especially during administration of loading doses; it may occur within 1 week to several months after initiation of therapy and may persist for more than one year after withdrawal. Especially at high doses peripheral neuropathy, paraesthesia and tremor occurred, rarely reversible myopathy.
Common >1% to <10%
Dose-dependent undesirable effects: headache, dizziness.

Eye disorders:
Very common >10%
Bilateral and symmetric asymptomatic corneal deposits (lipofuscin deposits) appearing as yellow-brown pigmentation on slit-lamp examination occur in mostly all patients after 6 months of treatment, but may appear sooner; symptomatic corneal deposits (visual disorders: blurred vision or blue-green halos seen around objects) occur in up to 10% of patients; in this case a reduction of the dose is recommended. Corneal deposits are reversible after reduction of the dose rsp. withdrawal of Sedacoron®, although it may take up to 7 months.
Rare >0.01 to <0.1%
Optic neuritis, optic neuropathy, macular degeneration and papilloedema rsp. decreased visual acuity are rarely reported. Photosensitivity.

Cardiac disorders:
Common >1% to <10%
Cardiac side-effects occur and are more likely caused by high dosage than by pharmacological effects.
Pre-existing congestive heart failure may be intensified, depending on its severity.
Rare >0.01 to <0.1%
Asymptomatic sinus bradycardia, AV prolongation, up to bradycardia of higher degree. Sino-atrial (SA) block, atrioventricular (AV) block and cardiac arrest, requiring a pacemaker, occur rarely. The ECG shows a prolonged and deformed T wave, the appearance of an U wave and a QT prolongation.
New or exacerbated arrhythmias are rare and may include extrasystoles, paroxysmal ventricular tachycardia, ventricular fibrillation (-flutter), torsade de pointes and cardiac arrest. New or exacerbated arrhythmias may also be a sign of hyperthyroidism. Marked hypotension and shock may occur especially with too rapid intravenous application (which may cause reflective tachycardia).

Vascular disorder:
Very rare <0.01%
Petechia.
Thrombophlebitis after intravenous application.

Respiratory, thoracic and mediastinal disorders:
Below mentioned pulmonary changes are generally reversible after early withdrawal of Sedacoron®.
Very common >10%
Pulmonary fibrosis or interstitial pneumonitis or alveolitis are clinically significant in 10 to 15% of the patients, but abnormal diffusion capacity occurs in a much higher percentage; more frequent with doses of 400mg per day and after several months of treatment.
Uncommon >0.1 to <1%
After surgery, several cases of ARDS occurred, which were fatal in single cases.
Rare >0.01 to <0.1%
Rarely diffuse interstitial pneumonitis, alveolitis, pleuritis, bronchiolitis obliterans organising pneumonia (BOOP) or pulmonary fibrosis with the symptoms of cough, dyspnoea, slight fever, hypoxia, reduced pulmonary function and X-ray verifiable infiltrates.
Very rare <0.01%
Atypical pneumonitis.
Single fatal cases were reported.

Gastrointestinal disorders:
Very common >10%
Occur in general at the beginning of the therapy and disappear after reduction of the dosage: nausea and vomiting, epigastric abdominal pain, constipation, sensation of fullness and anorexia. Also taste disturbances (metallic taste).
Very rare <0.01%
Gingival haemorrhage.

Hepato-biliary disorders:
Common >1% to <10%
Hepatic complaints: increased serum transaminases which were reversible after dose reduction or withdrawal and cases of histologically detectable liver disturbances, hepatitis, jaundice and liver cirrhosis, liver failure, cholestasis (in single cases fatal) (see "Special warnings and special precautions for use"). A transient isolated increase of liver enzymes can appear dose-independent.

Skin and subcutaneous tissue disorders:
Common >1% to <10%
Dermatologic side effects: photosensitivity, particularly to long-wave ultraviolet A [UVA] light, sensitivity of skin to sunlight, sunburn. Photosensitivity may occur even through window glass and thin cotton clothing; not dose-related and reversible. Since most sunscreens are not useful for protection because they only block ultraviolet B [UVB] light, a barrier sun-block such as zinc or titanium oxide and protective clothing are recommended.
Uncommon >0.1 to <1%
Pseudocyanotic, blue-grey colouring of skin on face, neck and arms occur with prolonged use, usually longer than 1 year, especially in patients with fair skin or with excessive sun exposure; slowly and occasionally incompletely reversible after withdrawal.
Rare >0.01 to <0.1%
Depending on the predisposition of the patient, in rare cases a psoriasis eruption may be induced or an existing psoriasis may worsen.
Very rare <0.01%
Purpura.
Lyell's syndrome (toxic epidermal necrolysis, erythema multiforme).
Eythema nodosum.
Pruritus.
Alopecia.
Exfoliative dermatitis.
Angioedema.
Drug induced lupus have been described; symptoms disappeared gradually after discontinuation of Sedacoron®.
Ecchymosis.

