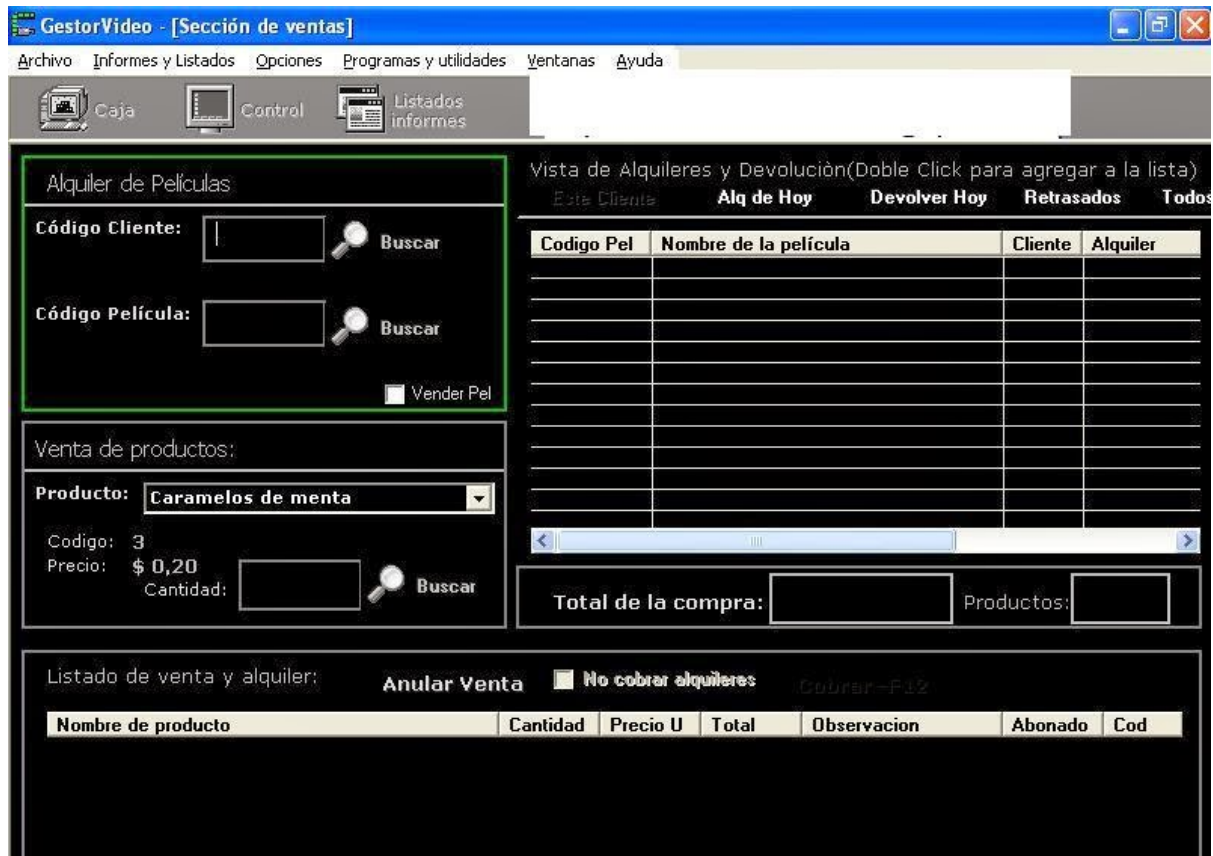
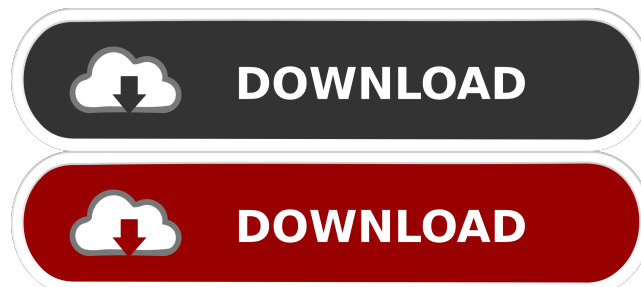


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Heparin-induced thrombocytopenia and thrombosis: Pathophysiology, diagnosis, treatment, and prognosis. Heparin-induced thrombocytopenia and thrombosis (HIT) is a life-threatening adverse drug reaction. Patients with HIT present with thrombotic and/or thromboembolic events after exposure to heparin. HIT is most commonly associated with heparin exposure, but has been described after administration of nonheparin anticoagulants such as lepirudin and bivalirudin. The pathophysiology of HIT is complex and involves multiple factors. The presence of multiple causative factors at a specific time allows for multiple potential causes of HIT, and underscores the importance of its timely diagnosis. HIT is diagnosed by measuring platelet-associated heparin-dependent antibodies. However, these assays are not entirely sensitive and specific, which is partly due to the use of a single monoclonal anti-heparin antibody in some commercially available assays. The optimal management of HIT is intravenous recombinant tissue-type plasminogen activator (rt-PA) if a patient is clinically stable, but if the patient presents with significant life- or limb-threatening thromboembolic complications, the standard of care is to administer heparin with the addition of thromboprophylaxis with low-molecular-weight heparin. Newer oral direct thrombin inhibitors (dabigatran and argatroban) have been shown to be at least as effective as warfarin in preventing thromboembolic events in HIT patients. The combination of an oral anticoagulant with rt-PA may be more effective than rt-PA alone. Bivalirudin is effective for the treatment of HIT 82157476af

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