Reproductive system and breast disorders:
Very rare <0.01%
Arthropathy and orchialgia as a result of epididymitis.
Erectile dysfunction (impotence).
Orchiatrophia.
Gynaecomastia.

General disorders and administration site conditions:
Common >1% to <10%
Dose-dependent undesirable effects: tiredness.
Very rare <0.01%
Hypersensitivity reactions involving vasculitis.

Investigations:
Very rare <0.01%
Reduced renal function.

4.9. Overdose
Symptoms:
In general the symptoms are sinus bradycardia, sinuauricular and nodal conduction disturbances. Because of its special pharmacokinetic properties overdosage occurs within long-term indication.
For treatment of overdose:
- Treatment is primarily supportive and symptomatic and may include the following:
- Recent oral ingestion may benefit from induced vomiting and/or lavage.
- Monitoring of cardiac rhythm and blood pressure is important.
- For bradycardia, a beta-adrenergic agonist or pacemaker may be indicated.
- Hypotension may respond to positive inotropic and/or vasopressor agents.

5. PHARMACOLOGICAL PROPERTIES
Drug Class: Antiarrhythmic. ATC Code C01B D01
Molecular weight: 681.8
pKa: 5.6.
Contains 37.3% iodine by weight; highly lipophilic.

5.1. Pharmacodynamic properties
Prolongs action potential duration and refractory period in all cardiac tissues (including the sinus node, atrium, atrioventricular [AV] node and ventricle) by a direct action on the tissues, without significantly affecting membrane potential. Decreases sinus node automaticity and junctional automaticity, prolongs AV conduction and slows automaticity of spontaneously firing fibers in the Purkinje system. Prolongs refractoriness and slows conduction in accessory pathway tissue in patients with Wolff-Parkinson-White (W-P-W) syndrome. Also causes noncompetitive alpha- and beta-adrenergic receptor antagonism and calcium channel inhibition and affects thyroid hormone metabolism, but relationship of these effects to its antiarrhythmic action is unknown. In the Vaughan Williams classification of antiarrhythmics, amiodarone is considered to be a predominantly class III agent, with some class I, II and IV properties.
Other actions/effects: Has a mild negative inotropic effect, more prominent with intravenous than with oral administration, but usually does not depress left ventricular function. Causes coronary and peripheral vasodilatation and therefore decreases peripheral vascular resistance (afterload), but causes hypotension only with large oral doses.

5.2. Pharmacokinetic properties

Absorption
Slow and variable; about 20 to 55 % of an oral dose is absorbed.

Distribution
Large and variable volume of distribution as a result of extensive accumulation in fatty tissue and highly perfused organs (liver, lung, spleen, heart) leads to slow achievement of steady state respectively therapeutic plasma concentrations and prolonged elimination.

Bioavailability
22–86 %

Protein binding
Strongly protein bound (96%)

Biotransformation
Hepatic, extensive; one active metabolite (desethylamiodarone: biliary elimination); possibly also by deiodination (a dose of 300mg releases approximately 9mg of elemental iodine, which are eliminated in the urine).

Half-life
Initial phase of the half-life 8 min, slow phase 4–10 days (mean 7 days).

Therapeutic plasma concentration
1 to 2.5mg per litre at steady state (after 2 months of therapy). However, antiarrhythmic effect is difficult to predict by means of plasma concentrations and toxicity may occur even at therapeutic concentrations.

Duration of action
Variable weeks to months; plasma concentrations are measurable for up to 9 months after amiodarone is discontinued.

Elimination
Biliary.
In breast milk: About 25% of maternal dose is excreted in breast milk.
In dialysis: Not removable by hemodialysis.

5.3. Preclinical safety data

Carcinogenicity/Tumorigenicity
Studies in rats at doses one-half the maximum recommended human maintenance dose and greater found a dose-related increase in the incidence of thyroid follicular adenomas and/or carcinomas.

Mutagenicity
Mutagenicity studies (Ames, micronucleus and lysogenic tests) with amiodarone were negative.

6. PHARMACEUTICAL PARTICULARS

6.1. List of excipients
Polysorbate 60, water for injections.

6.2. Incompatibilities
Dilute only with 5 % isotonic glucose or 0.9 % physiological saline solution. Other solutions are not recommended.

6.3. Shelf life
36 months

6.4. Special precautions for storage
Do not store above 25°C.
Keep container in the outer carton, in order to protect from light.

6.5. Nature and contents of container
Ampoule of Ph.Eur. Type I glass.
5 ampoules containing 150mg/3ml of amiodarone HCl, each.

6.6. Instructions for use and handling
None.

7. MANUFACTURER
EBEWE Pharma Ges.m.b.H. Nfg.KG, A-4866 Unterach, AUSTRIA

8. DATE OF (PARTIAL) REVISION OF THE TEXT
September 2004